The role of psycho-social experience in chronic disease

James McAlister Alexander

Southern Cross University
THE ROLE OF PSYCHO-SOCIAL EXPERIENCE IN CHRONIC DISEASE:

James McAlister Alexander
B. Arts Degree (Psychology/Sociology); Post Graduate Diploma in Counselling Psychology
School of Education, Southern Cross University
Submitted for the degree of Doctor of Philosophy
Date submitted: 29th of June 2006
Thesis Declaration.

I certify that the work presented in this thesis is, to the best of my knowledge and belief, original, except as acknowledged in the text, and that the material has not been submitted, either in whole or in part, for a degree at this or any other university.

I acknowledge that I have read and understand the University’s rules, requirements, procedures and policy relating to my higher degree research award and to my thesis. I certify that I have complied with the rules, requirements, procedures and policy of the University (as they may be from time to time).

Names: James McAlister Alexander

Signature: ……………………………

Date: 29th of June 2006.
ABSTRACT:

This study examines the role of psycho-social factors in contributing to chronic illness, in particular Coronary Heart Disease (CHD) and cancer. Research conducted over the last 50 years indicates a modest role for psycho-social factors as risk factors for these conditions. The research suggests that in combination with other modest risk factors, psycho-social factors play an equally important role. Grossarth-Maticek & Eysenck (1990) gained notoriety by reporting research results which seriously question current wisdom. Grossarth-Maticek & Eysenck (1990) conducted what must be considered the largest long term prospective psychological health study. They followed around 20,000 probands for 15 years in order to determine the relative contributions to health outcomes (primarily cancer and CHD) of standard medical and psycho-social risk factors. They reported an 81% accuracy of prediction of death by cancer or CHD with the use of a psychometric test. Further, answers to the test were reported to be six times more predictive of cancer or CHD than were any of the standard medical risk factors, either taken on their own or together.

Being far in excess of claims made by any other researchers, the treatment/prevention implications of Grossarth-Maticek’s study are far reaching. It is an important endeavor to examine possible influencing factors in these results. While scientific fraud can never be excluded, in the absence of any incriminating evidence, this assertion is considered to be non-empirical. Amelang (1991) wonders whether unknown, favourable and non replicable conditions were present in Grossarth-Maticek’s samples or methodology which influenced the results.

The current research program represents an attempt to ascertain what some of those factors may have been. It is suggested here that Grossarth-Maticek’s probands learnt of their cancer or CHD proneness in the course of the interview which formed the basis of the data collected in their study. Focusing on this aspect of Grossarth-
Maticek’s data collection methodology, the possibility of a large scale treatment effect emerges. The most psychologically vulnerable of his probands, having been stimulated by the interview process, may have ruminated and ‘catastrophized’ over their health prospects during the ensuing years, thereby making themselves physiologically more vulnerable to the feared conditions. The possibility of probands being adversely influenced by the interview is tested in the current study with subjects being measured for changes in cancer/CHD risk perception and anxiety levels as a result of participation in the ‘Grossarth-Maticek interview’ format.

In order to test this hypothesis, two studies were undertaken in which Grossarth-Maticek’s data collection method was replicated with 105 subjects. Subjects were assessed on a range of self report items prior to the ‘Grossarth-Maticek’ interview, and again post to this interview. They were assessed as to their beliefs about vulnerability to cancer and CHD; and their levels of anxiety in relation to contracting these conditions. Subject’s responses to learning of their ‘health prediction’ according to Grossarth-Maticek’s assessment tool were also measured via a heart rate monitor. Changes in subject’s self report measures and heart rate responses were noted and associations to the type of health prediction which they were given at the end of the interview was assessed. In the second study, the relative effects of family history, Neuroticism and time on disease risk perception and anxiety were studied via statistical analyses of self report measures.

The results of this study do not support the notion of the interview having any adverse influence. Problems with the methodology and sample selection may have influenced the outcome. It is concluded that for these reasons, the current study did not represent a thorough test of the current hypothesis. Recommendations for a superior test of this hypothesis are made.
Thesis Declaration.

I certify that the work presented in this thesis is, to the best of my knowledge and belief, original, except as acknowledged in the text, and that the material has not been submitted, either in whole or in part, for a degree at this or any other university.

I acknowledge that I have read and understood the University's rules, requirements, procedures and policy relating to my higher degree research award and to my thesis. I certify that I have complied with the rules, requirements, procedures and policy of the University (as they may be from time to time).

Name: James McAlister Alexander

Signature:..........................................................

Date:- ..........................
This thesis is dedicated to the various generations of my family. To my ancestors who crossed the planet in the belief that their children would do well in the Great Southern Land. To my parents who have always encouraged education and stressed its importance, even when I showed few promising signs. To my children who have spent most of their lives with a father who was working on his PhD. And finally, to my wife Karen, whose support and encouragement made it possible for me to attain this goal. I hope that the rest of my career in psychology proves worthy of their faith.
The term ‘chronic disease’ is used by Beaglehole (2005) of the World Health Organisation to include such conditions as stroke, diabetes and respiratory diseases in addition to cancer and Coronary Heart Disease (CHD). Rather than restricted as the leading causes of death in the developed world alone, Swan (2005) states that chronic disease is the biggest cause of death and disability globally. Demonstrating that this is not just a Western phenomenon, the Chinese national economy is predicted to lose around $558-billion, the Russian Federation around $300-billion, and the Indian economy around $236-billion to chronic disease over the next decade. Amongst other factors, Beaglehole (2005) states that the leading causes of these diseases are unhealthy diets, physical inactivity and tobacco consumption- the more that these factors spread into developing countries, the more the chronic disease rate increases. He estimates that chronic disease will account for around 60% of all deaths in the world this year, amounting to 35 million deaths for 2005 (Beaglehole 2005). Such is the social, medical and economic gravity of the arena into which Health Psychology bravely enters. The use of the term ‘chronic disease’ in this paper will be restricted to cancer and CHD, the two leading causes of death in Australia (Abraham, d'Espaignet & Stevenson- 1995), and more generally in the entire Western world (Brown & Lees-Haley -1992).

The notion that psychology could have a role to play in the assessment for risk of chronic diseases such as cancer and CHD, the prevention and even the treatment of such conditions comes as something of a surprise to many psychologists outside of the health psychology arena- the strength of some of these claims come as a surprise to many within Health Psychology.
Holland & Lewis (in Goleman & Gurin 1993 p.85) state that there has evolved a “cottage industry” of tapes, books, lectures and self help groups in the prior two decades promoting the view that people can successfully treat cancer simply by “putting their heart and soul into recovery”. They state that a wide spread concern within the medical profession exists in regards to belief about the role of guilt, lack of self love, repression etc in the causation of cancer. These beliefs, they contend, are firstly unhelpful and secondly unsupported by the evidence. One apparent demerit of these beliefs is that if personal ‘deficiencies’ can be held responsible for chronic disease conditions like cancer and CHD, then an unhelpful guilt can ensue following the onset of the condition. A notion of ‘why did I let this happen to me?’ may only add to the burden of the disease with additional and perhaps unnecessary psychological overlay. The other potential problem with these notions is the lack of research based evidence to support some of the contentions. This is problematic in that such beliefs can deter the seeking of medical attention for serious conditions, with the disease being more advanced and therefore more difficult to successfully treat when medical intervention is eventually sought.

Psychologists who have approached or remained functioning within their discipline/profession with a degree of intellectual honesty have been aware of controversial ethics in the fields of applied psychology such as psychotherapy and psychological assessment. In fact, these applications of psychology have been so riddled with controversy that in promoting or practicing psychotherapy, the psychologist is faced with a host of considerable ethical dilemmas. Szasz (1961) began the modern era of critical psychology with an intellectual attack on the notion of mental illness, favouring a re-working of neo-psychoanalytic and civil libertarian notions and an attack on the ‘medical model’ of mental illness. Psychiatry was his primary target, however psychology was not exempt from his critique to the extent that clinical psychology in particular has adopted, endorsed and worked within the ‘medical model’. While supportive of cognitive approaches in psychology (referring to them as ‘pre-psychological’, and therefore as ethically sound), Gross (1978) was highly critical of any form of ‘depth’ or psychoanalytic psychology. Masson (1988) suggests that all applications of psychology, regardless of the approach taken, are morally bankrupt as
they can never be performed from a position which does not rob the client of their social
power- all recipients must be disempowered by their contact with a psychologist, as all
psychologists must be empowered by the same process. As such, he suggests that the
only morally dignified path is to decline to conduct any form of psychotherapy.

Rappaport (1977) views the practice of psychology, in addressing standard clinical
issues, as being an inherently political process. The psychologist is either promoting an
adjustment of the individual to social/political circumstances and standards, thereby
assisting in the maintenance of the status quo; or s/he is assisting the individual to
challenge things as they are. As long as the level of intervention remains aimed at the
individual (as is the case for most psychology interventions, rather than at the
societal/cultural level), either option is inherently conservative as broad social/political
structures are not challenged. As such, psychological intervention aimed at the individual
level is viewed as an inherently political action. Regardless of how s/he feels about it, the
politically aware practitioner is unable to escape this perspective.

On learning of such controversies, the psychologist can choose to ignore them and
proceed practicing as though such issues did not exist; or alternatively, s/he can attempt
to struggle with and come to some kind of personal resolution of the issues involved.
Regardless of the preferred option, it is proposed here that some psychologists, once they
have explored such issues and have attempted to wrestle with them in actual practice,
often become disillusioned with their discipline/profession. In a culture which has been
described as ‘The Psychological Age’ (Gross 1978), wherein psychology is popularly
assumed to provide the answers to many compelling personal, ethical, and social
questions, psychologists who have actually explored the difficulties and issues referred to
above often have less confidence in the whole enterprise than does the general public. As
such, they may become cynical in regards to the value of psychology per se- many of the
critical perspectives are simply too compelling for a thinking professional with an open
attitude to ignore.

Few people enter the field of psychology aware that their actions may be viewed from
this critical perspective and many, if not most will find it a source of disquiet. When such
a psychologist is confronted with the notion that despite these problems in relation to psychological treatment for emotional distress, psychology may actually be of demonstrable value in regards to assessment for risk, prevention and treatment of cancer and CHD, some of the stated dissonance and strain may be alleviated. What relevance do political ramifications have for issues of prevention of serious chronic disease? Could Masson (1988) argue that it is a political violence to implement a psychological intervention aimed at preventing the onset of premature death by cancer or CHD? Could critics of psychometrics remain hostile to such endeavours if it is established that a person’s answers to a test can accurately predict the risk of cancer or CHD, and thereby alert the person to the need for ‘life saving’ change? Can it be argued that the required changes are simply an enterprise of adjusting people to a socially desired norm (unless survival itself is to be viewed as a ‘social norm’), when such changes may stall or prevent the onset of life threatening illnesses? If such knowledge is found to be empirically supportable, would Szasz (1960), Gross (1978), Masson (1988), Rappaport (1977) or any of the other critics of psychology suggest that it is a more politically ‘pure’ action to not present this information and allow people to die prematurely from preventable conditions? Few political commentators would suggest so.

When addressing issues of life and death, as can this venture, health psychology enters a new arena, beyond the mere pursuit of happiness or adjustment to social norms. This can be perceived by the disillusioned practitioner as having the potential to ‘save’ psychology from the moral quandaries as described above- in fact, it renders many of them superfluous. It is little wonder that many psychologists have become excited by such prospects.

Psychology introduces the possibility of a research evidence basis in addressing the possible contribution which psychosocial factors may have in the causation of cancer and CHD. In the contemporary ‘marketplace’ of ideas, such ‘New Age’ authors as Louise Hay (1984a; 1984b) have become renowned for promoting the view that thought patterns and deep levels of feeling are able to either damage biological functioning, or can have a curative role. Such ‘intuitive’ propositions are made not from an empirical research base,
but from a more philosophical/spiritual and anecdotal epistemology. Perhaps as a function of wishful thinking as well as the psychological need to respond to the threat of these conditions in a manner which restores some sense of control (Davidson 2005), a portion of the general public appears to have readily accepted these views, along with a very wide range of other non-empirically validated health propositions. Davidson (2005) found that amongst a sample of cancer patients, those who chose to use alternative therapies displayed a higher internal recovery locus of control than those who chose to use only standard medical therapies- the use of complementary therapies fulfils an important psychological need for some patients, even if they are not empirically validated. A recent study estimated that Australians currently spend around $2.3 billion a year on alternative medicines and therapies which, for the most part are of no empirically demonstrated value. MacLennan (2002 p.1) states “Most alternative medicines are unproven. They often have had no scientific evidence to show that they work and there have been no long-term trials of their safety.” The healing properties of the mind, promoted by authors such as Hay (1984a; 1984b) fall well within this category, however it would not appear that an unwilling public has had these views foisted upon it- in fact, judging by the general public’s enthusiastic embrace of such practices, quite the opposite seems to be the case.

With the decline of institutionalized and formal religion, there is a need in the community for quasi-spiritual beliefs that are perceived to have the power to make a difference in people’s lives (Carroll 1998). ‘New Age’ health beliefs and practices may currently be fulfilling this need for many in our society. Judging by the amount of interest within psychology, it would appear also that research health psychologists share a level of excitement about the prospect of psychology’s role in the causation of major ill-health, albeit not from an overtly ‘spiritual’ perspective. Unlike authors such as Hay (1984a; 1984b), the attempt of psychologists has been to introduce a degree of scientific rigour into the discussion rather than have it rely entirely on intuitive appeal and anecdotal accounts of recovery. If the public is vulnerable to the deleterious effects of such social phenomenon as the cancer ‘cottage industry’, as Holland & Lewis (in Goleman & Gurin 1993) suggest, then psychology has a role to play in testing such claims and protecting
the public from spurious assertions. It also has an ethical responsibility to determine if psychosocial factors do play such a role in the causation of chronic disease, and to articulate these as well as possible treatment approaches. If an empirical basis to claims of psychological prevention or treatment for chronic disease is found, how many of the 35 million deaths this year could be prevented?

This paper will describe a wide range of research programs that have attempted to bring an empirical basis into the discussion of psycho-social factors in the causation of cancer and heart disease. The claims of the researchers are often more conservative than the ‘cottage industry’, however there are some noticeable exceptions to this conservatism which will be reviewed, one of which will be the main focus of the current study.

Based on the current state of research evidence within psychology, there would seem to be a role for psychology in delineating, defining, assessing, measuring, and then testing the role of psychosocial factors. This is a brave venture for psychology to have embarked upon- not only can it be accused of participating in the cottage industry referred to as above, but it can easily alienate medical colleagues with claims to expertise in issues which may impact upon health, a realm traditionally viewed as medicine’s prerogative.

Psychological researchers, Eysenck & Grossarth-Maticek (1991) are claiming to have developed psychometric tests assessing a respondent’s status in terms of vulnerability to CHD and cancer. These researchers are claiming to have demonstrated that proband’s profiles, as assessed by these questionnaires, have *six times* greater power in predicting subsequent cancer and CHD rates as do the known physical risk factors such as cigarette smoking, alcohol consumption, hereditary factors. In addition to being able to predict susceptibility to these conditions according to a psychosocial profile, Eysenck & Grossarth-Maticek (1991) claim to have demonstrated that a specifically designed version of behaviour therapy can actually prevent or delay the onset of these illnesses in vulnerable people for up to a period of 15 years, if not indefinitely. In addition, other researcher/practitioners are claiming that various forms of psychosocial treatment, such as social/emotional support groups are at least as effective or more so in ‘treating’
advanced cancers as are the conventional forms of medical treatment, if not more effective (Spiegel, Bloom, Kraemer & Gottheil 1989).

The strength and veracity of some of Grossarth-Maticek & Eysenck’s claims have been disputed (Amelang 1991; Binik 1991; Fox 1991), however, independent of them there does appear to be some empirical basis to accepting the role which psychological factors play in the causation of cancer and CHD. Mathews, Yousfi, Schmidt-Rathjens & Amelang (2003) suggest that there is a growing body of evidence supporting a general role for psychology. They state that traditional medical risk factors such as cigarette smoking, diet, genetics etc, are actually unable to account for the onset of most chronic diseases. This view is contrasted with WHO authorities such as Beaglehole (2005), who that proposes that smoking and diet are amongst the leading causes of chronic disease.

Psychologists are attempting to ascertain the nature of the stressors implicated. Research in health psychology is suggesting that neuroticism is involved in increased risk of disease and premature morbidity, although ‘considerable uncertainty remains’ (Mathews et al 2003 p159) as the relationships found tend to be ‘low in magnitude’ (Yousfi, Mathews, Amelang, Schmidt-Rathjens 2004). Butow, Hiller, Thackway and Kricker (1997), after reviewing the methodology and results of all published psychosocial-breast cancer studies concluded that there is ‘weak’ evidence for the relationship between psychosocial factors and breast cancer, especially emotional repression (particularly of anger) and loss of a significant other. While being very cautious in concluding a role for psychosocial factors, they were unwilling to completely dismiss the possibility, stating that “few studies have been sufficiently rigorous to definitely rule out a minor role” for psychosocial factors. Amelang (1997 p. 320) suggests that psychological factors have been repeatedly shown to account for around 3% of the variance in health/illness outcomes “over and above traditional medical risk factors such as smoking, high blood pressure, high cholesterol levels, and lack of exercise”. Respected researchers such as Seligman (1991), Cooper & Faragher (1992), Spiegel (in Speigel, Bloom, Kramer & Gottheil 1989), amongst many others have provided empirical evidence of at least
modest psychosocial correlates for ill health; while others, such as Marmot (1998), have proposed a more prominent role for psychosocial factors.

Garson (2004) conducted a review of prospective study findings over the preceding 30 years which have attempted to answer whether psychological factors have a role to play in the causation of cancer. From analyzing 70 studies, he provided the qualified view that there is not any psychological factor which has been found to convincingly demonstrate an influence on cancer development in a series of studies. There were psychological factors found for which an influence ‘cannot be totally dismissed’ and which emerged as ‘most promising’ (Garson 2004 p. 315). These factors were helplessness and repression, while less convincing evidence was found for having experienced loss events, low level of social support and chronic depression in the causation of cancer. Factors such as life events (other than loss events), stoic acceptance/fatalism, active coping, locus of control, negative emotional states, and fighting spirit were found to have no or only minor influences on the onset of cancer. Garson (2004) points out a methodological limitation in his analysis in that the interactive effects of psychological factors, biomedical and demographic risk factors were not investigated. In regards to the causation of CHD, Smith, Glazer, Ruiz and Gallo (2004) state that their review of published research findings concluded both considerable evidence in support of the role of psychological factors in the causation of CHD, as well as limitations. As such, the dispute within psychology appears to be not so much whether psychosocial factors may play a role in the causation of illness, but the nature and strength of that role, as even the critical studies conclude some role even if only minor.

If traditional risk factors such as cigarette smoking, diet, genetics etc, are unable to account for the onset of most chronic diseases (Mathews et al 2003), then a role for psycho-social factors, or a synergistic relationship between psycho-social and traditional risk factors is suggested. Clearly, there is not yet a consensus within psychology in regards to the role of psychosocial risk factors in the causation of cancer and CHD- the role which psychology may play is juxtaposed to the traditional biomedical model of medicine.
The Biomedical Model of Medicine:
The biological ‘medical model’ of even physical illnesses is unable to accommodate notions of psychosocial involvement in the causation of serious medical conditions (Felton in Ader, Felton, Cohen 1992; Capra, 1982; Engel 1977). Engel (1977) states that at least until the beginning of the 20th century many physicians still maintained a belief in the contributing role of emotions towards the development and course of disease. However, towards the end of the 19th century Pasteur discovered the role of microbes as infectious agents with the implication that diseases came to be viewed as specific entities, each with a specific germ cause.

“The dominant model of disease today is biomedical, with molecular biology its basic scientific discipline” (Engel 1977. p130).

The implications of this particular approach to medicine are both physicalistic reductionist, as well as dualistic wherein the mind is viewed as being distinct from the body (Russell 1961).

In terms of the physicalism, Engel (1977) states as the guiding assumptions that the language of chemistry and physics will ultimately explain all biological phenomena with “the philosophic view that complex phenomena are ultimately derived from a single primary principle” (p.130). Engel (1977) suggests that the biomedical model of medicine has become contemporary culture’s popular ‘folk model’, rather than a scientific model. It has become a cultural imperative operating as a dogma, with its limitations rarely questioned. In contrast to science, dogmas require that un-supporting data are either excluded from the domain, or are modified to fit the existing model.

Eysenck (1985) suggests that with the rise in popularity of the physicalistic view within medicine, those who subscribed to the view that, for example, a virus may be only one agent in a complex chain of events, became ignored and discredited, or excluded. The traditional holistic view appears to have prevailed until the beginning of the 20th century.
Sir William Osler, who Eysenck (1994 p 167) refers to as the ‘father of British medicine’, stated in 1906,

“It is many times much more important to know what patient has the disease than what kind of disease the patient has”.

It would seem, however, that this type of sentiment was shortly to be eclipsed by the ascending Biomedical model of medicine. Perhaps at the time it was one of the last statements of a traditional wisdom that had been developed through careful observation over the preceding centuries. That wisdom had run its course and was soon to be disparaged as a quaint relic of a pre-scientific age.

In recent years, medical science has developed what is referred to as the bio-psycho-social model, taking into account psychological, social and physical factors. The Australian Pain Society (2005. p1) states that “This model, which has developed over the past 30 years has aided the management of many forms of chronic illness”. As the title suggests, the bio-psycho-social model attempts to integrate both psychosocial and biological models of ill-health in order to develop a comprehensive picture of human functioning. This has been a necessary adaptation in light of compelling research evidence which posits a role for psycho-social aspects of functioning, and an ‘Information Age’ which has disseminated these findings to the general public. Osterman (2004) points out that the Internet is being increasingly used by general medical patients, and by cancer patients in particular. As the public becomes increasingly educated, with the aid of such technologies as the Internet, the need to incorporate psychosocial factors in medical explanations and treatments of health problems becomes more compelling—patients are generally very aware of their own psychosocial realities, even if their physicians have not been. Medical students are now taught the bio-psycho-social model, and this must be considered a great advance in the zeitgeist of contemporary medicine. Ultimately, it will lead to cultural change not only within the medical profession but also in the public’s expectations about medical services.
Despite this recent advance, the role of psychological factors in the causation of chronic diseases such as cancer and CHD is still excluded from the discussion in favour of exciting advances in micro-biology and genetic science (see Trichopoulos, Li & Hunter (1996) and Weinberg (1996) in the Scientific American special edition on cancer). Ten years after this Scientific American special edition, Ezzati (2006) reports on International Health research conducted by the Harvard School of Public Health. The purpose of the study, conducted by over 100 research groups across the world, was to determine the major causes of chronic disease and articulate the risk factors. Of the 20 risk factors for chronic disease studied, the following were found to be related to cancers:- cigarette smoking, alcohol consumption, obesity, low consumption of fruit and vegetables, unsafe sex practices, urban air pollution, injection with used syringes, and indoor cooking with coal and biomass fuels. Of interest to this paper is the fact that none of the risk factors identified related to psychological factors such as distressing life experiences, neuroticism, repression of emotions, etc- psychology does not enter the discussion.

For the medical establishment, the psychosocial assertions in regards to conditions such as cancer and CHD can represent a challenge to medical authority and expertise. However, as several commentators have pointed out the lack of accommodation of psychosocial factors is largely a recent phenomenon, occurring in medicine only over the last hundred or so years (Eysenck 1991b; Engel 1977).

Psychology and traditional medicine:
The oldest reference which Eysenck (1991b, p 230) quotes is that of an ancient Indian sage, who stated 4000 years ago:

“There are two classes of disease- bodily and mental. Each arises from the other. Neither is perceived to exist without the other. Mental disorders arise from physical causes, and likewise physical disorders arise from mental causes”.

The ancient sage is suggesting a philosophy of monism as opposed to dualism, or perhaps better stated as a ‘holistic model’ of medicine. In the history of Western culture, dualism...
can be seen as a general trend in philosophy from some of the ancient Greeks, through to Christian philosophers (Russell 1961). This trend culminated in the doctrine espoused by Rene DeCartes, where by the worlds of physical reality and mental reality were presumed to be independent from each other. Engel (1977) suggests that Christian theology was largely responsible for this division of mind and matter- around five centuries ago, the body was viewed as an imperfect vehicle for the transmission of souls from this to the next world. As such, the Church approved the study and dissection of the human body, however it did not approve of the study of the mind or behaviour, as this was seen as the rightful domain of religion. With mind-body dualism being the Church approved doctrine, fledgling medicine developed the metaphor of the body as machine, disease being the breakdown of the machine and the physicians job being the repair of the machine. With this thrust were the seeds of what Engel (1977) has referred to as the crisis of medicine. This crisis is the logical inference from the definition of disease, as concerning only somatic parameters, that psychosocial issues are of no interest or consequence to the biomedical model of medicine.

The West, however, also has a history of what may be referred to as ‘holistic medicine’. The application of Cartesian dualism to medical practice as seen in the Biomedical Model of disease (Engel 1977), only gained dominance in the West over the last century. Eysenck (1993) states that 2000 years ago Hypocrates was suggesting notions similar to the Indian sage quoted above, and was thereby promoting a non dualistic model of illness. Specifically, Hypocrates suggested that the bodily fluids were causal in the aetiology of various mental disorders (Davison & Neale 1995), and that personality factors, eg. melancholia, were relevant in the aetiology of physical illnesses such as cancer (Eysenck 1993). In the second century AD. the Roman physician Galen, viewed as being the last of the Classical physicians, promoted the notion of personality as being a relevant factor for physical ailments- specifically, he suggested that personality was important in the formation of neoplasms (Eysenck 1985).

Early observers and writers on the subject continued in Galen’s line of thought. In a review of literature from 1701-1893, Eysenck (1985) states that there were 14 anecdotal
studies cited in the medical literature which suggested a link between extreme emotional stress and/or loss and the development of cancers. For example, Gendron in 1701 (cited in Eysenck 1994), was amongst others from the next two centuries who suggested that stress, leading to feelings of helplessness, hopelessness and depression was an important factor in the onset of cancer. Other observations suggested that temperaments that were of a more sensitive and easily frustrated nature were more susceptible, as were those who suppressed their emotions. In summarising the clinical observations up to that point in time, Walsh in 1846 (cited in Eysenck 1994 p 168) stated:-

“Much has been written on the influence of mental misery, sudden reverses of fortune, and habitual gloominess of temper on the deposition of carcinomatous matter...whether this be the real catenation of circumstances or not, and although the alleged influence of mental disquietude had never been made a matter of demonstration, it would be vain to deny that facts of a very convincing character in respect to the agency of the mind in production of this disease are frequently observed”.

Information from another quarter, the Pavlovian school of physiology, experimentally demonstrated early on that acute stress induced in dogs would create not only behavioural disturbances, but also chronic diseases. Petrova, a student of Pavlov, demonstrated that spontaneous tumours could be created in dogs undergoing such experimentally induced stress (Quander-Blaznik 1991).

**Early Psychological Research on Cancer:**
Weinberg (1996) states that cancer is a term used to refer to over 100 diseases which share a common feature, ie. a dysfunction of the controls for growth and reproduction within the cells. As compared to normal body cells, cancer cells reproduce and drain the system without providing any benefit to the body. Normal cell growth is inhibited by bodily processes, however for reasons not yet clearly understood, the growth of cancer cells is not controlled in the same fashion.
Eysenck (1993) states that the medical researchers and practitioners of previous centuries, such as those referred to earlier, proposed psychological factors in the genesis of cancer and based these speculations on careful, systematic observations. Medicine during those centuries had not yet developed the statistical and experimental approaches which later gained ascendancy along with the ‘germ theory’ of disease. From the time of those mentioned reports until the 1950’s, no medical research was focused on the possibility of the psychology-cancer link. As medical research was then entering its scientific age, the methodologies of the past era of observers were not perceived as scientific, and were therefore ignored. Interest in this hypothesis re-emerged around 1950 due to the research work of Miller and Jones, who noted extreme emotional distress before the onset of chronic myelocytic leukaemia in six patients (Eysenck 1994). Subsequent research in the late 1950's, including work conducted by Miller, Greene, and Le Shan reported that the occurrence of cancer was often preceded by personal loss; and that depression was a common precursor of cancer (cited in Eysenck 1994). Much of the research addressing this question during the 1950's and early 1960's was based on psychoanalytic notions of personality structure, and much of it suffered from poor methodology, eg. the absence of control groups; ad hoc and subjective personality assessments; at times not distinguishing between different types of cancers, etc.

Despite these limitations, and the use psychoanalytic notions which Eysenck (1994) states simply confused the issues, this particular generation of research in the main supported the observations of the pre-scientific medical observers. For example, the following conclusions were drawn from such research:-

“women with breast cancer were found to share a ‘masochistic character structure’; the ‘cancer personality’ was described as being characterized by more defensiveness, higher anxiety levels, more presentation of serenity while inside they were experiencing significant distress; depression following the separation from a key object or goal was found to be linked to lymphomia or leukaemia; and a life pattern of misfortune, including poor childhood experiences, leading to
intense relationship difficulties and subsequent relationship losses with a period of repressed extreme despair; inability to express hostility on one’s own behalf; feelings of despair; being over co-operative, appeasing, unassertive, over patient, avoiding conflict; using suppression and denial as coping mechanisms; self sacrificing, rigid, predisposed to experience hopelessness and depression” (Eysenck 1994).

These early studies, while being of limited use because of the above mentioned methodological problems, nevertheless served as a transition from the pre-scientific observational studies of the previous two centuries.

A common observation from this generation of studies was that the cancer prone personality could be characterised as highly repressed. Kreitler, Chaitchik & Kreitler (1994) state that the role of emotional repression in the causation of cancer became one of the main cornerstones of this field of inquiry. However, in challenging the role of repression in the causation of cancer, they suggest that most of the observations have been made on people already suffering from cancer. On the basis of studying 98 women, 40 of which were healthy, 32 with breast cancer and 26 who underwent surgery unrelated to cancer, Kreitler et al (1994) concluded that emotional repression was the result of the cancer diagnosis, not the precursor. The researchers administered relevant psychometric tests both before and after surgery- the diagnosis was not known before the surgery, and became known to the first two groups of women after the surgery. Before their operations, the three groups of women did not differ in their levels of repression, anxiety and defensiveness; however after the surgery and knowledge of the diagnosis, the cancer group showed higher defensiveness and repression compared to the non cancer groups. As such, any research which obtains high repression scores from a cancer group would be unable to confidently attribute the cancer to the level of repression, as it appears from Kreitler et al's (1993) study, that the high level of repression is in fact a result of the emotional trauma caused by the cancer diagnosis. Obviously, a very different type of methodology is required to confidently pose repression as a causal agent in cancer. Prospective studies, whereby healthy people are evaluated at one point in time and re-
evaluated at another, with psychometric scores associated with health outcomes, is the most valuable type of methodology- such studies will be described shortly.

Amelang (1997) states that over the last three decades research into psychosocial contributors to cancer has focused on two possibilities

i) the loss-depression hypothesis, and ii) the ‘cancer prone personality’.

The personality characteristics described as being significant in the causation of cancer from the research of this period are described in two categories (Eysenck 1994 p169):

1) **Suppression of emotions** of fear and anger, and suppression of the behaviours appropriate to these emotions, eg. assertiveness, aggression, confidence, dominance, selfishness, and

2) **inappropriate coping mechanisms**, leading to failure, feelings of hopelessness and helplessness and finally depression and despair.

The modern period of cancer prone personality research, utilizing better controlled studies and more scientific rigour began, according to Eysenck (1994) with the research by Scottish oncologist, David Kissen. He conducted several controlled studies testing the proposed relationship between cancer and personality. Eysenck (1990) states that during the late 1950’s it had begun to occur to him that while cigarette smoking may or may not have a causal role to play in cancer, other factors like personality may be at least equally important. After researching the link between cancer and personality dimensions in 1960, Eysenck joined Kissen in conducting one such study of male cigarette smokers and the development of lung cancer (Kissen & Eysenck 1962), concentrating on the first category as mentioned above, ie. the suppression of emotions. Kissen’s research was cut short by his untimely death in the late 1960’s. Their research was aided by the Tobacco Research Council (itself ultimately funded by the tobacco industry).

The key features of Kissen and Eysenck’s (1962) empirical work, which marked the turning point in the scientific methodology of the research, included:- the testing of a theory informed specific hypothesis; the use of a well matched control group; a double
blind procedure; the use of an objective personality measuring device rather than subjective psychoanalytic devices; and the use of proper statistical analysis.

The study on which Kissen and Eysenck (1962) collaborated involved research with 239 male patients who attended the former’s chest clinics with a variety of chest disorders, including possible lung cancer. Based on his clinical experience, Kissen hypothesised that patients who had contracted lung cancer had a diminished outlet for emotional discharge compared to non-lung cancer patients. That is, he suggested a causal link between the amount of emotional discharge or expression characteristic of some personalities (high emotional discharge accompanying personalities characterized as neurotic) and subsequent lung cancer. As such, people who scored high on the neuroticism dimension, thereby freer in expressing their distressed emotions, were expected to be less susceptible to cancer. On the other hand, patients who were assessed as being low in neuroticism and therefore did not have a high rate of emotional discharge, were expected to be more likely to contract cancer.

In this study, Kissen interviewed all of the patients attending his lung clinics, generally within the week following their admission- none of the patients at this stage had yet received a diagnosis from Kissen or other physicians, even if some of the patients may have known of a diagnosis (eg. pneumonia or bronchitis). In most cases, the physician or surgeon had not yet confirmed a diagnosis, especially when cancer was considered a possibility. Acting as the interviewer, Kissen issued each patient with a standardised personality measure, the Maudsley Personality Inventory (the M.P.I, a precursor to the Eysenck Personality Questionnaire), which they completed according to standard instructions. This 48 item tool, developed by Eysenck measured respondents on two personality dimensions, extroversion and neuroticism. An official medical diagnosis was made by Kissen after the administration of the M.P.I. Ignorant of the medical diagnosis, Eysenck scored and analysed the M.P.I results- Kissen was ignorant of the M.P.I results when he made his diagnosis. Those patients who were diagnosed to be suffering from cancer were designated as the experimental group (n=116), while those for whom cancer of any organ was ultimately excluded constituted the control group (n= 123). The study
concluded that the control (non cancer) group had scored much higher on the M.P.I measure of neuroticism (N) than did the cancer group of patients, ie. the patients with cancer scored much lower on neuroticism than did the non cancer patients. The chance of a low neuroticism (N) scorer developing cancer was six times greater than the chance of a high N scorer developing cancer (p<0.01). Additionally, lung cancer patients were found to be more extroverted than non cancer patients.

Eysenck (1994) states that Kissen repeated this study several times and repeatedly found the same results; in addition, many other researchers subsequently replicated these findings, extending the studies to other types of cancers, and ‘purifying’ the measure of suppression of neuroticism. As such, he suggests that there is sufficient empirical evidence to conclude that ‘cancer is correlated substantially with suppression of emotion’ (Eysenck 1994 p172). One can imagine that the tobacco industry would be somewhat pleased by these findings, as they could argue that tobacco was not the single cause of lung cancer, ie. a complex interactive effect was assumed to be occurring. However, perhaps to the disappointment of the industry, Eysenck never proposed or suggested that tobacco smoking does not appear harmful. He stated that after the publication of ‘Smoking, Health and Personality’ in 1965, the Tobacco Research Council decided to withdraw the research grants. Apparently the industry did not want to hear that cigarette smoking was at least implicated in cancer and CHD even as one of the interacting causal agents- his research had become a nuisance to the tobacco industry. (Eysenck 1990).

In relation to the second psychological trait which was proposed by the early psychosomatic researchers, ie. the use of inappropriate coping mechanisms, the first major modern work was conducted by Schmale and Iker (1971). These researchers hypothesised that women with high scores of ‘hopelessness potentials’ and/or an actual hopelessness reaction six months before the first sign of abnormal or suspicious pap smear result were more likely to be amongst the sample population who contracted cervical cancer. Interviews to assess the above mentioned psychological tendency were conducted prior to a medical diagnosis being made. Of a sample of 68 women, 28 were found to have cervical cancer and 40 were found to be free of cancer. On the basis of
their assessments of psychological factors, the researchers were able to predict the positive diagnosis of cancer in 19 of the 28 women who were later to receive that diagnosis, and 31 of the 40 non cancerous women were accurately predicted to be cancer free (there were 9 incorrect predictions for each of the categories). This result represented a 74% accuracy of predictions (p<0.001) on the basis of interviewer’s assessment of the subject’s coping style.

Eysenck (1994) cites six subsequent studies, mostly working with other types of cancers, which have reached similar conclusions. Faragher & Cooper (1990) have also focused on coping styles and the development of cancer-these researchers represent a different strain of British research than Eysenck’s.

Taken together, the above mentioned research, if not viewed as evidence establishing the link, is in the very least suggestive of a level of correlation between psychological factors and cancer. Each of the main findings have been replicated with various types of cancers by a range of researchers (Eysenck 1994). More recently, Mathews et al (2003) have demonstrated from a cross sectional study of over 5,000 subjects that subjects with multiple health problems were higher in the factor of ‘Emotional Lability’ when compared to healthy or subjects with just one health condition. They conclude that their research adds weight to the body of evidence demonstrating that neuroticism is a general risk factor for disease.

An outstanding criticism of research such as this, however, is that it has been carried out with samples which included people who already had contracted cancer, even if as yet undiagnosed. As such, there exists the possibility that the causal link between personality and cancer is not in the direction hypothesised (ie. from personality to cancer), but in reality may be from the other direction (i.e from cancer to personality). Could the contraction of cancer actually cause certain personality types, eg. suppression of difficult emotions, and certain coping strategies such as denial and avoidance?
Eysenck (1994 p 186) offers research conducted by C.B Thomas as evidence suggestive of the personality cancer link direction. Thomas began researching in the 1950’s, culminating in what is now one of the oldest continued studies which addresses this question. Using 14 personality measures obtained while the subjects were in medical school, the resulting profiles of 972 physicians were clustered into five groups using a two-stage cluster analysis procedure. (Schaffer, Graves, Swank & Pearson 1987).

Subjects were followed over a 30-year period to determine the cumulative survival rate (proportion of subjects remaining free of cancer) in each group. Statistically significant group differences in survival rate were found, with the group characterized by acting out and emotional expression having the most favorable curve (less than 1% developing cancer). Thomas and the subsequent researchers found that ‘those who were loners and suppressed their emotions ‘beneath a bland exterior’ had the highest risk of cancer; in fact, the loners were sixteen times more likely to develop cancer than those who gave vent to their emotions!’

Thomas was in fact intending to study CHD, not cancer, so the chance of her expectations influencing the outcomes is remote at best. Her results provide longitudinal support for the direction of the relationship between personality and cancer as suggested by Eysenck and various other psychological researchers.

The largest claims for the role of psycho-social factors in the causation of cancer and CHD in the last two decades have come from the research enterprise of Yugoslavian psychologist, Ronald Grossarth-Maticek with the assistance of the now deceased Hans Eysenck. Their claims will be examined in the next chapter and constitute the focus of the current study.
Chapter 2

The Reports of Grossarth-Maticek:

In attempting to address the question of causality between personality and chronic disease, it first needs to be noted that Eysenck and other like minded researchers are not proposing that personality is the single cause- in fact, they are suggesting that chronic diseases are multi variable in causation, however the role of psychological factors has been largely ignored in the 20th century. Their attempt is to redress the imbalance which they perceive medical research has developed by concentrating only on the physical risk factors. As Eysenck (1994) states “The term cause has a very definite meaning in science, and is clearly inappropriate here” (p.195).

Eysenck (1994) makes the point that the psychological line of enquiry needs to go beyond establishing correlational links between personality factors and chronic disease. In order to establish causal relationships in the proposed direction, researchers need to adopt more advanced methodologies than what have been reported in this paper so far. Specifically, intervention studies need to be conducted, demonstrating that the causation occurs in the direction proposed. By attempting to experimentally manipulate the variables, causation can be demonstrated in the following way:- by reversing the proposed causal chain via prophylactic therapy (designed to change the personality variables that are implicated), causation can be demonstrated when the intervention has prevented disease in a group of people assessed to be disease prone while still being disease free. In brief, the methodology involves assessing a group of people at point T1 and analysing their rates and causes of death at point T2. These type of prospective studies are considered to be the ‘gold standard’ of mind-cancer research (Eysenck 1994). In the early 1980’s Eysenck learnt of the reports of the prospective longitudinal work being conducted by a largely unknown Yugoslavian psychologist.
Enter Ronald Grossarth-Maticek:
Following Kissen's death in the late 1960’s, Eysenck (1991b) states that he searched for other oncologists with whom to continue the line of study he had conducted with Kissen. He was met with a negative reaction, and was unable to find any oncologists who were either willing to collaborate with him, or to allow him access to their patients in order to gather data. As a result, he set aside his interest in pursuing this type of research.

Grossarth-Maticek entered the psychosomatic literature in the early 1980’s, reporting on the longitudinal prospective studies which he had began in Yugoslavia in the late 1960’s and early 1970’s, addressing much the same questions which Eysenck and Kissen had studied (Grossarth-Maticek 1980). He later reported the results of a 10 year prospective study which he conducted in Heidleberg, West Germany, sampling around 20,000 subjects. As he was reporting such positive results, Eysenck sought him out and investigated the massive amount of data which had been accumulated in the course of the studies conducted in Yugoslavia and West Germany. In summary, the reported results of the 10 year follow up studies strongly suggested the following conclusions (Eysenck 1990):-

- there are personality characteristics which can be designated as high risk for cancer and CHD.
- these personality characteristics are measurable via a purposely designed inventory, the Interpersonal Reactions Inventory (IRI) and the Short IRI.
- according to scores on the IRI, a particular person’s risk of later developing cancer or CHD can be predicted with an accuracy rate of 81%.
- disease proneness according to personality characteristics is around 6 times more predictive of subsequent cancer and CHD than are any of the standard known physical risk factors taken together.
- there is a synergistic relationship between the standard known physical risk factors and personality in terms of subsequent cancer and CHD.
- highly stressed probands have higher mortality rates than non highly stressed probands, and are more likely to die of the diseases which their
personality profile would suggest they were more vulnerable to.

• conventional forms of behaviour therapy, focused on changing those facets of personality functioning which predispose one to cancer or CHD, can alter the said tendencies, and thereby act as a prophylactic against these diseases.

Eysenck (1990) states that at the time of their meeting, Grossarth-Maticek was a largely ‘defeated’ researcher. Many within the established cancer research arena suspected that his reported results were simply too good to be true. As such, he was informally and verbally accused of tampering with results- no such accusations were made in professional literature, thereby not allowing an opportunity to respond in a professional forum. Eysenck (1991b) gives details of some of the accusations as well as his responses. According to Eysenck (1990), Grossarth-Maticek was experiencing the effects of having upset the medical cancer research institution, the “Cancer Mafia” as it is has been referred to. Newspapers were warned away from printing his results by the established cancer authorities; the manuscripts which he sent to journals for publication were returned to him; young oncologists and epidemiologists who demonstrated an interest in his mind-cancer research were warned away from any involvement with him with threats to their careers; he was unable to obtain research grants in order to further analyze the results or to extend his research for more years. Eysenck states that after evaluating Grossarth-Maticek’s data, and investigating the claims of fraud, “I concluded that a terrible injustice was being done to a man of considerable integrity and honesty” (1990.p 178).

Eysenck & Grossarth-Maticek (1991) state that the traditional orthodox medical establishment has an overt hostility to concept of ‘holism’ implied by mind-cancer research, and to any formal incorporation of psychological factors such as personality, stress, or psychological treatment. In addition to these pressures mitigating against his research efforts, Eysenck (1991b) reports that there were problems of a methodological and statistical analysis nature with Grossarth-Maticek’s work. In weighing up the prospect of assisting Grossarth-Maticek, Eysenck decided that the value of the data base,
despite its problems, justified providing him with the supervision assistance required to salvage important findings from the ‘sinking ship’ of his research project.

Eysenck took a leading role in improving the methodology and statistical analysis of Grossarth-Maticek’s work post-data collection, extending the longitudinal research into the subsequent years. As Grossarth-Maticek had been ‘black-balled’ by the cancer research establishment on the publication of his 10 year follow up data (1972-1982), he was therefore unable to gain grants to enable further follow up work. Eysenck, in delivering a symposia on the origins of cancer and CHD (organized by the Reynolds Tobacco Company) suggested that the company may like to provide some funds to re-analyze Grossarth-Maticek’s data and to further extend the study from 1982-1987, making it a 15 year study. The source of funds becomes an important issue, even if only a peripheral one as Amelang (1991) casts doubt over Grossarth-Maticek’s results, suggesting that the studies could not have been conducted on these limited funds. He also doubts Grossarth-Maticek’s statement that he in part provided his own research funds. In reply, Grossarth-Maticek (in Eysenck 1991b. p312) states that

“..the prospective studies were supported by four German and one Swiss foundation. In addition, money was contributed from wealthy relatives impressed by the social value of the research. In addition, Reynolds supported the further investigation and mortality ascertainment of the Heidelberg samples from 1982 to 1986”.

The Reynolds Tobacco company agreed to provide some of the required research funds, and a team of eminent scientists were formed to conduct the follow up study. If the combined efforts of Eysenck (Professor of Psychiatry and Psychology at the University of London), Dr. Heller (Statistical Institute of the University of Karlsruhe), Professor Charles. D Spielberger (Centre for Research in Behavioural Medicine and Community Psychology, University of South Florida), and Dr. van der Ploeg (Department of Medical Psychology, University of Leiden, The Netherlands) found results at the 15 year follow up which were consistent with Grossarth-Maticek’s reports of the 10 year follow up, then
the possibility of data manipulation or substitution would be considered extremely minimal, at least by Eysenck. The data had now become freely available to all critics, and had been re-analyzed by some- the collection and interpretation of death certificates was supervised independently by a member of the Karlsruhe Institute of Statistics; a random sample of the paid interviewers who had gathered the original data were interviewed.

Grossarth-Maticek and Eysenck first published the results of the 15 year follow up work in 1989, and over the next eight years they co-authored around 15 articles, describing the ongoing analysis of the population samples which Grossarth-Maticek began working with 15 years earlier. Their publications discuss the findings of the research, addressing questions of:- assessment of the ‘cancer personality’ and the ‘CHD personality’; the prediction of cancer and CHD on the basis of personality assessments; the various effects of smoking, alcohol, and caffeine consumption on the development of these diseases; the effects of rigorous physical activity, continued and discontinued in older ages; the effects of prophylactic behaviour therapy on the mortality rates of people who 15 years earlier were assessed to be at risk of these diseases; the comparative effects of behaviour therapy, psycho-dynamic therapy, and conventional medical therapy on the course of the disease for people suffering from terminal advanced cancers.

Assessment of the Cancer and the CHD Personality:
As stated above, the value of the results from prospective studies is based on the assertions that:-

i) there is in fact such a thing as a ‘cancer personality’ and ‘CHD personality’, and

ii) it is possible to assess and measure these personality characteristics.

The prospective studies themselves are where the bulk of the evidence for the above assertions are found. Eysenck (1991b p221) states that the characteristics most associated in the literature with cancer-proneness include the following:-
“appeasing, unassertive, over co-operative, over patient, harmony seeking and conflict avoiding, compliant, and defensive; as well as nice, ... unexpressive of negative emotions like anger, fear, anxiety”.

More refined, the two most frequently noted characteristics are:-

a) suppression of emotional expression, and denial of strong emotional reaction, and
b) failure to cope successfully with stress, and the reaction of giving up, linked with feelings of hopelessness and helplessness.

According to Eysenck (1991b), research has found positive correlations between CHD and the following characteristics:-

anger, hostility, aggression, and a generally contumacious attitude.

Dixon & Dixon (1991), drawing on Grossarth-Maticek & Eysenck’s work, suggest that the differences between the cancer and the CHD prone personality can be described by the following contrasts respectively:-

<table>
<thead>
<tr>
<th>Contrast</th>
<th>Cancer Personality</th>
<th>CHD Prone Personality</th>
</tr>
</thead>
<tbody>
<tr>
<td>Emotional repression</td>
<td>Emotional overindulgence</td>
<td></td>
</tr>
<tr>
<td>Excessive control of behaviour</td>
<td>Impatience</td>
<td></td>
</tr>
<tr>
<td>Over-niceness</td>
<td>Hostility</td>
<td></td>
</tr>
<tr>
<td>A depleting life stance</td>
<td>Unbounded ambition</td>
<td></td>
</tr>
<tr>
<td>Introversion</td>
<td>Extraversion</td>
<td></td>
</tr>
</tbody>
</table>

Eysenck & Grossarth-Maticek (1994) are proposing a diathesis-stress model of illness, whereby a person’s characteristics (acting as predispositions) interact with stressful events to produce the malady. The role and nature of stress will be discussed in more detail in a later section. It is sufficient, however, to state here that a ‘number of traumatic life events evoking chronic hoplessness’ was reported by Eysenck (1994) to have a roughly equivalent correlation with cancer in Grossarth-Maticek’s studies as did a highly predictive personality characteristic (rational and anti-emotional behaviour): .43 and .41 respectively. These results are reported on the basis of two large scale longitudinal studies conducted by Grossarth-Maticek.
Grossarth-Maticek’s Yugoslav Study:
The first cancer/CHD inventory developed by Grossarth-Maticek entailed subject’s responses being sorted into four Types. Eysenck (1988) states that beginning with his research in Yugoslavia in 1969, Grossarth-Maticek measured the personality of his probands in one of two ways. Firstly, he administered a series of short questionnaires designed to measure characteristics which he had conceptualized as being important in terms of proneness to cancer and CHD. Amongst the characteristics which he considered important and measured in the 88 question form, the ‘RGM Prospective Questionnaire’, were:- tendencies towards hopelessness and helplessness; rational and ‘anti emotional’ behaviour; and an absence of angry responses to traumatic life events. Also, a ‘semi-ipsative’ type of scale construction was developed by Grossarth-Maticek, resulting in the four types of personality as mentioned below, correlating with the above mentioned normative questionnaires. Eysenck (1994) states that the semi-ipsative is a somewhat clumsy kind of statistic, which he presumes Grossarth-Maticek developed primarily because it is of the kind which most appeals to the medical profession. As an alternative to the above, Grossarth-Maticek conducted intensive interviews with his probands, for the purpose of sorting them in to one of four Types. A parallel form was administered to relatives.

Eysenck (1990) states that Grossarth-Maticek also used a scale which he called the Rational-Anti-emotionality Scale (R-A Scale), which appears to measure the exact opposite of Eysenck’s Neuroticism scale. People who scored high on the R-A Scale (a major factor of cancer proneness) “refused to admit to feelings of fear and anxiety, they pretended that their life was governed entirely by rational motives, and that intellect was more important than emotion.” (Eysenck 1990, p 176).

The proposed four personality Types were as follows:-

Type 1- cancer prone.
Type 2- CHD prone.
Type 3- a mixed type, relatively immune to cancer and CHD
Type 4- the healthy, autonomous type.

(It is interesting to note here that Type 3 has been variously described in the following ways:- an hysterical type (Grossarth-Maticke & Eysenck 1995); a mixed type with psychopathic tendencies (Grossarth-Maticke & Eysenck 1991b).

Eysenck (1988) reports that in Grossarth-Maticke’s original Yugoslav study the probands were a random sample of mainly elderly people- the subjects were mostly the oldest person in every second house in a small Yugoslavian town of 14,000 people, Crvenka. He adds that in addition to these elderly probands, Grossarth-Maticke also included a number of subjects who were suggested to be ‘highly stressed’, with the total sample population of 1353 S's. (Eysenck (1988) comments that it was an unscientific error of Grossarth-Maticke to add the stressed group with the elderly group, committed long before he had any involvement in the data analysis. This mistake drew criticism, as the association found between personality, stress and illness may have been artificially strengthened by the inclusion. However, subsequent data re-analysis has shown that the demonstrated connection is actually stronger without the ‘stressed group’ of probands- Eysenck 1988.)

The probands, mostly aged between 50-65 years of age, were also assessed on a wide range of physical characteristics, such as:- height, weight, blood pressure; cigarette smoking was also assessed in terms of amounts smoked. Additional medical information was periodically gathered between 1969 and 1976. Amelang (1991p 233) cites Grossarth-Maticke (in 1977 and 1979 articles- not available in English) as stating that the, “criterion for inclusion in the sample was that the person in question was not suffering from any cardiovascular disease or from cancer, had no substantial subjective troubles and showed no symptoms characteristic of cancer or cardiovascular disease”.

28
Ten years later, a physician recorded the causes of deaths as stated on death certificates, and assessed the occurrence of different diseases in the surviving sample. The results at the 10 year follow up are shown in Table 1 (in Eysenck & Grossarth-Maticek 1989).

Table 1: Death from Cancer and CHD according to Personality.

<table>
<thead>
<tr>
<th>Type</th>
<th>Alive</th>
<th>Cancer</th>
<th>Infarct/Stroke</th>
<th>Other</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>Type 1</td>
<td>76 (25.1%)</td>
<td>140 (46.2%)</td>
<td>25 (8.3%)</td>
<td>62 (20.5%)</td>
<td>303</td>
</tr>
<tr>
<td>Cancer prone</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Type 2</td>
<td>101 (29.8%)</td>
<td>19 (5.6%)</td>
<td>99 (29.2%)</td>
<td>120 (35.4%)</td>
<td>339</td>
</tr>
<tr>
<td>CHD prone</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Type 3</td>
<td>129 (59.4%)</td>
<td>4 (1.8%)</td>
<td>20 (9.2%)</td>
<td>64 (29.5%)</td>
<td>217</td>
</tr>
<tr>
<td>Relat. Health</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Type 4</td>
<td>438 (90.9%)</td>
<td>3 (0.6%)</td>
<td>8 (1.7%)</td>
<td>33 (6.8%)</td>
<td>482</td>
</tr>
<tr>
<td>Opt. Health</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Unclassified</td>
<td>6</td>
<td>0</td>
<td>4</td>
<td>2</td>
<td>12</td>
</tr>
<tr>
<td>Total</td>
<td>750 (55.4%)</td>
<td>166 (12.3%)</td>
<td>156 (11.5%)</td>
<td>281 (20.8%)</td>
<td>1353</td>
</tr>
</tbody>
</table>

It can be seen from Table 1 that, according to the predictions, Type 1 probands (‘cancer personality’) tended to die of cancer rather than of CHD; and Type 2 probands (‘CHD personality’) tended to die of CHD rather than of cancer. Types 3 & 4 are less likely than either Type 1 or 2 to die of either of these causes. In the sample population, 117 men developed cancer, predominantly of the stomach, lung, rectum, and prostrate; while 87 women developed cancer, mostly of the breast, uterus and cervix. Using factor analysis it was established that the personality types were around six times more predictive of disease than were the conventional physical predictors, including smoking, cholesterol level, blood pressure, either singly or in combination.

Amelang (1991 p 233) argues in a critique that these results, rather than providing evidence in favour of the mind-disease link theory, are more likely to be the result of ‘...non typical but favourable conditions, methodological errors, and the specifics of data analysis’. In a response article written by Eysenck (1991b), Grossarth-Maticek presents
his own defense, acknowledging some of the methodological problems, but also emphasizing that the Yugoslav study was an early preliminary study—many of the methodological problems were addressed in the subsequent Heidelberg studies. The results of the Yugoslav Study are shown in Figure 1 and Table 2.

Figure removed due to copyright restrictions

Figure 1. Death from cancer and CHD according to personality type—Yugoslav Study (from Eysenck 1994 p.189)

Table 2. Determinants of cancer incidence—correlations of psychosocial factors and cancer (Eysenck 1994).

<table>
<thead>
<tr>
<th>Determinant</th>
<th>Correlation</th>
</tr>
</thead>
<tbody>
<tr>
<td>Number of traumatic life events evoking helplessness</td>
<td>$r = .43$</td>
</tr>
<tr>
<td>Number of traumatic life events evoking chronic excitement</td>
<td>$r = -.32$</td>
</tr>
<tr>
<td>Rational and anti-emotional behaviour</td>
<td>$r = .41$</td>
</tr>
<tr>
<td>Tendency towards self abnegation for the sake of harmonious relationships</td>
<td>$r = .18$</td>
</tr>
<tr>
<td>Lack of hypochondriases</td>
<td>$r = .17$</td>
</tr>
<tr>
<td>Absence of psycho pathological symptoms such as anxiety</td>
<td>$r = .008$</td>
</tr>
<tr>
<td>Lack of emotional contact</td>
<td>$r = .13$</td>
</tr>
</tbody>
</table>
It was these results which led some critics to suggest that Grossarth-Maticzek had tampered with his data, proposing that they were simply ‘too good to be true’.

**Grossarth-Maticzek’s Heidelberg Studies:**
The first Heidelberg study was a replication of the Yugoslav study, however using a cross-sectional analysis of a random sample of 1026 subjects, and attempting to eliminate some of the methodological problems that were detailed by critics. The results of the Heidelberg studies were published by Grossarth-Maticzek, Eysenck & Vetter (1988). The Heidelberg subjects were younger than those in the Yugoslav sample, with men and women aged between 40-60 years. In total, the Heidelberg studies utilized a sample of around 20,000 probands.

In the Heidelberg studies, Grossarth-Maticzek decided to examine two groups of probands:- in the first study, the probands were referred to as ‘normal’ on the basis of not experiencing undue stress; the sample in the second study was defined as the ‘stressed’ sample. They were selected as being severely stressed on the reports of the probands in the first Heidelberg study, being family members or friends who they knew experienced a great deal of stress (Eysenck 1991b). The two Heidelberg study samples were similar in age and sex distributions and smoking habits, differing only on the degree of stress experienced. The experience of stress is presumed to be one of the psychological variables which has a deleterious effect upon the immune system, thereby making the person more vulnerable to serious illnesses like cancer and CHD. Again, details about the proband’s personalities, levels of stress, smoking and drinking habits, cholesterol level, blood pressure and blood sugar were gathered through the use of interviews, questionnaires and physiological measurements. Probands were assigned scores on a 7 trait questionnaire and were allocated to one of the previously mentioned 4 personality types according to their questionnaire answers (Eysenck 1993). Data were collected by over one hundred trained interviewers who were at the time post graduate psychology students. Mortality information was gained by a physician obtaining details from proband’s death certificates.
The results for the normal sample are shown in Table 3. As can be seen, they replicate the results that were found in the Yugoslav study. Although fewer probands had died, being in a younger age group, again the outcome purports to demonstrate that Type 1 (cancer prone personalities) tend to die of cancer at a far higher rate than of CHD or any other causes; Type 2 probands (CHD prone personalities) tend to die of CHD at higher rates than they die of cancer; and Types 3 and 4 are relatively healthy, as predicted.

Table 3. Death from cancer and CHD according to personality type: Heidelberg 'normal' study (Eysenck & Grossarth-Maticek 1989).

<table>
<thead>
<tr>
<th></th>
<th>Alive</th>
<th>Cancer</th>
<th>Infarct/stroke</th>
<th>Other</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>Type 1</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Cancer prone</td>
<td>78 (71.6%)</td>
<td>19 (17.4%)</td>
<td>2 (1.8%)</td>
<td>10 (9.2%)</td>
<td>109</td>
</tr>
<tr>
<td>Type 2</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>CHD prone</td>
<td>109 (64.1%)</td>
<td>10 (5.9%)</td>
<td>23 (13.5%)</td>
<td>28 (16.5%)</td>
<td>170</td>
</tr>
<tr>
<td>Type 3</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Relat. Healthy</td>
<td>185 (98.4%)</td>
<td>0</td>
<td>1 (.5%)</td>
<td>2 (1.1%)</td>
<td>188</td>
</tr>
<tr>
<td>Type 4</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Opt. Health</td>
<td>387 (99%)</td>
<td>0</td>
<td>1 (0.3%)</td>
<td>3 (0.8%)</td>
<td>391</td>
</tr>
<tr>
<td>Unclassified</td>
<td>14</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>14</td>
</tr>
<tr>
<td>Total</td>
<td>773 (88.6%)</td>
<td>29 (3.3%)</td>
<td>27 (3.1%)</td>
<td>43 (4.9%)</td>
<td>872</td>
</tr>
</tbody>
</table>

In relation to the added variable of stress, the results purport to demonstrate a significantly higher rate of death for the ‘stressed’ sample when compared to the non stressed ‘normal’ sample, suggesting the important role of stress in mortality. Eighty nine percent of the non stressed group survived, where as only 54 % of the stressed group had survived by the time of follow up, a difference of 35% (Eysenck 1991b). Again, the results demonstrate (Table 4) a higher rate of death from cancer for those that have been assessed as having a cancer prone personality (Type 1), and a higher rate of death from CHD for those assessed as having a CHD prone personality (Type 2), and the relative immunity of Types 3 & 4 to these diseases.
Table 4. Death from cancer and CHD according to personality type: Heidelberg ‘stressed’
study (Eysenck & Grossarth-Matieck 1989).

<table>
<thead>
<tr>
<th>Personality Type</th>
<th>Alive</th>
<th>Cancer</th>
<th>Infarct/Stroke</th>
<th>Other</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>Type 1 Cancer prone</td>
<td>188 (38.4%)</td>
<td>188 (38.4%)</td>
<td>34 (7%)</td>
<td>79 (16.2%)</td>
<td>489</td>
</tr>
<tr>
<td>Type 2 CHD prone</td>
<td>148 (47.9%)</td>
<td>7 (2.3%)</td>
<td>86 (27.8%)</td>
<td>68 (22 %)</td>
<td>309</td>
</tr>
<tr>
<td>Type 3 Relat. Healthy</td>
<td>153 (92.7%)</td>
<td>4 (2.4%)</td>
<td>0</td>
<td>8 (4.8%)</td>
<td>165</td>
</tr>
<tr>
<td>Type 4 Opt. Health</td>
<td>71 (97.3%)</td>
<td>0</td>
<td>0</td>
<td>2 (2.7%)</td>
<td>73</td>
</tr>
<tr>
<td>Unclassified</td>
<td>6</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>6</td>
</tr>
<tr>
<td>Total</td>
<td>566 (19.1%)</td>
<td>199(19.1%)</td>
<td>120(11.5%)</td>
<td>157(15.1%)</td>
<td>1042</td>
</tr>
</tbody>
</table>

The reported results of the 15 year follow up are consistent with the 10 year follow up, suggesting the integrity of the original data and results, if one can have confidence in their authenticity (which Amelang (1991) doubts). As with the Yugoslav study, the relationships between the possible causal factors for the diseases were extensively analyzed by Eysenck, with him concluding that in the causation of cancer and CHD,

“personality is something like *six times* as important as these factors (ie. blood pressure, cholesterol, amount of smoking, singly or in combination) taken together”. (Eysenck & Grossarth-Matieck 1989, p35 - italics added).

Cigarette smoking, according to their analysis, only correlated with disease for people whose personalities were either cancer or CHD prone; it did not correlate with disease for Type 3 or 4 people, whose personality profile shows a relatively healthy prognosis.

A further conclusion of the statistical analysis was that rather being an additive effect with the various cancer or CHD risk factors, there was a *synergistic effect* between the risk factors. Referring to the studies mentioned here, Eysenck (1991b p222) states “The
data also indicated that the relationship between physical variables, like smoking, and psycho social variables, like personality type, was synergistic”. The results show that the only smokers who had a high amount of deaths from smoking were those of Type 1. Smokers of all other personality types and non smokers had equally negligible rates of cancer deaths. When the relative contribution of either smoking or personality to cancer were analyzed, it was concluded that cancer prone personality actually represents a stronger risk factor than does smoking. Six of the 735 Type 2,3 & 4 smokers died of lung cancer, while only 3 of the 850 Type 2,3 &4 non smokers died of lung cancer. The fact that these are not largely different figures suggests that smoking represents a risk factor primarily for people of Type 1 personalities, but not for all others. The same relationships were reported for CHD and smoking, with cigarettes only representing a genuine risk factor for people who are CHD prone according to their personality (Eysenck 1991b). This is not to say that smoking is not an important risk factor in the production of cancer and CHD, but that it becomes a more valid predictor of cancer and CHD only in conjunction with other risk factors, especially psychosocial ones. The data from both the Yugoslav and Heidelberg studies claim to demonstrate that there is an association between cancer and Type 1 personality statistically significant at the p=.0001 level. Figures 2 and 3 display the different results obtained for Grossarth-Maticek’s normal group versus the stressed group in the Heidelberg Study.

Figure removed due to copyright restrictions

Figure 2. Death from cancer and CHD according to personality type: Heidelberg 'normal' study (Eysenck & Grossarth-Maticek 1991b).
In a special edition of Psychological Inquiry, Eysenck’s (1991b) target article describes the above mentioned results and responds to criticisms from other researchers in this field concerning issues of methodology and statistical analysis. There is acknowledgement of some of the issues raised by the critics, however Eysenck (1993) makes the point that with any large scale longitudinal research (in this case with around 3 million data entries) there are going to be some mistakes made. He goes on to state that two critical and eminent researchers, statistician H. Vetter and epidemiologist van der Ploeg, re-analysed the data and came to the conclusion that the effect of any errors were indifferent to the hypothesis, ie. they did not favour the theory under study.

In relation to methodological errors, independent re-analysis of original data, this time by Professor C.D. Spielberger, again demonstrated that any mistakes made actually artificially loaded the results against the hypothesis, not in favour of it. In addition,
aspects of these results have been independently supported by other researchers (van der Ploeg, Kleijn, Mook, Hunge, Pieters & Leer 1989; Bleiker, van der Ploeg, Hendricks, Leer, Kleijn 1993), as well as researchers who have also concluded that Types 1 and 2 are disease prone while Type 4 are healthy (Quander-Blaznick 1991; Schmitz 1992; Amelang & Schmidt-Rathjens 1992). A re-analysis of the data by Vetter (1991), initially as one of the main critics of Grossarth-Maticek’s methodology, led him to withdraw his criticism and support the original conclusions as being valid.

In summary of the research findings reported by Eysenck & Grossarth-Maticek (1991b):

1) personality factors, concerned mainly with reaction to stress and coping mechanisms (autonomous, self regulating, hardy), play a powerful part in longevity.

2) Specific personality traits play a part in predisposing certain people to cancer

3) Specific personality traits, differing in many ways from the cancer prone type, predispose certain people (CHD prone) to coronary heart disease.

The results presented for the Yugoslav and Heidelberg studies to this point are purely correlational, and as such do not demonstrate a causal relationship between personality and disease. They are presented here to demonstrate the claims by Grossarth-Maticek and Eysenck that there is a cancer prone and a CHD prone personality; that they have been able to measure those personality characteristics; and that those measures have a statistically significant predictive power in terms of subsequent disease, in fact a greater predictive power than the known physical risk factors.

Independent research groups have demonstrated mixed support for Grossarth-Maticek’s claims. Amelang & Schmidt-Rathjens (1992) report a partially non confirmatory finding. Their study with 204 subjects suggests that while Type 1 & 2 scores taken together were able to discriminate between healthy and unhealthy groups, the typology was unable to discriminate between groups with cancer and groups with CHD. If the typology was able to accurately predict the incidence of cancer and CHD, one would expect the measurement tool to be able to distinguish between subjects currently experiencing
cancer and subjects currently experiencing CHD- the Interpersonal Reactions Inventory was found to be incapable of making this distinction. Amelang et al (1992) conclude that in their sample there was no evidence of a dichotomy between cancer and CHD personality as measured by Grossarth-Matichek’s inventory items. Amelang’s research will be further examined in a later chapter. Dixon & Dixon’s (1991) research with a population of 294 professional women does, however, support the dichotomy proposed by Grossarth-Matichek & Eysenck. Schmitz (1992) reports that from a study of 192 subjects, it was found that subjects assessed by Grossarth-Matichek & Eysenck’s assessment tool as being cancer or CHD prone utilized coping strategies described as ‘emotion-oriented’ and ‘avoidance-oriented’. Those subjects assessed as being health prone utilized ‘task-oriented’ coping behaviours. In terms of subject’s reactions to stressful events, Schmitz (1992) found that cancer prone people reacted with ‘depressive reactions’ and ‘relying on significant others’, while CHD prone people responded with ‘depressive reactions’ and ‘aggressive-critical behaviour’. Health prone subjects responded to stressful situations with ‘positive appraisal’, ‘acceptance’, ‘revision of expectations’, ‘adjustment’, and ‘hope for change’. Ghorbani, Watson & Morris (2000) tested 94 Iranian managers with the IRI as well as other measures of stress and psychopathology. They concluded that their results demonstrated support for the relationships between Grossarth-Matichek’s constructs and other measures of psychopathology, suggesting a cross cultural relevance. Correa, del Paso (2004) report from a study in which they tested the convergent and divergent validity of Grossarth-Matichek’s personality typology through an analysis of the IRI types with other hypothesized related psychological constructs and reports of somatic symptoms. Using a university student sample, they found that Type 1 and Type 2 scores were positively related to measures of stress and negative affect; Type 2 scores correlated with measures of anger, hostility and aggression; Type 1 scores were related to a learned helplessness attributional style and to lower social support; Type 3 scores correlated with histrionic personality measures; Types 1 & 2 were associated with different categories of somatic symptoms; while the health prone Type 4 scores were negatively associated with stress, negative affect, anger-hostility-aggression, the presence of somatic symptoms, and a learned helplessness attributional style. Finally, they report that there was not an ability
of Type 1 and 2 scores to differentially predict illness, although they were both equally associated with somatic symptoms. As such, the empirical evidence does suggest some level of support for some of Grossarth-Maticek’s claims.

Grossarth-Maticek's Therapeutic Intervention Studies:
This chapter has reviewed the reports of Grossarth-Maticek and Eysenck in favour of there being at least a correlational relationship between psychosocial factors and cancer and CHD. The current section will discuss the therapeutic intervention studies conducted by Grossarth-Maticek in Heidelberg, reported at the 13 year follow up in conjunction with Eysenck (Eysenck & Grossarth-Maticek 1991). As stated earlier, these researchers do not suggest that such psychosocial factors are the single cause of cancer and CHD, but discuss them in terms of being one of the important features of a multi factorial chain of causal events. The evidence in support of this contention is derived from another aspect of Grossarth-Maticek’s Heidelberg studies, wherein he compared the morbidity rates of sub samples of people who received different therapeutic approaches designed to modify the relevant personality characteristics. The various therapeutic conditions are described by Eysenck & Grossarth-Maticek (1991) as being:-

Study 1: Extended Individual Therapy
Study 2: Group Therapy
Study 3: Bibliotherapy
Study 7: Freudian Therapy

An hypothesized outcome of the intervention studies was that the personality characteristics found to be relevant to cancer and CHD could be modified through a variant of behaviour therapy, as implemented in Study 1 (titled ‘Creative Novation Behaviour Therapy’, and/or ‘Autonomy Training’ by Eysenck & Grossarth-Maticek 1991). More importantly, it was hypothesized that modifications of these characteristic behaviours would, over the course of subsequent years, produce a decrease in the actual mortality rate from cancer and CHD in probands who had been assessed as susceptible to these conditions according to their personality profiles. For example, modifying the
personality characteristics which demonstrated a correlation with cancer (ie. denial of strong negative emotions, use of inappropriate coping styles) in probands who are assessed to be high on those traits, would lead to statistically significant fewer deaths from cancer when compared to a matched control group of Type 1 cancer prone probands who received no such treatment. An equivalent effect for the therapeutic effects of behaviour therapy with CHD prone probands was hypothesized.

In researching the hypothesized benefits of behaviour therapy, the attempt was to ascertain if changes in the proposed psychological factors (independent variable) would produce changes in the causes and rates of death (dependent variable). Such an outcome would demonstrate the causal role which psychological variables play in the development of cancer and CHD.

The Intervention Studies were conducted as aspects of Grossarth-Maticek’s 1972 -73 Heidelberg studies, which utilized two sub samples, the ‘normal’ group and the ‘stressed’ group (as detailed in an earlier section). One hundred and ninety two subjects were selected from the ‘stressed’ group of the larger sample population of 2449 subjects, as probands for the intervention study. For the purposes of comparison, they were allocated into pairs that were matched so as to be similar in age, sex, degrees of stress, intensity of cigarette smoking, blood pressure, blood sugar and cholesterol- probands who were suffering from heart infarct, cancer, stroke, or any other severe chronic disease were excluded from the study. Subjects were sorted into the personality typology as already described. The pairs of Type 1 (cancer prone) and Type 2 (CHD prone) were offered psychotherapy, resulting in 100 pairs of Type 1 and 92 pairs of Type 2 accepting the offer. Probands were then randomly assigned to either the treatment group (n= 192) or the control group. Between 1972 and 1974 psychological and medical data were obtained from the probands, eg. cholesterol, blood sugar and blood pressure measures were taken three to four times prior to therapy, 1-3 months apart.

In Study 1 the intervention constituted 20-30 hours of Creative Novation Therapy conducted by Grossarth-Maticek over the course of 6 months- Eysenck (1987) describes
Creative Novation Therapy as being a type of cognitive behaviour therapy which was developed by blending elements of Wolpe’s method of desensitization, Beck’s cognitive therapy, and Lazarus’ method of teaching coping strategies. The aim was to modify the proposed disease promoting personality characteristics, i.e. it aimed at encouraging the expression of emotions, and relieving depression and helplessness. At the completion of the course of psychotherapy, the set of physiological measures were again taken, with at least two measures of blood pressure, blood cholesterol and blood sugar. Probands were again psychologically assessed in the period 6-12 months after the completion of the therapy in order to measure their membership to one of Grossarth-Maticek’s typology, and the extent to which that type was expressed. The probands were contacted by research assistants 13 years later, beginning with a phone call to determine if the proband was alive or dead; followed up with a visit to the proband or his/her surviving family, and finally a search of the death certificate to ascertain cause of death. This follow up work was independently supervised by Dr Heller (Statistical Institute of the University of Karlsruhe) who also checked all death certificates. In addition, incidence of cancer or CHD was determined by personal contact with the treating physician.

The results suggest that for the control group of Type 1 probands, the passage of 6-12 months between psychological assessments (self assessment questionnaires conducted in the pre and post treatment period) had produced no significant changes in their membership to the Type 1 profile. As predicted, Type 1 probands who had received the Creative Novation Behaviour Therapy demonstrated a change in their belongingness to Type 1 (from scores of 9.84 ± .76 to 5.7 ± 2.21) significant at a p<0.0001. In regards to CHD, the results also indicated no significant changes in the assessments for those Type 2 probands in the control group, and a significant difference (p<0.0001) for Type 2 probands who were in the Therapy group, with changes in scores from 8.96± 1.19 to 4.87± 3.04. Table 5 shows the prophylactic effects of behaviour therapy on cancer prone and CHD prone probands after the 13 year period.
Table 5 Deaths and incidence of cancer and CHD in therapy and control groups over 13 year period: individual therapy.

<table>
<thead>
<tr>
<th>Type</th>
<th>Cancer</th>
<th>Other causes</th>
<th>Living</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>n</td>
<td>Deaths</td>
<td>Incidence</td>
</tr>
<tr>
<td></td>
<td></td>
<td>(%)</td>
<td>(%)</td>
</tr>
<tr>
<td><strong>Type 1</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Control group (no therapy)</td>
<td>50</td>
<td>16(32%)</td>
<td>21(42%)</td>
</tr>
<tr>
<td>Experiment. group (therapy)</td>
<td>50</td>
<td>0 (0%)</td>
<td>13(26%)</td>
</tr>
<tr>
<td>Total</td>
<td>100</td>
<td>16(16%)</td>
<td>34(34%)</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>CHD</th>
<th>Other causes</th>
<th>Living</th>
</tr>
</thead>
<tbody>
<tr>
<td>n</td>
<td>Deaths</td>
<td>Incidence</td>
</tr>
<tr>
<td></td>
<td>(%)</td>
<td>(%)</td>
</tr>
<tr>
<td><strong>Type 2</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Control group (no therapy)</td>
<td>46</td>
<td>16(34.8%)</td>
</tr>
<tr>
<td>Experiment. group (therapy)</td>
<td>46</td>
<td>9.3(6.5%)</td>
</tr>
<tr>
<td>Total</td>
<td>92</td>
<td>19(20.7%)</td>
</tr>
</tbody>
</table>

The results suggest that the effect of the behaviour therapy over the 13 years period was to prevent the deaths from cancer of a statistically significant amount of Type 1 cancer prone probands when compared with matched probands who did not receive the treatment; and to prevent the deaths from CHD of a statistically significant amount of Type 2 CHD prone probands when compared with matched probands who did not receive the treatment (“all statistical tests were are well outside the p=0.01 level.” Eysenck & Grossarth-Maticc 1991, p19). Of interest is also the amount of deaths of other causes which are significantly fewer in probands who had received the treatment condition as compared to those who did not receive the treatment. This is important in that Eysenck (1985) reports that the stated causes of death on death certificates are highly unreliable, suggesting that for this study the stated causes of deaths are less important than the actual incidence of deaths. Despite this issue, ‘ the overall difference in the proportions still alive between the control groups and the therapy groups does indicate the efficacy of the treatment’ (Eysenck & Grossarth-Maticc 1991. p.19)
Table 5 also indicates that although the effect of the treatment condition was to lessen the amount of deaths in probands, there was still quite a high proportion of probands who were experiencing cancer and CHD, but who had not died at the 13 year mark. The implication of the high incidence of cancer and CHD was that the behaviour therapy may have either prevented the diseases, or it may have delayed the onset of the disease for at least a 12 year period- the question of either prevention of death or the postponement of death can only be answered with further study after another substantial time period, ie. it is too early to know whether the true effect of the behaviour therapy has been prevention of death from those diseases or postponement- enough time now has to be given to allow deaths to occur in order to answer that question.

In Study 2, the Group Therapy condition was applied to one of two groups of probands who were again matched for age, sex, personality type (ie. Type 1 or 2) and smoking history, creating a number of 245 matched pairs, after 86 refusals of the offer for group therapy. The probands were recruited as part of the 1973 Heidelberg study, with the results being followed up after 7 years, from 1974-1981. Grossarth-Maticek conducted the group behaviour therapy, with groups containing around 20-25 participants who met together for several hours at a time for between 6 (minimum) to 12-15 (maximum) occasions. Members of the group decided when they felt they had benefited enough from the group to cease their involvement. After the 7 year period, the same data gathering procedure as with the Study 1 method was conducted.

As can be seen in Table 6, the reported results of the group behaviour therapy are consistent with those of individual behaviour therapy.
Table 6. Deaths and Incidence of Cancer and CHD in group therapy and control groups.

<table>
<thead>
<tr>
<th></th>
<th>Group Therapy Condition</th>
<th>Control Group</th>
</tr>
</thead>
<tbody>
<tr>
<td>N</td>
<td>245</td>
<td>245</td>
</tr>
<tr>
<td>Not contacted</td>
<td>6</td>
<td>11</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Deaths</td>
<td>Incidence</td>
</tr>
<tr>
<td>Cancer</td>
<td>18(7.5%)</td>
<td>75(31.9%)</td>
</tr>
<tr>
<td>CHD</td>
<td>10(4.2%)</td>
<td>29(12.3%)</td>
</tr>
<tr>
<td>Other causes of death</td>
<td>20(8.4%)</td>
<td>-</td>
</tr>
<tr>
<td>Living</td>
<td>191(79.9%)</td>
<td>56 (23.9%)</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Deaths</td>
<td>Incidence</td>
</tr>
<tr>
<td>Cancer</td>
<td>111(47.4%)</td>
<td>129 (55.8%)</td>
</tr>
<tr>
<td>CHD</td>
<td>36(15.4%)</td>
<td>45 (19.5%)</td>
</tr>
<tr>
<td>Other causes of death</td>
<td>33(14.1%)</td>
<td>-</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

The Mortality and Incidence rates are significantly higher (p=0.01) for probands who were in the control (no therapy) group, as compared to those who received group behaviour therapy (with a difference level less than p=0.01 significance for CHD)- deaths from other causes is also significantly different. Nearly 80% of the therapy group were still alive, while only 23.9% of the control group were still alive. Overall, these results were interpreted as supporting the conclusion of the Study 1 (Individual behaviour therapy) treatment condition, i.e “...demonstrating the value of behaviour therapy in preventing death from cancer and CHD, and in lowering the incidence of cancer and possibly from CHD”. (Eysenck & Grossarth-Maticek 1991, p. 20).

In Study 3, the Bibliotherapy treatment, another variation of the behaviour therapy condition was applied to 600 probands of either Type 1 or 2 personality profiles during the 1973 Heidelberg study. These probands were matched with a control group of 500 who received no therapy, matched for type, smoking, age, and sex. A group of 100 similarly matched probands were chosen to evaluate a placebo condition in which a printed statement was given to the probands, followed by a discussion of its application with the interviewer- this was conducted in a way similar to the treatment condition, where by a printed explanation of the meaning, aims and methods of Creative Novation Behaviour Therapy was given to each proband, followed by a 1 hour treatment during
which the statement was explained and discussed. In the treatment condition, after the proband had sufficient time to consider the treatment statement and apply it to their circumstances, s/he was given a further 3-5 hours with the therapist, attempting to tailor the principles specifically for the proband’s particular problems. For the probands in the placebo condition, the therapist provided standard psychoanalytic explanations and suggestions, which were not believed to share any of the essential factors for therapeutic change which the behaviour therapy was assumed to possess.

The results indicate that after 13 years (1973-1986) there are no significant differences between the control and placebo groups, leading Eysenck & Grossarth-Maticek (1991) to combine them and treat them as a single control group of n= 600. When this larger control group is compared with the treatment group, similar results to Study 1 and 2 are evident. In the larger control group 128 probands died of cancer and 176 died of CHD, whereas in the treatment group 27 died of cancer and 47 died of CHD- deaths from other causes were also again in favour of the treatment group, with 192 controls dying, compared to 115 probands of the treatment group. These results were again statistically significant, exceeding the p=0.01 level, this time also for CHD. The results for the Bibliotherapy condition were even more clear than those for the Group therapy condition, perhaps, as suggested by Eysenck & Grossarth-Maticek (1991) because the sample sizes were more than twice as large, and the follow up was conducted for 13 compared to 7 years, i.e. with more time the prophylactic effects of behaviourally oriented therapy become even more apparent and significant.

In Study 7 Eysenck & Grossarth-Maticek (1991) investigated whether psychoanalytic therapy, conducted by other therapists would have a similar effect to their style of behaviour therapy. Part of the rationale for conducting this study was to attempt to determine if the results of the behaviour therapy conditions could be ascribed to a treatment effect. Study 3 had investigated for a placebo effect, however the researchers concluded that the failure of the placebo group to differentiate itself from the control group did not constitute sufficient evidence to disprove the placebo hypothesis. In a methodology which they acknowledge is far from perfect, Eysenck & Grossarth-Maticek
(1991) report that during the series of prospective studies discussed so far, probands were asked if they were or recently had been in psychoanalytic treatment or a similar dynamic method of psychotherapy, distinguishing between those who had been in such treatment for less than 2 years, and those who had been in treatment for more than 2 years—none of them were treated for physical diseases and none were diagnosed with cancer or CHD. For the purposes of creating appropriate controls, matching procedures were adhered to as described with the other studies. In addition to the above mentioned treatment groups, a small number of probands had experienced therapy of a short term non-psychoanalytic nature. The study concluded that the no treatment group survived the best, followed by the discontinued (<2 years) treatment group, followed by the psychoanalytic continued treatment group which had the worst death and incidence scores for cancer and CHD. The different control groups showed no difference from each other, but they were significantly better off than the two psychoanalytically treated groups. Eysenck & Grossarh-Maticke (1991.p.28) conclude that

“The overall impression given by these studies must surely be that psychoanalysis and other similar psychotherapies have either a negative influence on survival, as compared with short term therapies which have little or no influence on survival, while behaviour therapy has a very positive influence on survival.”

In essence, they suggest that therapies which are perceived by the patients to increase their autonomy (eg. behaviour therapy) are related to lower mortality and incidence rates of cancer and CHD; while therapies perceived by patients as decreasing their autonomy (eg. psychoanalytically oriented therapies) were related to higher mortality and incidence rates.

The studies reported here are collectively considered by Eysenck & Grossarh-Maticke (1991) to constitute clear evidence of a causal link between personality and cancer and CHD in the direction which they hypothesised, ie. personality factors (in addition with other factors) → cancer/CHD (Eysenck 1994).
In summary, Grossarth-Maticzek claims to have demonstrated that his personality assessment and typology has predictive powers for cancer and CHD; that probands who have been assessed to be cancer or CHD prone are more likely to experience and/or die of those diseases than probands who have been assessed as not vulnerable to them; when such personality characteristics are treated as an independent variable, with the diseases Incidence and Mortality as the dependent variables, probands who receive therapy designed to alter those characteristics have significantly less incidence and deaths when compared to vulnerable probands who receive no such treatment. If one can have confidence in these reports, the ability to manipulate the independent variable in controlled studies (ie. vary the degree of vulnerability creating personality characteristics via effective therapy compared to no or ineffective therapy) must in the very least seem highly suggestive of the importance of psychological variables in the incidence and mortality rates of cancer and CHD.

Taking the research project a step further, an additional set of studies were conducted during the 1972-73 Heidelberg Grossarth-Maticzek research, referred by Eysenck & Grossarth-Maticzek (1991) as:-

Study 5: Therapy of terminal cancer patients, and
Study 6: Behaviour Therapy and Chemotherapy Compared.

In Study 5, Grossarth-Maticzek wanted to investigate whether behaviour therapy could be effective in prolonging the lives of people who were suffering from inoperable, terminal cancer. To this end, 24 pairs of cancer sufferers were formed, each pair matched in terms of type of cancer, progress of the cancer, type of treatment received, age and sex. The patients were then randomly assigned to either a control or behavioural treatment condition (provided individually by Grossarth-Maticzek), with length of survival time considered the dependent variable. The results indicated an average survival time for the behavioural therapy group of 5.07 years, and of 3.09 years for the control group (the differences were highly significant by t-test). Eysenck & Grossarth-Maticzek (1991) consider these results as evidence that behaviour therapy can prolong the lives of patients with terminal cancers. They point out that this result is consistent with the reports of
Spiegel, Bloom, Kraemer & Gottleib (1989) who conclude an even stronger finding in the same direction as a result of involvement in a therapeutic support group- participants survived post diagnosis on average nearly twice as long as similar patients who received standard medical treatment.

Study 6, reported in Eysenck & Grossarth-Maticek (1991) attempted to ascertain if behaviour therapy was as effective as standard medical chemotherapy for inoperable cancers. Eysenck and Grossarh-Maticek (1991) hypothesized that patients who received psychotherapy would have a longer survival time than patients who had not; there would be a synergistic effect on survival time when psychotherapy and chemotherapy were combined; and that different therapeutic interventions and concepts would have different effects on survival time. To this end, they gathered a sample of 100 women who were suffering from mammary carcinoma and visceral metastases. Initially, 129 women with breast cancer and visceral metastases were asked if they would like to receive psychotherapy in conjunction with the Doxorubicin (adriblastine and Adriamycin) combination chemotherapy which had been proposed for them as the standard medical treatment- numbers of patients refused both psychotherapy and/or chemotherapy options. A 2 by 2 experiment design was created with the remaining patients, whereby 25 women were placed in each of the following conditions:- psychotherapy/no psychotherapy: chemotherapy/no chemotherapy.

In relation to the psychotherapy condition, patients were randomly allocated to one of the following therapy modes:- 24 received Creative Novation behaviour therapy; 12 received depth psychotherapy; 14 received standard behaviour therapy (relaxation training and desensitisation). Patients who had accepted or refused the chemotherapy option were proportionally distributed across the different psychotherapy types. Fifty of the women who accepted chemotherapy were divided into pairs and matched on extent of cancer and medical treatment, age and social background- the same procedure was conducted for the 50 women who accepted psychotherapy.
The chemotherapy consisted of a range of combinations of drug agents administered in 3-4 weeks cycles, repeated between 4-9 times, with alterations during the course as per the standard treatment. The psychotherapy condition saw patients receiving 30 hours of one of the variants of psychotherapy as described.

The outcome of the study was measured in terms of the length of the time period between the detection of metastases or new recidivism and the interval between metastases and death of the patient, in combination constituting the total survival time. Multiple linear regression and analysis of variance or covariance were used in the statistical analysis to evaluate the relationship between the intervention variables (chemotherapy and psychotherapy) and the outcome variable (total survival time, changes in leukocyte concentration and lymphocyte percentage, and changes in psychosocial variables).

As Table 7 shows, the hypotheses under investigation were reported to have been supported. The mean survival time for all patients was 15.7 months after the diagnosis of metastases, with a maximum of 38 months and a minimum of 6 months. Patients who had rejected both psychotherapy and chemotherapy lived less than the average of all patients, with survival time being only 11.28 months. Patients who had received psychotherapy but no chemotherapy lived an average of 3.64 months longer than the no treatment group, surviving to 14.92 months. Patients who received chemotherapy only (no psychotherapy) lived 2.8 months longer than the no treatment group, with survival time being 14.08 months- this represented no statistically significant difference to the psychotherapy only group. Overall, cancer patients who had received psychotherapy had a significantly longer survival time than matched patients who had received no psychotherapy (p<0.001). Table 7 displays these results.
Table 7. Survival time in months according to type of therapy (Grossarth-Maticek & Eysenck 1989).

<table>
<thead>
<tr>
<th>Type of Therapy</th>
<th>n</th>
<th>Survival time In Months</th>
</tr>
</thead>
<tbody>
<tr>
<td>A</td>
<td></td>
<td></td>
</tr>
<tr>
<td>neither chemotherapy nor psychotherapy</td>
<td>25</td>
<td>11.28</td>
</tr>
<tr>
<td>psychotherapy</td>
<td>25</td>
<td>14.92</td>
</tr>
<tr>
<td>chemotherapy</td>
<td>25</td>
<td>14.08</td>
</tr>
<tr>
<td>both types of therapy</td>
<td>25</td>
<td>22.40</td>
</tr>
<tr>
<td>B</td>
<td></td>
<td></td>
</tr>
<tr>
<td>behaviour therapy</td>
<td>14</td>
<td>15.29</td>
</tr>
<tr>
<td>creative novation therapy</td>
<td>14</td>
<td>23.54</td>
</tr>
<tr>
<td>depth psychotherapy</td>
<td>12</td>
<td>12.83</td>
</tr>
<tr>
<td>Total</td>
<td>50</td>
<td></td>
</tr>
</tbody>
</table>

Also of interest is the result which suggests a synergistic as opposed to an additive effect on survival time when psychotherapy and chemotherapy are combined, with the interaction effect significant at the p<0.05 level. Patients who had received chemotherapy alone on average increased their survival time (above the mean survival time for no treatment at all) by 2.80 months. Psychotherapy alone increased the survival time by 3.64 months, suggesting that if the combination of psychotherapy and chemotherapy were merely additive, an average survival time when combining the two should result in 17.72 months. (ie. 11.28+2.80+3.64= 17.72). However, as Table 7 demonstrates, the mean survival time for psychotherapy and chemotherapy combined was in fact 22.40 months, 4.68 months above the additive value (p=0.05).
As can be seen from Table 7, there were significant differences in the effectiveness of the various psychotherapies, with Grossarth-Matickek’s Creative Novation behaviour therapy reportedly contributing to the longest survival time when compared to the other modes (p<0.001).

In an effort to measure physiological correlates of therapeutic change, lymphocyte production of patients who were receiving chemotherapy alone compared to those who were receiving chemo and psychotherapy together were taken. Chemotherapy patients who also received psychotherapy were measured to be producing a statistically significantly higher percentage of lymphocyte concentration over time (p<.01. Grossarth-Matickek & Eysenck 1989). The lymphocyte percentage of the total amount of leukocytes are taken as a measure of immune functioning. Chemotherapy patients who received no psychotherapy were measured to have a steady decrease in lymphocyte percentage under the chemotherapy condition. Patients who had psychotherapy in addition to chemotherapy also showed a rapid decrease in lymphocyte percentage after the beginning of chemotherapy, but thereafter a steady increase in the percentages to a level higher than the pre-treatment percentages, p<.01 (Grossarth-Matickek & Eysenck 1989).

Eysenck (1994) states that the information presented here constitutes a large body of evidence which must be considered at least highly suggestive of a causal relationship between psychological factors (personality related behaviour patterns), their alteration by means of appropriate therapy, and the vulnerability to contract cancer and CHD. He does, however, caution that Grossarth-Matickek’s results cannot be taken as proof beyond doubt of the main concepts (Eysenck 1991b). There is obviously a need for replication of the intervention studies, as there remains the possibility that the beneficial outcomes were the result of a charismatic therapist, ie. Grossarth-Matickek, and not of the therapy itself. There have already been some partial replications of Grossarth-Matickek’s work (eg. Schmitz 1992; Dixon and Dixon 1991); as well as several studies which are supportive of the main conclusions, eg. Speigel et al. (1989). Dixon & Dixon (1991) suggest that the controversy in health psychology is moving away from the question of whether a cancer
prone personality exists to what are its key dimensions, ie. depleting life events in early life, excess social conformity, repression of emotion, or depression?

In the target article of a special edition of Psychological Inquiry, Eysenck (1991) presents the major findings of his work with Grossarth-Maticzek, and responds to the comments of a series of invited critics. The general thrust of many of the critics is that the results are simply ‘too good to be true’. While acknowledging the validity of some of the criticisms, he suggests that several of the critics are stating relatively obvious and unimportant critiques of a methodological problems which in fact have little effect on the overall outcomes of the work- he shares the concerns which some critics have raised about methodology, however he asks the reader to consider the degree to which these problems can affect the overall findings, suggesting that they do not have an important influence on the conclusions. Some critics are making accusations and innuendoes of data tampering- these are dismissed by Eysenck (1991b) with reference to the level of independent supervision at the later follow up reviews. In an exasperated tone, Eysenck (1991b) wonders how much proof is required for the acceptance of Grossarth-Maticzek’s conclusions? Other researchers, eg. Spiegel et al (1989) have found even more positive results from psychosocial interventions with cancer, again suggesting the role of psychological variables in causation, however the establishment has not reacted with such hostility to their work. Eysenck (1989) states that epidemiological research on the role of cigarette smoking in the causation of cancer and CHD is substantially more methodologically flawed than Grossarth-Maticzek’s work, however the relationship between smoking and cancer has become one of the ‘sacred cows’ of the cancer research establishment.

In summary, Eysenck (1991b) suggests that the level of hostility and resistance to Grossarth-Maticzek’s work can only be understood in social, psychological and political terms, not scientific ones. He states that further replication studies as the next step in providing more evidence of the theory in question, and the validity of Grossarth-Maticzek’s work. In one of his last written comments on the topic before his death in
September 1997, Eysenck (1995, p223) stated “The medical profession is fighting a rearguard battle to deny psychology its rightful place in this vitally important exercise.”

The role of cigarette consumption:
An equally valid concern is the role which tobacco industry funds play in such research, especially when one reviews the conclusions promoted by Eysenck and Grossarth-Matichek. Cigarette smoking is generally viewed as one of the major causal factors for both cancer and CHD, so any discussion of psycho-social factors as causal agents begins to challenge the legitimacy of the cigarette and cancer/CHD relationship. Obviously the tobacco industry will have a large interest in the nature of this discussion.

Eysenck (1995) and Grossarth-Matichek (1989) question the role of cigarettes in the causation of cancer and CHD. Eysenck (1995) states that in the USA the Centre for Disease Control and Prevention estimated that 418,000 deaths were caused by tobacco in 1990. He suggests, however, that this is not a scientifically meaningful statement for the following reasons:- only one in ten of all heavy smokers die of lung cancer (therefore smoking is not a necessary or sufficient cause of lung cancer); the lung cancer generally develops around 30 years after the alleged cause (ie. a great deal of time elapses between ‘cause’ and ‘effect’); and there are many other demonstrated risk factors including heredity, poor diet, drinking, stress/personality factors. Grossarth-Matichek & Eysenck’s claims suggest that all of the mentioned risk factors interact with cigarettes in complex and synergistic ways. Other research cited in Eysenck (1995) has concluded that cigarette smoking is not a risk factor for heart disease (Seltzer in Eysenck 1995); and under certain circumstances (depending on the personality type of the smoker) may actually protect the smoker from myocardial infarction (Friedman, Fireman, Petitti, Siegelaub, Ury, & Klatsky in Eysenck 1995). As previously mentioned, the 40 year longitudinal study of 1,300 medical students conducted by C.D Thomas concluded that ‘loners’ and those ‘who suppressed their emotions behind a bland exterior, were sixteen times as likely to develop cancer than those who gave vent to their emotions’ (a risk ratio of 16 to 1), while the risk ratio for cigarette smoking is 2.4 to 1 (Eysenck 1995. p222). Eysenck (1995) points out that to publish results such as those mentioned above does not equate with suggesting that
cigarette smoking is not a risk factor - rather, it does make the point that neither physical or psychological risk factors work independently from each other, but they operate in a synergistic fashion with personality acting as a **moderator** of the effects of smoking.

Sensitive to allegations of collusion with the tobacco industry, Eysenck (1990 p172) states,

> “Note that I have never stated that cigarette smoking does not appear to be causally related to cancer and coronary heart disease; to deny such a relationship would indeed be irresponsible and counter to the evidence. I have merely stated that the available evidence is insufficient to prove a causal relationship, and this I believe to be true”.

All of the above mentioned research may indeed lead one to question to prominence given to cigarette smoking by medical researchers, however it is really only Grossarth-Maticke’s reports that suggest psychosocial factors have the greatest causal role in cancer and CHD. Grossarth-Maticke & Eysenck (1989) report research which the former claims to have conducted, questioning whether the evil consequences of cigarette smoking repeated in the media has a role to play in creating a self-fulfilling prophecy by increasing the stress levels of those who continue to smoke.

In order to test the ‘self fulfilling’ hypothesis, Grossarth-Maticke, in the course of obtaining other information for his 1973 Heidelberg study, also asked a number of questions relating to proband’s beliefs about the effects of smoking on health, and the source of those beliefs. The questions included:-

> “Are you convinced that as a smoker you will be very likely to develop lung cancer, heart infarct, or some other smoking related disease?”

Those who answered ‘Yes’ were then asked “What caused you to develop this conviction?” They were to choose from the following three options:-
a) ‘My own experience, eg. the observation that smoking wasn’t doing me any good’,
b) ‘Public expositions and information, (Media) eg. newspaper articles concerning the medical harm done by smoking’, and
c) ‘Own experience as well as public information’.

The probands were not significantly different in terms of personality type, or intensity or duration of smoking. As Table 8 shows, probands who answered ‘No’ to the first question had similar survival rates to probands who answered ‘Yes’ and giving their own experiences, or a combination of those with public information as the source of their beliefs. Probands who answered ‘Yes’ and gave media/public information alone as the source of their belief showed a significantly lower rate of survival (by Chi square), with more deaths from cancer, CHD and other causes.

Table 8. Causes of death, beliefs and sources of beliefs (Grossarth-Maticek & Eysenck 1989).

<table>
<thead>
<tr>
<th>Death from:</th>
<th>n</th>
<th>Cancer</th>
<th>CHD</th>
<th>Other causes</th>
<th>Survival</th>
<th>%</th>
</tr>
</thead>
<tbody>
<tr>
<td>No, smoking will not harm me.</td>
<td>196</td>
<td>16</td>
<td>17</td>
<td>18</td>
<td>145</td>
<td>74</td>
</tr>
<tr>
<td>Yes, impression from:</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>a. own experience</td>
<td>134</td>
<td>15</td>
<td>15</td>
<td>16</td>
<td>88</td>
<td>66</td>
</tr>
<tr>
<td>b. media</td>
<td>72</td>
<td>22</td>
<td>24</td>
<td>17</td>
<td>9</td>
<td>12</td>
</tr>
<tr>
<td>c. both a. &amp; b.</td>
<td>126</td>
<td>10</td>
<td>11</td>
<td>15</td>
<td>90</td>
<td>71</td>
</tr>
</tbody>
</table>

These results would again tend to suggest a strong role for psychosocial factors (in this case, stress, or a self fulfilled prophecy caused by media information) in the multi variable causal chain of events. Combined with Grossarth-Maticek’s other claims, these results do suggest that cigarette smoking per se is not as the main cause of cancer and
CHD, but rather psychological experience is posited as a primary causal factor. One would expect the tobacco industry to be at least very pleased with these reports, if not to enthusiastically embrace them.

As Grossarth-Maticek & Eysenck’s claims are so large, it is important to contextualize them in the body of scientific research enterprises which are addressing the same issues. Are they alone in asserting such relationships? Are Grossarth-Maticek & Eysenck’s general conclusions supported by the conclusions reached by the community of researchers in Health Psychology and Psychoneuroimmunology? The next chapter will address these questions.
Chapter 3

**Psychoneuroimmunology & cancer:**

The term Psychoneuroimmunology (PNI) was coined in 1964 by Solomon & Moos (Kielcott-Glaser & Glaser in Ader, Felton & Cohen 1992). Following research conducted by Ader & Cohen in 1975, which demonstrated that the immune system could be classically conditioned, there was a resurgence of interest in the topic area. Behavioural Immunology is a related term, again exploring the notion that psychological factors (eg. stress) could impact negatively on the CNS, resulting in the adverse functioning of the immune system. ‘Psychoimmunology’ was reintroduced as an abbreviation of ‘psychoneuroimmunology’- all of the above mentioned terms are synonymous.

Ader, Cohen & Felton (1995. p99) state that

“Psychoneuroimmunology offers the concept of an integrated defense mechanism of neural, endocrine, immune, and behavioural aspects. The focus of scientific attention has shifted from separate entities of the immune response to an interactive immunology model. In the neural-immune concept the brain has specific, two-way pathways to the immune system. Brain lesions in the hypothalamus, for instance, affect immunological responses such as antibody production. The endocrine-immune concept addresses the hormonal flow from the pituitary gland. Adrenocortical steroids directly suppress immune reactions by reducing inflammation or by preventing the rejection of transplanted tissues. The behavioural-immune aspects are adaptations to external disease and stress factors mirrored in the immune changes.”

Such changes in conceptualization are viewed by these authors as representing a ‘paradigm shift’, suggesting that what have been considered separate systems are now better considered as components of “a single, integrated defense mechanism in which the interaction between the systems is as important to an understanding of adaptation as the
interaction within a system” (Ader et al 1995,p.105). For example, the nervous and immune system’s signal molecules, neurotransmitters and cytokines are found to be expressed and received by both systems, and as such their separate identities are considered by these authors to be a ‘misnomer’. Ader et al (1995) go on to state that in the 15 years prior to the 1990’s, the emergence of interest and research in PNI was seen, with the notion of a functionally autonomous immune system being challenged by this relatively new discipline. The study of behavioural-neural-endocrine-immune system interactions in PNI represent the piecing together of a puzzle, where the different pieces are only now being identified. Cohen & Herbert (1996,p113) suggest that PNI

“...provides psychologically and biologically plausible explanations for how psychological factors might influence immunity and immune system-mediated disease.. (further, it provides)... substantial evidence that factors such as stress, negative affect, clinical depression, social support, and repression/denial can influence both cellular and humoral indicators of immune status and function.”

While Eysenck, Grossarth-Maticek and other psycho-social researchers explored the psychological contribution to the psycho-neuroimmunology puzzle, the PNI researchers who have adopted this term to describe their own research efforts appear to be more focused on ‘developing the means to explore the relations of interactive signaling’ (Ader, 1995. p 143), or in Eysenck’s terms, studying the biological pathways of the hypothesized relationships between the interrelated systems and the relevant variables. In support of the basic thrust of PNI, Jemott & Locke’s (1984 p78) extensive literature review concluded that “the bulk of evidence favours the view that psychosocial variables may play a role in modulating the human immune response”.

The Role of Psychological Stress in PNI:
Cooper (2006) states that in the process of conducting a meta analytic review of the literature pertaining to job satisfaction and health outcomes (Faragher, Cass & Cooper 2005), he found between 20,000 and 30,000 studies which verified the role of
psychological stress in the production of ill-health- the particular type of ill-health experienced depends on the individual’s bodily predisposition.

Kendall & Harman (1995) state that stressors can be thought of as excessive levels of environmental challenges, threats or demands, considered in conjunction with the person’s perception of the stressors and their psychological resources for coping with them. This is a transactional definition of psychological stress wherein the stress experienced is presumed to be the result of the situational demands in relation to the individual’s perception of his/her ability to meet those demands. Eysenck (1988.p 454) emphasizes the point that

“stress cannot be objectively defined in terms of situations only, but always in relation to the reaction to the stress on the organism (strain). Stress is the objective situation which impinges on the individual; strain is the reaction produced in the individual by the externally imposed stress. The strain depends of course in part on the stress, but also on the specific type of stress used, and the personality of the individual reacting. This distinction is absolutely vital in understanding the importance of stress for physical disease, and unfortunately has been much neglected in the literature”.

An indicator of the importance of the type of stress being discussed is the different effects of chronic stress as compared to acute stress. Based on Grossarth-Matickek’s claims, and therefore going further than what other researchers appear to be willing to do, Eysenck (1988) suggests that chronic stress appears to ‘inoculate’ the person against cancer, while acute stress appears to increase the likelihood of the person developing carcinomas. There appear to be few other research groups willing to make this claim. Obviously, the two types of stress are qualitatively different, and it appears that Grossarth-Matickek and Eysenck are amongst the few willing to assert differing effects on the immune system. Eysenck (1987 p 91) states
“...cortisol is associated with acute stress, the development of learned helplessness and depression, and immunosuppression. Chronic stress, on the other hand, results in the tolerance of endogenous opiates and cortisol, and enhances ACTH, which in turn is related to neuroticism and introversion, and is known to increase immune reaction... It is acute stress which has immunosuppressive effects, and is associated with the development of cancer; chronic stress has been shown in several studies and experiments to have the opposite effect.”

The only recent support for this contention comes from a Danish research team led by Nielsen (2005). In an 18 year longitudinal study of 6500 women, looking for a possible link between stress and CHD, Nielsen (2005) reports to have found that daily stress (as opposed to stressful life events) was negatively correlated with breast cancer. That is, the more frequency and intensity of global stress reported by the women in 1981 and 1983, the less they were likely to develop a first time breast cancer during follow up compared to women who reported no stress at base line- in fact, they were 40% less likely to develop the condition. Nielsen (2005) suggests that lower estrogen production appears to result from prolonged activation of stress hormones, and estrogen levels are one of the main risk factors for breast cancer. There also appears to be a dose dependent relationship between intensity and frequency of stress and breast cancer 18 years later.

Sarason & Sarason (1993) provide the following information on the physiology of the organism’s response to environmental stressors - they suggest that the endocrine system will respond to a range of stressors such as biological factors like diseases and germs, and psychological experiences such as receiving an insult or engaging in combat.

The pituitary gland and part of the adrenal gland (the adrenal cortex) are involved in the body’s stress response. The brain is activated in response to the stressor, sending a message to the hypothalamus, which in turn:-

i) releases a corticotrophin releasing factor (CRF). On reaching the pituitary gland, the CRF causes the release of adrenocorticotropic hormone (ACTH). The ACTH is then released into the blood stream, flowing directly to the adrenal cortex in the adrenal gland,
where adrenal corticosteroids are formed and released. The corticosteroids have the effect of increasing fat and protein metabolism; increasing access to energy storage; and decreasing inflammation.

ii) the biological pathway initiated by the hypothalamus is via the sympathetic nervous system of the autonomic nervous system. The secretion of hormones from the adrenal glands (medulla) has the effect of increasing the heart rate; skin and digestive organs contract; blood vessels in the major muscles dilate; more oxygen is sent to the major muscles; glucose is released from the liver to fuel the increased energy needs. The medulla secretes epinephrine (which produces ACTH- Eysenck 1985) and norepinephrine (ie. adrenaline and noradrenline) which enhance the responses of the sympathetic nervous system, as described above (Kendall & Harman 1995).

Kendall & Harman (1995) state that these physiological responses to stress are not usually harmful to the organism because of the efforts of the parasympathetic nervous system which acts to calm down the level of arousal after the perceived demand has diminished. Further, they state that constitutional weaknesses or vulnerabilities are evident in the different types of particular autonomic responses- some people tend to be ‘blood pressure responders’ to stress, while others appear to be ‘skin or stomach responders’ (Kendall & Harman 1995).

The Immune System and Stress:
Kendall & Harman (1995) state that the immune system is composed largely of white blood cells called lymphocytes, of which there are several specific types located in the blood, spleen, thymus, and lymph nodes. Invading antigens (foreign organisms or substances) can be ‘trapped’ by macrophages, a particular type of large lymphocyte. T cells are alerted by the trapping macrophages, then multiply and present the antigen to yet another type of lymphocyte, Helper T cells, which then stimulate the proliferation of several other types of lymphocytes, including:-

- B lymphocytes, which produce anti bodies (immunoglobulins, Ig), attack antigens while circulating in the blood.
- Cytotoxic T- lymphocytes, which destroy antigens, primarily in the lymph nodes.
• natural killer cells, which directly attack foreign cells, especially virally infected cells and tumour cells.
• suppressor T cells, which ‘turn off’ the system.

These cells communicate with each other through products of the Helper T cells, substances called interleukin and interferon.

Ader et al (1995) state that although the precise biological pathways have not yet been firmly established, there is sufficient evidence from experimentally induced and naturally occurring stressful situations in animals and humans to conclude that behavioural and emotional states affect immune functioning. For example, the death of a family member, with associated depression, has revealed effects for both enumerative and functional measures of immunosuppression.

“Clinical depression is associated with an increased number of circulating neutrophils and a decreased number of NK cells, T and B lymphocytes and helper and suppressor/cytotoxic T cells.” (Ader et al. 1995. p 103).

Other losses, such as marital separation and divorce and the associated emotional response have been found to create humoral and cell mediated changes in immunity; other, less severe naturally occurring stressors such as examinations, chronic care giving behaviour, distress associated with environmental disasters, and marital discord all have been found to create transient immuno-impairment (Ader, et al 1995). However, it is as yet unclear how much stress and immuno-impairment is required to cause clinical symptoms of ill health.

Watkins (1997) states that an enormous amount of animal and human studies are able to describe the physiological responses to stress.

“Stressful situations are known to activate central autonomic nuclei and the sympathetic-adreno-medullary (SAM) axis, in addition to the HPAC axis...The
SAM axis is involved in the fight and flight response, and is associated with the feelings of anger and anxiety. Fighting involves noradrenaline and the central nucleas of the amygdala, while the flight response involves adrenaline and the basal nuclei of the amygdala. In contrast, the activation of the HPAC axis promotes submission and the emotions of defeat and despair, and is thought to involve the septohippocampal system. Typically, chronically stressful events promote vacillation between anger and despair, as individual's fight to gain control over their environment or give up, believing that they have no control. These cycles of anger and despair promote the production of a destructive range of catabolic hormones, injurious to a number of bodily systems, not just the immune system”. (Watkins 1997 .p 11)

Watkins (1997) suggests that while the research data on the immunosuppressive effects of depression are unclear, the effects of stress are very consistent across many different populations and stressors. Such environmental stressors as isolation, separation, overcrowding, disrupted dominance hierarchy, introduction of an aggressive intruder, restraint, cold, noise, and inescapable foot shock have been found to produce typical hormonal responses in regards to in vitro assessments of lymphocyte function, such as mitogen proliferation, cytokine production and cellular cytotoxicity, or non specific, in vivo assessments of immunity, such as antibody production. Watkins (1997) states that human and animal research makes it clear that the immunological consequences of stressful situations depend on factors such as the nature, duration, intensity and controlability of the stressor, as well as the organism’s perception of the situation.

As a result of the psychological energy required to adjust to situations which the organism perceives as stressful, the immune functioning as just described can be adversely affected, making the organism more vulnerable to disease. Stress which activates the parts of the brain as described above, leads to the secretion of hormones which eventually reduce immune functioning if the stressor is sustained. Kendall & Harman (1995) state that the current state of knowledge does not as yet support any one single explanation of the precise details of the process of hormones adversely affecting
immuno functioning. Further, they claim that stress has also been implicated in the impairment of another bodily defense against cancer, DNA repair. The healthy body can minimize the dangerous impact of exposure to carcinogens through DNA repair, unless something (perhaps the physiological effects of stress) interfere with the repair process.

Eysenck (1987) refers to cortisol as a ‘stress hormone’. The role of cortisol in the causation of cancer is that corticosteroids have been demonstrated to have immunosuppressive qualities, that is, to lead to a general weakening of the immune system- cortisol is the most widely researched intermediary between stress response and immuno-suppression (however others are worthy of consideration as well, eg. ACTH, endogenous opiates (Eysenck 1994). Ader et.al (1995. p 104) state that,

“it is not possible to attribute all immunological consequences of altered behavioural states to increased andrencortical steroids... opioid peptides and catecholamines... are also part of the response to stressful experiences and exert immunomodulatory effects”.

Ader et al (1995) state that ACTH-induced releases of adrenocortical steroids are the most conspicuous hormonal influence on immune functioning. Various research conclusions are cited by Eysenck (1987) to point to an inverse relationship between plasma corticosterone level and the spleen’s capacity to synthesize antibodies; and that in the healthy organism there are homeostatic regulatory mechanisms which counteract the suppressive qualities of corticosteroids. The natural killer cells help to detect and control malignancies, and suppress the rate of tumour growth. If the protecting properties of these cells are compromised by cortisol, then so is the immune system’s ability to destroy the carcinogens and to control cancerous tissues. Watkins (1997 p114), referring to the stress hormones, states:-

“Each has a powerful effect on immunity. However, the effects are complex with each hormone having different effects on immunity. While these hormones surge through the body the immune cells are hampered in their ability to function well.
Stress suppresses immune resistance, at least temporarily, and this may be a survival mechanism to attempt to conserve energy, with the body giving priority to the emergency at hand. However, if the stress is constant and intense, the immunosuppression may become long lasting.” (italics added)

This would suggest a disagreement with Eysenck’s (1987) view that chronic stress is not immuno-suppressive. It is interesting to note that as leaders in the PNI field of study, Ader et al (1995) make no use of Eysenck & Grossarth-Maticek’s work. In fact, they state that,

“the association between stressful life experiences and changes in immune function has not established a causal link between stress, immune function, and disease...This chain of events has not yet been definitely established”. (p.105).

This implies either a (most unlikely) lack of familiarity with Grossarth-Maticek and Eysenck’s research, or a rejection of their conclusions. It is true that the latter do present their conclusions with caution, stating that there is an urgent need for replication, however this hardly constitutes a sound reason for failing to mention Grossarth-Maticek’s claims. Eysenck & Grossarth-Maticek (1991) presented the results of their experimental intervention studies as providing evidence for a clear causal relationship between stress-personality and ill health. It appears that there may be a lack of acceptance of Grossarth-Maticek’s reports in the PNI arena.

Eysenck’s theory of causal links:
Eysenck (1985,1987, 1991b, 1994) advances a theory of a biological pathways which suggests the physical effects of psychological factors. His theory rests in part on the acceptance of the evidence provided so far by Grossarth-Maticek (as reported in Chapter 2), that there is a demonstrable link between personality and stress on one hand, and cancer on the other via the intermediary of the immune system; as well as other evidence provided by animal studies and psychoneuroimmunology studies.
In short, the pathway which Eysenck (1994) proposes for cancer is:

Stress-strain (ineffectual personality response to stress) $\rightarrow$ cortisol secretion $\rightarrow$ immunodepression $\rightarrow$ cancer growth $\rightarrow$ death.

Eysenck (1994, p 200) cites a list of references which provide summaries of the experimental evidence in support of these links from research in the field of psychoneuroimmunology. In a cautious note, however, he does suggest that this is not yet a proper theory, but rather is a set of suggested directions to explore for the purpose of establishing some of the intermediaries between behaviour and disease (Eysenck 1991).

The following is a sequential presentation of the state of the theory, deductions and findings from a variety of sources (Eysenck 1987,p99):

1) Malignant growths occur all the time in animals and humans, but are kept in check by the immune system. In support of the notion that cancer cells are more common than what has been previously thought, Welch (2006) cites autopsy studies which demonstrate that around 30% of all adults have some pathogenic evidence of thyroid cancer; around 40% of women in their 40’s show microscopic evidence of breast cancer; and more than 50% of older men have microscopic evidence of prostrate cancer. Welch (2006) states that in recent years, an increasing amount of pathology examinations have been undertaken for the purposes of early detection of cancer. As a result, smaller and smaller specimens are being examined and consequently a larger than expected amount of cancers are being detected- “a lot more than anyone ever thought existed” (Welch 2006: p2). The majority of these small cancerous cells either do not grow at all, while some become smaller with time, and others grow so slowly that they will not threaten the life of the person. As such, there is contemporary support for Eysenck’s (1987) contention that cancer cells are relatively common, even in ostensibly healthy bodies. That the immune system become less effective with age explains the rapid increase of carcinomas with advancing years.
2) The workings of the immune system are influenced and to some extent controlled by peptides and hormones. Corticosteroids such as cortisol act in an immunosuppressive fashion, and endogenous opiates may also be involved in mediating some of the immunosuppressive effects of stress. ACTH, on the other hand, may have an immuno-enhancing effect.

3) Uncontrollable stress (related to learned helplessness) is significantly related to the occurrence of disease and death, whereas controllable stress does not act in a similar fashion.

4) Chronic and acute stress also appear to be somewhat dissimilar in their relation to disease and death, with acute stress increasing the probability of malignant growths developing, whereas chronic stress, through a process of inoculation, may have the opposite effect.

5) The peptides and hormones mentioned as affecting the immune system also seem to be related to certain behaviour patterns. Thus ACTH is related to anxiety, neuroticism, and introversion; whereas cortisol is related to depression, hopelessness, and helplessness. This may explain the positive relationship between hopelessness/helplessness and cancer, and the negative relationship between anxiety, neuroticism and introversion on the one hand and cancer on the other. Eysenck (1985) suggests that Neuroticism, Introversion and Psychoticism appear to be negatively correlated with cancer and proposes the following possible explanations:- scorers high in N or P are more likely to have an emotional outlet for their distress; they are more likely also to experience elevated ongoing chronic stress, which appears to ‘inoculate’ the person against cancer; Ader et al (1992) have demonstrated that the immune system can be classically conditioned by Pavlovian procedures- introverts are more conditionable than extroverts (Eysenck 1985), so the possibility arises that “..through conditioning introverts may increase the protection afforded by the immune system against cancer” (p.551).

6) If there is a causal relationship between the personality traits just mentioned and the behaviour patterns on which they are built on the one hand, and the mediation of malignant growth through immunosuppression and immunoenhancement on the other, then alteration of these behaviour patterns through behaviour therapy should be able to reverse ongoing trends.
7) Creative Novation Behaviour Therapy, working along these lines, is claimed by Eysenck & Grossarth-Maticek (1991) to act in a prophylactic manner, preventing the occurrence of carcinomas, and prolonging the life of incurably ill cancer patients.

8) Eysenck & Grossarth-Maticek (1991) claim that by making uncontrollable stressors controllable, not only is there a significant decrease in cortisol levels but there is also a significant decrease in death rates.

In regards to the duration of the stressor, it appears that contrary to Eysenck’s (1987) contention that acute, short term stressful events can have a deleterious effect on the immune system. McEwan (1998) suggests that short term stressful events and associated negative emotions can actually help to activate the immune system. Kemeny (1993) cites research concerning the killer T-cell activity of method actors who were instructed to simulate sadness and happiness with improvised dialogues. Blood analyses revealed that contrary to expectations, intense simulated sad states were actually associated with increases in natural killer cells in the actor’s immune systems, not the reverse. A similar effect was found for actors simulating an intense happy state. In both types of states, increases in immune functioning were found within twenty minutes of the intense emotion, with killer T-cell levels returning to base line levels after the actors had been sitting quietly for half an hour. Pennebaker (in Kemeny 1993) found the same results when asking students to think about and reactivate intense negative feelings around past traumatic events- blood analyses found an enhancement in the measure of immune functioning studied.

Studying the short term effects of actual trauma outside of the laboratory, Kemeny (1993) found that the blood analyses of 19 people, taken 2- 4 hours after the devastating 1987 Las Angeles earthquake demonstrated the same effect. Not only had the subjects just experienced the trauma of a large earthquake, but were in fear of after shocks, or the ‘big one’ coming next. Again, they found that there was an increase in the number of natural killer cells immediately after experiencing intense negative emotions, on this occasion related to the earthquake. Blood analyses were conducted on the same individuals over
the next year in order to establish their base line levels, demonstrating the immune activating effects of the stressful event.

Kemeny (1993) concludes that there is sufficient evidence to assert a correlation between the individual’s experience of emotion per se and the number of natural killer cells. Short term emotions, whether they be happy, sad or stressful, appear to have a positive short term result on the immune system. She suggests that the immuno suppressive effects of negative emotions occur when the emotion is experienced for *prolonged* periods of time, for weeks or months, ie. chronic stress. The theory offered by Kemeny (1993) to explain this phenomenon proposes that,

> “the mechanisms that pump these killer cells into the bloodstream or increase their activity get depleted over time... it seems that emotions are important and have their adaptiveness when they are short term, when you experience them for a number of minutes, or maybe hours.” (Kemeny 1993 p 202).

Kemeny (1993) suggests that the immune system becomes activated as part of the fight or flight response as yet another adaptive aspect of that process.

> “While the fight or flight response mobilizes your ability to fight or get away from the threatening animal, the immune system might be protecting you from other kinds of organisms that are getting into your system as a result of that encounter”. (resulting from injuries such as bites, scratches, cuts, etc). (Kemeny 1993 p.200).

MeEwen (1998) confirms that the immune enhancing effects of adrenal secretion in response to acute stress can last for up to three to five days. As he puts it, “Acute stress has the effect of calling immune cells to their battle stations” (McEwen 1998 p. 176), however, the effect of stress is “completely different” when the stress is repeated, and the blood is depleted of fewer lymphocytes.
In summary, researchers have demonstrated that the immediate effects at the time of the emotion, either negative or positive, is an increase in the immune functioning.

If the negative emotion continues over time, the immune suppressive capacities of stressful experiences emerge. This explanation is in accordance with Seyle’s (1976) description of the General Adaptation Syndrome (GAS) in which three series of reactions to stress are proposed. The fight or flight response occurs in the first stage, referred to as the Alarm Reaction. During this phase the body is prepared by the endocrine system to resist the stressor in the manners described above, including heightened secretions of ACTH, adrenaline and cortisol. The intense arousal of the alarm reaction cannot be maintained by the body for very long- organisms that have been exposed to intense continuous stressors have died within hours or days. In stage two of the GAS, referred to as the Stage of Resistance, the body attempts to adjust to the continuing stressor. While the level of physiological arousal remains higher than base line levels, there is a decrease in arousal compared with the alarm reaction stage. During this stage, the body attempts to replenish the adrenal hormones which have been excessively released in the earlier stage. Seyle (1976) suggests that during this second stage, there is an impaired ability to respond to new stressors, with one result being an increased vulnerability to health problems and illnesses due to impaired immune functioning. Exhaustion is the final stage, in which prolonged physiological arousal due to severe long term or repeated exposure to stressors takes its toll on the organism by depleting the body’s reserves of energy until resistance is severely curtailed. The immune system is again further compromised during this phase as a feature of the exhaustion, leading to a heightened possibility of disease and physiological damage. It appears that the immune response is ‘worn out’, or depleted as a result of sustained stressors.

McEwen (1998 p. 172) states “Over weeks, months, or years, exposure to increased secretion of stress hormones can result in allostatic load and its pathophysiologic consequences.” Sharpley (2002) defines the allostatic load as the damage state which accrues to the organism as a result of having to adapt to chronic change. Anxiety is one of the psychological experiences which can add to this allostatic load. Sharpley (2002)
suggests a range of clinical and psychological symptoms apparent in people who are unable to “turn off” the allostatic mechanisms that are provoked by situations or cognitions in order to ward off threat. These include:

- Increased heart rate (with respondents typically endorsing anxiety scale items which state “my heart is always racing”).
- Increased blood pressure (“I feel a constant tightness in my head”).
- Muscle tension (“I feel tense all over”).
- Electrodermal responses (“my hands and feet are always wet”).
- Peristaltic processes (“I suffer from constipation/irritable bowel/have to urinate often”).

All of the above symptoms are common criteria for the diagnosis of anxiety disorders. A range of stress hormones are associated with these experiences, including cortisol.

The importance given to cortisol is described in terms of duration- in the short term, leading to moderate secretions only, cortisol is not thought of as highly dangerous to the immune system; however, repeated excessive secretions of cortisol released for sustained periods of time is when the physiological damage occurs. This view is supported by Kemeny (1993), Seyle (1976) and Fredrikson, Furst, Lekander, Rotstein, & Blomgren (1993), and research conducted by McEwen & Stellar (1993). Apart from the result recently reported by Nielsen (2005), Eysenck (1985) appears to be alone in suggesting that acute short term stressors damage the functioning of the immune system, and chronic stressors enhance it.

The neurological component of chronic stress:
Khalsa (1997) further describes in detail the neurological processes responsible for the immunosuppressive effects of stressful events. The limbic system, composed of the hippocampus, amygdala, hypothalamus, thalamus, and the pituitary gland, is described by Khalsa (1997 p.110) as the nexus of the mind/body connection as it “controls the emotions”. The limbic system links the brain to the endocrine system, the series of glands which secrete hormones and activate many other organs throughout the body.
“The hypothalamus is closely connected to the amaygdala, and helps to tell the body how to respond to various situations. But it does this only after the hippocampus, amaygdala, and neocortex have decided how important the situation is. The hypothalamus gives its messages to the pituitary gland, which relays the messages to the rest of the body (through its own hormones, and through “releasing factors”, which trigger other hormones)” Khalsa (1997 p.111)

In a situation which is perceived as a crisis, it is the hypothalamus that first ‘sends out orders’ for more adrenaline. The hypothalamus sends this message of crisis to the pituitary gland (‘the master endocrine gland’), which then instructs the other endocrine glands in the body what to do in terms of the production of hormones. The pituitary gland stimulates hormone release in the adrenal, the thyroid, the gonads and the mammaries.

Watkins (1997.p13) states that

“The central nucleus, through its extensive projections to the motor cortex, the lateral hypothalamus, and other brainstem autonomic nuclei, is then able to hijack all other neural pathways during an emotional emergency. Thus, the central nucleus of the amygdala acts as a command centre activating brainstem autonomic nuclei, neuroendocrine pathways and the motor cortex to initiate the appropriate behavioural response, be it avoidance (flight), readiness (fight), or playing dead (submission), and each one of these responses is underpinned by specific changes in heart rate, blood pressure, catecholamine and stress hormone production.”

Khalsa (1997) states that as cortisol remains in the system for much longer than does adrenaline, it has more opportunity to cause biological damage; whereas adrenaline is secreted quickly in a stressful situation and also dissipates quickly. Apart from acting in a fashion which suppresses the functioning of the immune system, cortisol has been identified as causing several other major neurological problems. Khalsa (1997) suggests
that cortisol is largely responsible for the death of large numbers of brain cells particularly in the limbic system, which controls the process leading to the release of cortisol. McEwen (1998) states that repeated stress affects brain function, especially in the hippocampus, which has high concentrations of cortisol receptors.

As stated by Khalsa (1997 p.8) “In moderate amounts, cortisol is not harmful. But when produced in excess, day after day- as a result of chronic unrelenting stress- this hormone is so toxic that it kills and injures brain cells by the billions.” McEwen (1998) states that repeated stress causes atrophy of the dendrites of pyramidal neurons in the CA3 region of the hippocampus through a mechanism involving both glucocorticoids and excitatory amino acid neurotransmitters released during and after stress. Confirming that the damage of stress is related to its duration, Sapolsky (1996) adds that this damage can be reversed if short lived, but stress endured over months or years can kill hippocampal neurons.

According to Khalsa (1997 p.39)

“There are 3 essential ways that stress destroys optimal function of the brain...First, when cortisol is released in a stressful situation, it inhibits the utilisation of blood sugar by the brain’s primary memory centre, the hippocampus.... Second, cortisol over production interferes with the function of the brain’s neurotransmitters.... Third, too much cortisol kills brain cells. This happens when cortisol disrupts normal brain cell metabolism, and causes excessive amounts of calcium to enter the brain. That excess of calcium eventually produces molecules called free radicals, which kill brain cells from within. Over long periods of time, excess cortisol can kill billions of brain cells this way”.

Khalsa (1997) is more interested in the neurological damage caused by excessive secretion of cortisol and the possibility that this is related to the onset of Alzheimer’s disease, rather than a concern with cancer. Seeman, Singer, Rowe, Horowitz & McEwen
(1997) report that elderly subjects who measured as having lower allostatic loads also had lower levels of cardiovascular disease, hypertension and diabetes, while they displayed higher levels of physical and mental functioning. Confirming Khalsa’s (1998) views detailed above, Seeman et al (1997) demonstrated that amongst elderly women measured as having high allostatic loads, increased cortisol secretion predicted a decline in memory function.

In addition to the immunosuppressive properties of cortisol and adrenaline, one by-product of free radical molecules is that they often contribute to cancer cells (Khalsa 1997 p.158). The free radicals in the brain also cause neurological degeneration. Sapolsky (1996) cites research which supports the contention that recurrent depression and post-traumatic stress disorders have been associated with atrophy of the hippocampus.

Apart from problems of poor memory and cognitive functioning caused by the brain damage resulting from excessive cortisol and free radical production, a major problem arises in that the part of the brain that is the most sensitive to this cortisol damage is the limbic system which governs the stress hormone process. Brain damage due to the toxic effects of cortisol is especially apparent in the hippocampus and the amygdala (Khalsa 1997 p.153). McEwen (1998 p. 175) states,

“Impairment of the hippocampus decreases the reliability and accuracy of contextual memories. This may exacerbate stress by preventing access to the information needed to decide that a situation is not a threat (Sapolsky 1990). The hippocampus also regulates the stress response and acts to inhibit the response of the HPA axis to stress”.

This neurological damage is referred to Khalsa (1997) as the “feedforward mechanism”.

“Normally, when too much cortisol is secreted, the brain senses the oversecretion, and stops further production of cortisol. This is the brain’s normal “feedback
mechanism”. Many years of damage by cortisol ruins the feedback mechanism, transforming it into the harmful feedforward mechanism.... (normally) when levels of cortisol rise to a certain degree- a “setpoint”- several areas of the brain tell the hypothalamus to turn off the cortisol producing mechanism. This is the proper feedback response. However, one of the areas of the brain that is most responsible for telling the hypothalamus to turn off cortisol production is the hippocampus. And the hippocampus... is the area that is most damaged by cortisol. The hippocampus is often so damaged in older people- who have lost about 20 to 25 % of their hippocampus cells- that it is unable to give the proper feedback to the hypothalamus. When that happens, the hypothalamus keeps pumping out the chemicals that cause cortisol oversecretion. This in turn causes even more damage to the hippocampus. And this of course causes even more cortisol production. Thus, a catch-22, “degenerative cascade” begins. And this cascade can be very difficult to stop. The net result is that many middle aged and older people simply can’t control their production of cortisol. Even when they are not under stress, they secrete high amounts of the hormone. The effects of this degenerative cascade can be anxiety, irritability, inability to relax, and difficulty sleeping- as well as memory and concentration impairment”. (Khalsa 1997 p 154-155).

The list of damaging effects of excessive cortisol include:-

- it deprives the brain of glucoes, its only source of energy
- it ‘wreaks havoc’ on the neurotransmitters in that norepinephrine, vital for maintaining a positive, happy mood is depleted by chronic stress
- as the brain physically deteriorates, it loses its ability to properly coordinate the hormone secreting endocrine glands. “When this happens you suffer declines in...immune function” (amongst other biological problems). (Khalsa 1997 p.9 italics added).

The failure to turn off the sympathetic activity after a stressful event has passed is a feature of age in laboratory animals, and there is limited evidence of this occurring in

“One speculation is that the allostatic load over a lifetime may cause the allostatic systems to wear out or become exhausted (Seeman & Robbins 1994). A vulnerable link in the regulation of the HPA axis and cognition is the hippocampal region. According to the “glucocorticoid-cascade hypothesis”, wear and tear on this region of the brain leads to dysregulation of the HPA axis and cognitive impairment... Recent data suggest that similar events may occur in humans (Lupien, Lecours, Lussier, Schwartz, Nair & Meaney 1994; Seeman et al 1997)”.

How badly affected by this aberrant feedforward mechanism a person becomes is dependent upon the amount and severity of chronic stress which they have experienced. Apart from the stress factor, people in the middle age to older age group are considered to be more vulnerable to this degenerative process for a variety of reasons.

“the more damage to the brain a person has suffered, the harder it is for him or her to “turn off” stress. The part of the brain that “shuts off” cortisol production—thereby reducing the detrimental effects of stress—commonly deteriorates with age. When this happens, the person reacts even more strongly to stress— and therefore suffers even more damage to the shut off mechanism. Thus a deadly downward spiral occurs.” (Khalsa 1997 p 52)

It needs to be noted at this stage that the subjects in Grossarth-Maticek’s Yugolsavian sample were of an average age of 60, while the subjects in his Heidelberg sample were of an average age of 50 at the outset of the study- as such, all of his subjects are well within the vulnerable age group described by Khalsa (1997). The effects of chronic stress on the feedforward mechanism are evidenced by a study cited by Khalsa (1997 p 156) wherein he describes Vietnam veterans suffering from post traumatic stress disorders. The difficulty that they had in turning off their biological responses to stress was
demonstrated in that they were measured to produce 40% less of the chemicals required to automatically shut off the feedforward mechanism. Considering the age groups of Grosarth-Maticek’s subjects, it is worth considering how much trauma had been suffered by these people during WWI and WWII? Subjects in Grosarth-Maticek’s Yugoslavian sample (average age of 60 in 1969) would have been of combatant age during WWII, as would have the subjects of his Heidelberg study. PTSD for war-zone veterans occurs in between 12-15% of all cases, and a further 12-15% of former combatants will experience at least some of the symptoms associated with PTSD (Australian Centre for Posttraumatic Mental Health 1999). American military psychologist Colonel D. Grossman’s (1999) research indicates that up to 98% of all servicemen in combat for more than 60 consecutive days display signs of psychiatric disorders.

Civilians as well as combatants are known to have suffered enormously during both of these conflicts either through direct experience of combat/bombing, or through the stress associated with having loved ones serving as combatants, or having the near impossible task of ensuring children’s safety whilst in a theatre of war. Evidence of severe psychological damage and trauma to civilian populations in war zones have been reported by de Jong, van der Kam, Ford, Hargreaves, van Oosten, Cunningham, Boots & Andrault (2004), researching non combative civilians in Cambodia, Gaza, Ethiopia, and Algeria; by Grinfeld (1999), researching Afghani refugees in Pakistan; and by Michultka, Blanchard & Kalous (1998) researching the responses of civilians to war in El Salvador. Civilians in certain parts of Europe during both WWI and WWII were exposed to relentless strains of being in a combat zone for extended periods of time, e.g. residents of French villages and towns in WWI, and residents of German cities that were being bombed daily during WWII. If PTSD is known to negatively effect neurological processes, and associated immunological functioning, it must be wondered how effected were Grosarth-Maticek’s probands from the events of WWII, as well as the post war conflicts in Yugoslavia?

In summary, excessive secretion of cortisol not only causes immuno-suppression, but it damages the limbic system of the brain to the extent that people experience a range of
psychological effects such as anxiety, irritability, inability to relax and difficulty sleeping. This increase in anxiety and stress damages the functioning of the immune system. The effect on the brain of the ‘feedforward mechanism’ is that excessive amounts of cortisol continue to be secreted and a vicious endocrine-affective-neurological cycle has been created- a degenerative cascade effect in which increasingly excessive amounts of cortisol continues to be released. More free radical cells are created, and the immune system is further and further compromised, leading to a heightened chance of cancer cells not being effectively controlled by the immune system. This is especially apparent in the middle to older age group. Loss of ability to feel joy and an over-sensitivity to stress is the general psychological result of this degenerative cycle- the functions and structures of the brain that are most related to feelings of happiness, excitement and well being are damaged. As such, people suffering from this degenerative cascade effect continue to find life increasingly stressful and unrewarding.

The relevance of the age of Grossarth-Matick’s sample, and their neurological vulnerability as described by Khalsa (1997) and McEwan (1998) will be explored in a later chapter where the main hypothesis of this study will be presented.

The most recent breakthrough in PNI comes from an Australian research team Wheway, Mackay, Newton, Sainsbury, Boey, Herzog & Mackay (2005). While re-iterating the notion that psychological stress compromises the immune function, Wheway et al (2005) state that the molecular mechanism behind this process has to this point remained unclear. This research team reports that neuropeptide Y (NPY), an abundant stress hormone that is released from sympathetic nerve endings, ‘shuts off’ T cells while activating antigen-presenting cells (Wheway et al 2005). NPY regulates physiological and emotional processes in the brain such as metabolism and heart rate, and has a role to play in depression. This neuropeptide has been shown to dampen cytokine production by macrophages, and inhibits the killer activity of natural killer cells. The authors view these results as evidence that psychological stress, via the molecular process which they have described relating to NPY, can make an organism more vulnerable to chronic disease such as cancer (Wheway 2005).
Chapter 4

Personality & Coronary Heart Disease:

Sloan, Shapiro, Bagiella, Myers, & Gorman (1999) state that a possible a connection between mental/emotional factors and heart disease has been a topic of medical interest for hundreds of years. They claim that there now exists sound empirical evidence of such a link:

“Strong emotion in general, and fear, anger and grief in particular, have been associated with angina pectoris, myocardial infarction and sudden cardiac death. As scientific study of these relationships has proceeded, association between emotional state and physical health have drawn strong empirical support from epidemiological studies and, more recently, prospective studies” (Sloan et al 1999 p:58).

Kubzansky, Kawachi, Weis & Sparrow (1998) state that the traditional risk factors for CHD such as smoking, hypertension, hypercholesterolemia, obesity, physical activity, diabetes and hormonal factors, explain only around 40% of the occurrence of CHD, leaving around 60% unaccounted for by such factors.

Type A and the measurement of the CHD personality:

Eysenck (1985 p540) quotes Sir William Osler as stating early last century that it was not the

“delicate neurotic person who was prone to angina, but the robust, the most vigorous in mind and body, the keen ambitious man, the indicatory of whose engine was always at full speed ahead”.

A range of commentators in the 1940's and 1950's characterised the CHD prone person in the following ways:- an excessively strenuous worker who went many years without vacation under stress and strain; marked by depressive or compulsive neurotic
tendencies; unremitting compulsive compensatory work habits, whose planned careers lead frequently to executive and managerial positions; contrasted traits of insecurity and inferiority; noted impatience, anxiety, conscientiousness and driving qualities which lead to long hours, little recreation, poor sleep and diets; paradoxical traits of aggression and mildness; less introspective and more capable of the free expression of aggression (Eysenck 1985). As with mind-cancer research of that era, these reports are mostly based on anecdotal rather than empirical observations.

The construct of Type A behaviour patterns entered the scientific discourse after being introduced by Friedman and Rosenman in the 1950’s, when they described the coronary prone behaviour pattern, termed Type A behaviour (Walker, Burnham, Borland 1994). Eysenck (1985) suggests that the notion of coronary prone Type A behaviour includes the following traits:- aggression, ambition, competitiveness, a chronic sense of time urgency, impatience, behavioural alertness, and an intense commitment to occupational goals. However, Eysenck (1985) is critical of the Type A construct as it is a non-continuous typology of personality, and like other non-continuous typologies, is deserving of rejection by psychologists in being a poor reflection of reality. Eysenck (1985) contends that practically all personality traits are normally distributed, and it seems unlikely that Type A could differ so fundamentally from all other personality traits.

The different components of Type A do not always correlate, suggesting that the components are not necessarily as related into a pattern as earlier thought. In addition, Eysenck (1985) states that researchers have not always found a correlation between Type A and CHD. Despite this, he does however concede that there is “a great deal of evidence that Type A behaviour is characteristic of patients with CHD” (Eysenck 1985.p 547), suggesting that within the vague and inadequate Type A construct could lay an important notion.

A further criticism is that the Type A construct is used as though psychology had not already developed reliable and valid dimensions of personality, and little attempt is made to relate the Type A construct to established dimensions. For example, Type A appears to
relate closely to Extroversion, Neuroticism, and anxiety; and the CHD prone patient appears to exhibit high degrees of Psychoticism, especially hostility/aggression/cynicism, and some aspects of neuroticism, eg. depression (Eysenck 1985).

Glassman & Shapiro (1998) state that by the late 1970’s, researchers began to have difficulties in replicating Rosenman & Friedman’s earlier finding in relation to Type A and CHD. Meta-analytic studies conducted in the 1980’s demonstrated that there was at best a modest relationship between Type A and CHD over a 30 year period, however this association began to weaken as time went on. In the 55 studies analyzed by Booth, Kewley & Friedman in 1987, the strongest association with CHD was found for depression, not for the Type A construct (Glassman et al 1998).

Rather than rejecting the entire Type A notion, Grossarth-Maticek & Eysenck (1990.p154) claim that certain aspects of Type A personality are relevant to CHD, ie. anger, hostility and aggression. These authors suggest that the CHD prone personality shares with the cancer prone personality the characteristics as stated below under (i), but is differentiated from the cancer prone personality by those characteristics displayed under (ii).

(i) characterised by failure to cope adequately with interpersonal stress, and
(ii) development of strong anger, aggression and hostility responses.

The construct of the Type 2 personality within the Grossarth-Maticek & Eysenck (1990) studies reflects the retention of these Type A characteristics as being highly correlated with, and predictive of CHD when viewed in conjunction with the tendency towards failure to cope adequately with interpersonal stress.

Smith et al (2004) define anger, hostility and aggression in the following terms:-
Anger- an unpleasant emotion ranging from intensity from irritation or annoyance to fury or rage. A personality trait of anger refers to the tendency to experience pronounced and frequent episodes of this emotion. Hostility- a negative attitude towards others involving
ill will, denigration and enmity. This factor involves cynicism in regards to other people’s motives, ie. a belief that they are motivated by selfish concerns, as well as a mistrust of them. A relational view of being in opposition to others and an expectation that others are likely sources of wrong doing are also involved in hostility. Aggression- this involves a variety of physical and verbal behaviours which are usually described as attacking, destructive or hurtful.

Eysenck (1994) suggests that there is a well demonstrated correlation between CHD and cholesterol, however there is little known of the relationship between personality and cholesterol. Sharpley (2002) cites research in which subject’s dangerous lipid levels (eg cholesterol) increased when they were required to play a demanding computer game. He suggests that this adds several SNS-activation/PNS withdrawal components to the traditional risk factor profiles to people who over react to stress. In regards to the role of cholesterol, Eysenck (1994 p.200) states,

“It does seem to be established that cholesterol performs a role in the genesis of CHD similar to that of cortisol in relation to cancer, and (is) equally subject to psychosocial factors” (p200).

Grossarth-Maticek, Eysenck, Gallasch, Vetter & Frentzel-Beyme (1991) report research with probands recruited in Grossarth-Maticek’s study begun in Heidelberg in 1972. After sorting probands into the personality typology, Grossarth-Maticek ascertained that there was a clear relationship between personality type (i.e Type 1 & 2) and the degree of sclerosis observed in the fundus of the eye- a statistically significantly higher rating of sclerosis in the eye (measured by ophthalmologists) was found to occur in the sample of CHD prone probands in comparison with the cancer prone probands. The results showed a “close correspondence between the values for blood sugar, blood cholesterol, diastolic and systolic blood pressure and the degree of sclerosis as shown in the fundus of the eye” (Grossarth-Maticek et al 1991, p 347). The measurements of sclerosis were gathered before probands for the therapy experiment had been selected, and again 2 years later. Incidence and mortality data were gathered in the 13 year follow up study. The
therapeutic interventions have already been described in an earlier chapter. The results of the study indicate that there was a close relationship between sclerosis observed in the fundus of the eye and death from heart infarct, stroke and other chronic diseases related to the circulation of the blood, and a strong negative relationship between observed sclerosis and cancer. From these results, Grossarth-Matick et al (1991) claim that personality plays an important role in both the degree of sclerosis and CHD, with the effects being mediated through other physical factors. Again, as with the proposed theory for cancer, Eysenck (1991) suggests that his speculations about the biology of the mind-CHD link are at the beginning stages.

Holland (in Goleman and Gurin 1993), who is very skeptical of the notion of a cancer prone personality, expresses acceptance of the role of psychological factors in the causation of CHD, based on research evidence. In a similar vein, Amelang (1997), a major critic of Grossarth-Matick’s claims, draws on research by Friedman demonstrating the role of personality factors such as anger, hostility and aggression in the causation of CHD. He states that such personality factors have around the same magnitude of effect on CHD as cigarette smoking and other standard medical risk factors, a correlation of around 0.14, where cigarette smoking, cholesterol and CHD correlate at a rate of under 0.15. All of these are low magnitude in absolute terms, but it suggests that personality factors are as meaningful as standard medical risk factors in the causation of CHD- again, this assertion does not take into account Grossarth-Matick’s findings as Amelang (1997) considers them to be on unproven value.

Smith et al (2004) state that while some reviews expressed skepticism, most of the recent evidence analysed in their review supports the basic psychosomatic hypothesis that psychological factors are involved in the causation of CHD. Six of eight recent prospective studies of initially healthy subjects reviewed by Smith et al (2004) demonstrated that measures of anger, hostility and aggression predicted CHD incidence and mortality- the effect sizes of these factors were at least as large as those of the traditional medical risk factors.
Heart Rate Reactivity and CHD:
Heart Rate Reactivity (HRR) is the extent to which the heart rate is measured to respond at the onset of a particular stimuli- of interest in this paper are the effects of environmental stimuli, such as stressful events. HRR is usually calculated by taking the HR during a stressful event from the HR during a period of rest, thereby demonstrating the effects of the stimuli (Sharpley 2002). The autonomic nervous system (ANS) is centrally involved in determining the speed at which the heart beats. The ANS operates via the parasympathetic nervous system (PNS) and the sympathetic nervous system (SNS). Guyton (1992) states that strong sympathetic stimulation can elevate the heart rate to as many as 200 beats per minute, causing cardiac output to increase as much as two or threefold. Parasympathetic stimulation has the opposite effect, actually stopping the “heart beat for a few seconds, but then the heart usually ‘escapes’ and beats at a rate of 20 to 30 beats per minute thereafter.” (Guyton 1992 p.86). Sharpley (2002 p.56) states that,

>“Activation of SNS and withdrawal of the PNS are generally associated with “stress” reactions, that is, increases in the physiological and psychological arousal of the body to a threat or demand…. Thus, SNS dominance over PNS acts to increase the HR and BP (as well as other aspects of our physiological functioning) so that blood and oxygen can be carried quickly to the muscles which we use when under stress…. Conversely, PNS dominance over SNS automatically decreases HR and BP (and other physiological responses).”

The combined effects of SNS and PNS activity work to allow a balance in terms of levels of arousal, preventing both a constant over arousal as well as ensuring an adequate response to threat when appropriate. Some people appear to be in a consistently over-aroused state for the stressors which they are experiencing. Sharpley (2002) states that the effects of this over-arousal is an increase in the absolute speed of heart beats, and the HR-reducing effects of the PNS are decreased. Such a state is referred to as “SNS dominance” as the heart is considered to be mostly under control of the SNS due to PNS ‘withdrawal’. Sharpley (2002) cites a range of studies which demonstrate that SNS dominance (and lack of PNS activity) is a strong predictor of heart disease.
Of concern is the effect on cardiac health when there is a sustained high level of HRR, or frequent and dramatic HRR. Several studies, cited by Sharpley (1998), suggest that HRR is either a correlate or plays a causal role in the build up of plaque which forms on the surface of arteries, leading to potentially dangerous blockages which have been linked with CHD in both animals and humans. However, there is some dispute in the literature as to whether the build up of plaque (atherosclerosis) results from dramatic and frequent increases in heart rate during stressful events, or as a result of high levels of HRR maintained over a long period of time leading to prolonged elevated HR which is causally associated with CHD (Sharpley 1998). The development of plaque on the surface of the arteries presents a danger as it may eventually block the arteries (Sharpley 2002). Other correlates of increased heart rate are increases in adrenaline and cortisol levels- the latter has been associated with increased risk of heart disease by Troxler, Sprague, Albanese, Fuchs and Thompson (1977).

Anxiety & CHD:
In a meta-analytic review of the research literature from 1980 to 1996, also including some relevant studies from earlier years, Kubzansky et al (1998) state that there is sufficient epidemiological, psychological and experimental evidence supporting the association between anxiety and CHD.

“the strength, consistency, and dose-response gradient of the association between anxiety and CHD, together with the biologic plausibility of the experimental evidence, suggests that anxiety may contribute to risk of CHD and that the relationship warrants further investigation” (Kubzansky et al 1998).

Following Barlow’s extensive review of the literature pertaining to emotion, Kunzansky et al (1998 p. 48) define anxiety as a “future oriented negative affective state resulting from perceptions of threat, characterized by a perceived inability to predict, control, or obtain desired results in upcoming situations”. The presence of fear in regards to
apprehensions about the future is a defining characteristic of anxiety. Kubzansky et al (1998) use the term anxiety to refer to the full spectrum of experience from the normal to the pathological, suggesting that the differences are a matter of duration, intensity and meaning of the experience to the sufferer. More will be said in a later section about the relationship between depression and CHD, however at this stage it is important to note that there is a high co-morbidity reported between anxiety and depression. Kubzansky et al (1998) state that there is research evidence supporting an association also between anger and anxiety, and that there are similar physiological components between them.

Four main studies were selected by Kubzansky et al (1998) for their review of the evidence:- the Northwick Park Health Study, entailing a 10 year follow up of 1457 initially healthy men; the Health Professionals Follow-up Study, in which 33,999 male health professionals have been tracked in a prospective study; the Normative Aging Study, in which men were prospectively studied in regards to self reports of anxiety and incidences of CHD; and the Framingham Study, which entails a 20 year follow up of 749 initially healthy women. While the effects of the Normative Aging Study did not reach statistical significance, overall, the magnitude and consistency of results from these studies suggested a strong link between CHD outcomes and anxiety scores as a predictor. The lack of female subjects in three of the four studies is an obvious weakness. In addressing the possibility that the discomfort associated with sub-clinical heart disease may lead to feelings of anxiety, Kubzansky et al (1998) state that there is no data supporting this explanation- in fact, with all of the studies cited, subjects were excluded from participation if they exhibited any symptoms of CHD. They do conclude, however, that this possibility cannot be excluded although it is an unlikely explanation.

There are three possible pathways which may explain the reported association between anxiety and CHD. Kubzansky et al (1998) suggest the following effects of anxiety:-

1) Anxiety disorders have been associated with poor health behaviours such as loss of sleep; increased smoking, alcohol and drug consumption; and decreases in physical activity. Kubzansky et al (1998) make the point that health behaviour differences mediate the relationship between anxiety and CHD.
2) Promotion of athero-genesis via hypertension arising from chronic SNS activation. “Some investigators have hypothesized that there is an important link between anxiety and atherogenesis via stress induced increases in sympathetic nervous system activity and the release of plasma catecholamines.” (Kubzansky et al 1998 p.52).

3) Triggering fatal coronary events such as arrhythmias, plaque rupture, coronary vasospasm, and thrombosis. Risk factors here are intense chronic psychological states which pervade and burden daily life, and acute psychological states associated with high neural activity input into the heart leading to a triggering event.

Anxiety is known to be a widely spread phenomenon in the general population. The review conducted by Kubzansky et al (1998) reveals that the risk of CHD posed by anxiety is not limited to clinical populations, but rather, sub-clinical anxiety also poses a significant threat.

Heart Rate Variability, hostility and CHD:
Sloan et al (1999) have drawn on research in autonomic nervous system physiology, pharmacology, cardiology, and vascular biology to develop a model which elucidates the possible mechanisms by which psychological/behavioural factors contribute to the development of cardiac disease. The model which they propose suggests that psychological/behavioural factors can have the effect of reducing cardiac autonomic control, which is in turn associated with an increase in blood pressure variance (BPV) in response to psychological challenges. This increase in BPV resulting from challenge is associated with increased BPV throughout the day which in turn adds to plaque formation, plaque rupture, and subsequent acute coronary events. Sloan (2001 p.3) states,

“The heart rate variability appears to act as a sort of lid on blood pressure variability, and when your heart rate variability falls, the blood pressure variability course is finally disinhibited, and that we’ve argued leads to the intermediate causes of coronary artery disease: damage to the lining of the
endothelium, of the coronary arteries which over the course of many, many years, produces atherosclerosis”.

Heart rate variability (HRV) has an inverse relationship with BVP, ie. as HRV goes down, BPV goes up, which is associated with coronary ill health. Rather than being a relatively steady event, heart rate in healthy people generally displays a degree of variability in cyclical patterns, i.e. it rises and falls in a patterned manner. Under stressful circumstances, the amount of HRV decreases, whilst when in happier circumstances, HRV is generally higher. HRV is affected by psychological experience and is an index of cardiovascular health.

Sloan et al (1999) state that although much of the literature regarding this relationship is from cross-sectional analyses, their working hypothesis is that increased BPV is causally related to heart disease outcomes. To date, their research is cross-sectional which, although demonstrating a relationship, is not able to demonstrate causality. As such, they recommend that further research of these variables is needed.

The psychological factor of hostility plays a central role in Sloan et al’s (1999) model, as they state that it is associated with increased cardiovascular reactivity in response to psychological challenge. Rhodes, Harrison & Demaree (2002) state that hostility was originally studied as a component of the Type A construct, however it is now a main focus of research in its own right. Rhodes et al (2002) favour a psychophysiological model of reactivity to explain the association between CHD and hostility. This suggests that hostile people are more vigilant to possible conflicts; more likely to respond with exaggerated physiology to stressors such as possible conflicts in their environment, leading to an eventual ‘burnout’.

Eysenck (1985) cites the research conducted by Williams, Bareford and Shekele in 1984 with 255 medical students were followed up after having completed the MMPI. They found that high scores on the Hostility scale of the MMPI were associated with coronary artery disease and prospectively with risk of CHD and with total mortality- the
psychological construct measured by the Hostility scale is described in the following terms: “cynicism, an attitudinal set that stems from an inadequately developed sense of basic trust and centres around beliefs that other persons are generally mean, selfish and undependable” (Eysenck 1985. p545). Williams et al (also cited in Eysenck 1985) speculatively propose that the biological path way leading to disease may involve excessive testosterone excretions during vigilant observations of other people, as well as excessive secretion of cortisol and catecholamines during frequent and intensive moments of anger.

Along with depression and anxiety, hostility has been associated with autonomic dysregulation and subsequent cardiac strain. Stein, Carney, Freedland, Skala, Jaffe, Kleiger & Rottman (2000 p493) define autonomic dysregulation as a “predominance of adrenergic activation and/or lack of parasympathetic modulation.” Elevated heart rate is one consequence of this dysregulation, leading to coronary risks detailed elsewhere.

The hostility which Sloan et al (1999) refer to is characterized by a cynical mistrust of others. Take as an example the person who becomes annoyed while waiting in a line at the bank, feeling displeasure at the people in front of him/her as they are just wasting his time, not attending to important or real business. This person displays a cynical mistrust of other people’s motives and actions. As to whether the risk with this scenario is involved in either holding the annoyance in or expressing it outwardly, Sloan (2001) states that not enough is yet known to answer this question with confidence- there is evidence supporting both options, however the presence of the cynical mistrust of other people is the common characteristic. It is presumed that the autonomic nervous system links these behaviours to heart disease as it is this part of the nervous system that regulates the cardiovascular and respiratory systems.

In a study of 568 occupationally diverse workers Ganster, Schaubroek, Sime & Mayes (1991) demonstrated that from a range of personality traits, only hostility (assessed in The Structured Interview) was significantly related to physiological reactivity and recovery in relation to the Stroop Colour-Word Conflict Task. Subjects who scored
higher in the hostility dimension displayed more physiological reactivity in their blood pressure, heart rates, skin temperatures, and electrodermal responses compared to low hostility subjects. Suarez & Williams (1991) also found an association between high hostility scores and levels of physiological response on a stressful anagram task which included harassment of the subject. High scores on hostility were related to greater systolic blood pressure and forearm blood flow, both measures of autonomic arousal. The findings reported by Rhodes et al (2002) also demonstrate an association between high levels of hostility and high degrees of autonomic responding during a stress task. Rhodes et al (2002) had their 50 subjects undergo a Cold Presser Test, 25 assessed as being in a high hostility range, and 25 assessed as being low in that dimension. High hostile subjects showed an increase in heart rate as a result of the test, while low hostile subjects showed a decrease in heart rate in response to the stressor.

To test the relationship between psychological experiences and the cardiovascular system Sloan, Shapiro, Bagiella, Myers & Gorman (1999) conducted a small pilot study with 23 normal subjects differing in cardiac control (operationalised as HRV) due to differences in cardiac capacity. Subjects were required to participate in a series of psychological challenges including mental arithmetic, a colour-word matching task and 70 degree head up tilt task. The dependent variable in this study was HRV. Sloan et al’s (1999) subjects were normal healthy people who were assessed as differing in levels of hostility, with some being defined as being high in hostility while others were defined as being low in hostility. Base line HRV data measured during a period lacking psychological challenge indicated that there was no significant difference between the high and low hostility subjects in HRV. However, when the challenges were presented to the subjects, HRV for the high hostility subjects fell to a significantly greater degree than it did for the low hostility subjects. Subjects with higher HRV were demonstrated to have higher levels of autonomic control, showing a pattern different to the dysregulation associated with high blood pressure variability. It is inferred from this that when high hostility people encounter challenges in their ordinary daily experience, their HRV falls to a larger degree than does the HRV for low hostility subjects.
In summary, Sloan et al (1999 p.62) propose,

“that attenuated cardiac autonomic control, principally cardiac parasympathetic modulation, is a significant contributor to CAD and acute coronary events. The effect of this attenuation is the reduction of the capacity to buffer fluctuations in blood pressure (BPV) in response to challenge both in the laboratory and throughout the day, which may confer risk of CAD independent of MAP (i.e mean arterial pressure). This hypothesis is consistent with the fact that subjects with psychiatric/psychological risk factors for CAD also have diminished cardiac autonomic control...”

Depression & CHD:
Glassman et al (1998) state that although the relationship between depression and CHD had been speculated upon for hundreds of years, the first scientific study testing this was conducted in 1937. This study, while finding an association between depression and CHD, failed to control for the fact that the subjects were institutionalized, and many subsequent studies until the 1970’s also failed to control for the effect of psychiatric treatments of depression. During the post WWII years and into the 1970’s, psychiatric interest in CHD research was typified by an interest in the Type A construct, rather than with depression per se. Glassman et al (1998) state that in the late 1970’s and 1980’s, researchers in this field became aware of the possible confound of cigarette smoking in terms of explaining the positive outcomes with depression. The possibility arose that the repeatedly reported association between depression and CHD may be no more than the relationship between the effects of cigarette smoking and CHD, as depression is highly correlated with cigarette smoking. Few studies until that time had statistically controlled for the effects of cigarette smoking. The first study to do so was the Medical Illness Follow-Up Study, a 12 ½ year prospective study. Glassman et al (1998) state that his research team became aware of this data set taken from 2832 subjects who were above the age of 45 and assessed as being medically healthy at the outset of the study. This data set controlled for smoking as well as other known risk factors such as gender, weight, blood pressure, and cholesterol. The relationship between depression and CHD held even
when the sample was separated into smokers and non-smokers. In the years between 1996-1998, Glassman et al (1998) report that 6 additional studies have controlled for the effects of treatments of depression and smoking, with five of the six reporting a strong relationship between depression and CHD.

Sloan (2001) states that there is ‘overwhelming’ evidence supporting the contention that depression confers a heart disease risk. The precise mechanisms are not yet clearly understood, however he suggests that there are at least four potential explanations.

i) depression, like hostility, leads to diminished autonomic nervous system control of the cardiovascular system.

ii) depression is linked with the degree to which clots develop in the coronary arteries, the stickiness of one’s platelets.

iii) there is considerable evidence linking heart disease with lack of social support, and patients with heart disease may have fewer social supports,

iv) depressed patients are less likely to adhere to their heart medications and treatment recommendations compared to patients who are not depressed.

A fifth factor, not presented by Sloan (2001) is that depressed people are more likely to engage in unhealthy behaviours such as smoking, excessive drinking, lack of exercise, poor diet etc, however in light of Marmot’s (1998) Whitehall Studies (to be discussed in a later section), the contribution of these factors would appear to be less than psychosocial factors. Stein et al (2000) state that a substantial portion of depressed patients smoke or have a history of smoking, and that current smoking also decreases HRV. Sloan (2001) concludes that any of the four above mentioned factors, or combinations of all of them, may link depression to heart disease.

Stein et al (2000 p.493) state that there is

“considerable evidence that clinical depression is a risk factor for cardiac morbidity and mortality in patients with coronary heart disease, that is, heart disease resulting from coronary atherosclerosis. Moreover, relatively severe forms
of depression are associated with a greater risk for cardiac events, for example, myocardial infarction, need for balloon angioplasty, coronary bypass surgery, or other cardiac related hospitalizations.”

Decreased HRV has also been associated with depression, with successful treatment of depression being accompanied by an increase in HRV (O’Connor, Allen & Kasznak 2002). O’Connor et al (2002) compared the HRV rates of 10 bereaved subjects, 10 depressed subjects and 10 control subjects, concluding that bereaved individuals with higher levels of depression showed lower levels of HRV. In addition, they found a correlation between HRV and coping styles, with the use of more adaptive coping styles correlating with higher HRV and lower heart rates; whereas the use of less adaptive coping styles was correlated with lower HRV and higher heart rates. In a study of 21 mildly depressed and 19 moderately-to-severely depressed cardiac patients, Stein et al (2000) concluded that patients with more severe depression evidenced slower HRV than did less depressed patients. All of the cardiac patients were required to cease their use of heart and depression medications for at least 3 days prior to the assessment of HRV so as to eliminate the effects of these medications on the HRV measure. Suggesting the importance of degree of depression in HRV, Stein et al (2000) found that there were higher HRV scores for non depressed compared to mildly depressed patients, although this difference did not reach statistical significance. The presence and degree of depression were assessed via a standard psychiatric interview as well as the Beck Depression Inventory. The importance of this finding relates to the fact that lowered HRV scores have been shown to predict subsequent mortality from heart disease (Stein et al 2000).

In a study of 309 bypass surgery patients, Sloan (2001) and his research team followed the patients for a year post surgery. The patients were assessed with a standard psychiatric interview to identify depression prior to their release from hospital. After statistically controlling for standard risk factors, Sloan (2001) found that depression was associated with a 2.3 greater increased risk of recurrent cardiac events (eg. another heart attack, the requirement of more surgery, unstable angina) in the year following surgery.
In a review of the research literature, Glassman & Shapiro (1998) cite many studies which concur with Sloan et al’s (2001) results- heart patients experiencing depression have higher incidences of further cardiac events and mortality rates.

After reviewing the available evidence at that time (published studies from 1970-1998), including studies from heart patient populations as well as community populations, Glassman et al (1998 p.11) conclude that,

“depression is unquestionably associated with cardiovascular disease. The presence of depression preceding the onset of ischemic heart disease individuals initially free of disease, the greater risk of sudden death amongst post-myocardial-infarction patients with both depression and arrhythmia, and the predisposition to increased platelet aggregation among depressed patients all seem to point to a causal relationship. Yet it is important to remember that what has been demonstrated is an association and not causality. It is conceivable that atherosclerosis could be a cause of both depression and heart disease.”

As a possible biological mediating factor in the relationship between psychological stress and CHD, Troxler, Sprague, Albanese, Fuchs and Thompson (1977) reported research from 71 outpatients who had coronary angiography as part of their medical evaluation. They found that plasma cortisol was second only to serum cholesterol as a discriminator between their subjects who had coronary artery disease (CAD) and those who were healthy. Further, they found significant correlations between the cortisol and the ‘cardinal risk factors’ for CAD, namely:- cholesterol, blood pressure, and smoking. The association between plasma cortisol and cholesterol was the strongest correlation, and the association increased with the severity of the disease. As such, it can be concluded that cortisol, a hormone which is highly reflective of psychological stress, is not only an indicator of risk for cancer, but also for CHD.
National Heart Foundation of Australia position paper:

In a review of literature on the effects of psychosocial factors on CHD, an Expert Working Group (EWG) constituted of cardiologists, cardiovascular physiologists, psychiatrists, behavioural scientists, public health medics and general practitioners was formed by the National Heart Foundation of Australia, culminating in a position paper publication (Bunker, Colquohoun, Esler, Hickie, Hunt, Jelnick, Oldenburg, Peach, Ruth, Tennant & Tonkin 2003). Bunker et al (2003) performed a review of systematic reviews, investigating the 57 literature reviews published between 1960 and 2001 which satisfied their criteria. In most of the reviews, the studies had controlled for the standard CHD risk factors; the reviews often included case-control as well as prospective studies, and all of the reported evidence reviewed conformed to the 1995 National Health and Medical Research Council classification E3 (Level III): Evidence obtained from well-designed cohort studies, preferably from more than one centre or research group. The EWG examined the factors that are commonly associated with the term “stress”. These included:-

- Depression, anxiety, panic disorder
- Social isolation and lack of quality social support
- Life events such as bereavement, earthquakes and terrorist attacks
- Work related “stresses”
- Anger and hostility

Bunker et al (2003) concluded from the research based literature that there is strong and consistent evidence across all reviews that depression is an independent risk factor for clinical CHD. This association was found for both men and women, for subjects living in different countries, and for various age groups. In addition, the risk of CHD was directly linked to the severity of depression, with a 1-2 fold increase in CHD for minor depression and a 3-5 fold increase for major depression. The conclusion of the EWG was that the contribution of depression to CHD was of a similar magnitude to that of the standard medical risk factors such as cigarette smoking or hypercholesterolaemia. The same association across populations was found between social isolation and CHD incidence, with social isolation leading to a 2-3 fold increase, and lack of quality social support.
leading to a 3-5 fold increase in CHD incidence. Again, these risk factors were found to operate independently from medical risk factors. The EWG was not able to find consistent and strong evidence in support of the role which chronic life-event stressors may play in CHD, however evidence was found for the role of acute life-event stressors in triggering CHD events- the magnitude of the effect was difficult to quantify from the literature reviewed. In regards to the role which work-related stressors play in CHD, the articles reviewed did not provide strong or consistent evidence of a causal association between work stress and CHD, although there were reviews demonstrating positive findings. Type A behaviour pattern was found in the review literature to have no effect on CHD, reflecting the shift from early positive studies now displaced by studies reporting no effect. The question of whether hostility is found to be a risk factor for CHD was answered in the negative, with Bunker et al (2003) being more impressed by the level of evidence presented by reviews reporting negative findings. Anxiety and panic disorders were also determined to be of little causal relevance to CHD according to the EWG’s review, with there being neither strong nor consistent evidence demonstrating such a link.

One obvious limitation of the EWG’s review of systematic reviews is that the conclusions drawn by it are dependent upon the rigour with which the relevant primary studies have been identified, appraised and summarized. This limitation must apply to both their positive as well as their negative conclusions. One conclusion which they arrive at is that the variation of CHD incidence cannot be explained by standard medical risk factors- the factors identified in their review (depression, social isolation and lack of social support) may explain some of the variance in CHD incidence. They consider that these “stressors” contribute to the incidence of CHD at a similar magnitude to the standard medical risk factors, and operate independently from such factors as smoking, alcohol consumption, dietary factors, sedentary lifestyle, cholesterol levels, etc. The possibility is raised that psychosocial risk factors may coexist with standard medical risk factors to produce CHD outcomes, e.g depressed people are more likely to smoke, be physically inactive, eat high fat and salty foods, etc. Despite this possible clustering of risk factors, Bunker et al (2003) concluded that the stated psychosocial risk factors have
been demonstrated with a high level of strong and consistent evidence to independently contribute to CHD outcomes.

Also suggesting a strong case for psychosocial factors in ill-health are Marmot’s Whitehall studies (Bosma, Peter, Siegrist & Marmot 1998). In their study of over 10,000 British Civil Servants, while factors such as smoking, sedentary leisure time and being over weight were associated with higher incidences of CHD, Marmot and colleagues found that these factors alone were not able to explain the differences in health outcomes such as CHD. Rather, the most important predictor was where the subjects were in the organizational hierarchy, having implications for issues such as internal/external locus of control, learned helplessness, and self efficacy. The Whitehall studies demonstrated a three fold higher mortality among office support grades than among administrators, and a four fold gradient between the lowest to the highest in the hierarchy. These studies also demonstrated that poor psychological functioning (low expectations, poor self esteem, negative affectivity) was associated with such behaviours as smoking, lack of exercise and an unhealthy diet (Stafford, Hemingway, Stansfeld, Brunner & Marmot 1998). However factors such as smoking, blood pressure, alcohol consumption, physical activity etc such were found to account for only 25-35% leaving the remaining outcomes unexplained by standard medical risk factors (Syme 1998). Members at the bottom end of the British Civil Service were those who had less sense of control over their destinies compared to those at the top end of the hierarchy- there is less opportunity for the former to have influence over the events which impact on their lives. In his attempts to ascertain the causes of the gradient which he found in the British Civil Service, Marmot discovered that the concept of control was able to statistically account for the gradient where all of the other physical risk factors studied were not.

Syme (1998) suggests that the list of standard medical risk factors for heart disease (containing 20 to 30 items, such as cholesterol, blood pressure, smoking, viruses, etc) explain around 40% of the disease which occurs, whereas psycho-social factors such as levels of social support and social class appear to be related to all classes of death. He suggests that a sense of control is related to both social support (as a ‘buffer’) and to
social class (lacking in lower income groups), and that these factors affect the body’s defense systems, making one more vulnerable to factors such as smoking and high cholesterol (Syme 1998). Marmot (1998) states that a similar step wise gradient (a fourfold increase in mortality) is seen in society as a whole when people are classified according to their social class. Marmot’s (1998) conclusions are that the greater is the sense of control one has over the issues which effect one’s life, the better will be one’s health outcomes. The lack of sense of control, as demonstrated in the workers at the lower end of the British Civil Service, the poorer will be one’s health outcomes.

In light of the above mentioned evidence, it seems difficult to conclude, as does Amelang et al (2003) that psychosocial factors play such a small role in the causation of CHD as to make them of no importance. The theories or models that have been developed to help one make sense of these type of research results will be examined in the next chapter.
Chapter 5

Models of Disease: Diathesis-Stress and the Cascade models:

Much of the mind-cancer/CHD research reported in the Health Psychology literature represents an application of the diathesis-stress model of disease. Grossarth-Maticzek, Eysenck & Boyle (1994, p 4) describe the diathesis-stress theory in the following terms,

“In a meeting between an organism and a particular stress situation, both the organism and the situation make a contribution to the outcome”.

Monroe and Simons (1991) state that the term ‘diathesis’ derives from ancient Greek, and can be traced back at least to the last great Classical physician, Galen (131-201 AD) who interpreted Hippocrates theories of disease. In Grossarth-Maticzek et al’s (1994) studies, the diathesis-stress theory of cancer finds the following expression: Hopelessness depression x stress = disease. The ineffective coping styles shown by their proposed cancer prone and CHD personalities in the face of stress constitute a major part of the psychosocial diathesis which then pre-disposes the person to disease.

Monroe et al (1991) make the point that there are four aspects of life stress which need to be considered:-

a) temporal factors (eg. acute vs chronic)
b) dimensional issues (eg. major vs minor stressors)
c) qualitative characteristics (eg. desirable vs undesirable stressors)
d) and the rules for combining temporal, dimensional and qualitative life stress.

They suggest that in research utilizing a diathesis-stress conception, the dimensions and the qualities of the stressor need to guide the process.

In an example of research designed to test the diathesis-stress theory of disease, Grossarth-Maticzek et al (1994) chose a quasi-experimental study design, avoiding some
of the problems of laboratory designs manufacturing only mild levels of stress. Their sample population, collected as part of Grossarth-Maticek’s Heidelberg studies was constituted of Jewish survivors of WW II Nazi concentration camps- people who, in varying degrees had experienced very high levels of real life stress. The actual levels of stress experienced covered the following range of situations:- length of time incarcerated in a concentration camp; age at time of incarceration; and number of proband’s family members killed in the camp. Predictions were made that spending a longer time in a camp, being incarcerated at an earlier age, and having more family members killed in the camp would be related to greater probabilities of subsequent disease compared to matched controls, as greater stress had been experienced. The diathesis in this study was measured in terms of high scores on the cancer prone and CHD prone personality measures devised by Grossarth-Maticek. They hypothesised that the higher the emotional vulnerability of the proband, in terms of higher illness prone scores compared to health prone scores on the IRI devised by Grossarth-Maticek, the greater would be the chance of subsequent disease. Finally, they hypothesised that the diathesis (illness prone personality scores) and the stress would synergistically interact to produce the health outcome.

Assessing the scores and mortality/incidence figures of the 1,121 Jewish camp survivors, compared with 367 Jewish controls, Grossarth-Maticek et al (1994) claim that all of their predictions were supported by the results- former camp inmates were nearly twice as likely to die of cancer, CHD and other causes of death compared to the controls. High scores in illness prone psychological dimensions (the diathesis) was found to interact synergestically with the stressful experiences producing higher mortality. The authors refer to obvious problems with this study, such as the effects of a starvation diet, over crowding, and physical maltreatment, all of which could not be controlled and may have affected the outcome. They do point out, however, that factors such as smoking and drinking, which are usually considered to be the risk factors for disease, were not related to health outcomes in the probands- the camp survivors had lower measures of drinking and smoking than the controls, but a much higher death rate. While the results are offered tentatively, Grossarth-Maticek et al (1994) do interpret them as positive support for the diathesis-stress theory of disease, particularly cancer and CHD.
Dykema, Bergbower & Peterson (1995) state that Seligman’s reformulated learned helplessness model also represents a diathesis-stress theory of disease: both a pessimistic explanatory style and bad events must be present for the disease to occur. According to these authors, a problem has arisen in that most of the helplessness research has not in fact tested the diathesis-stress theory, as only the diathesis (attributional style) has been measured to the exclusion of measuring the supposed ‘stress’. Dykema et al (1995) state that of those helplessness studies which have actually tested the diathesis-stress theory, the results have provided only mixed support. They suggest that either these inconsistent results have occurred because of poor operationalizations, or that an alternative reality to that proposed by the diathesis-stress theory is operating. One possibility is that attributional style may have an effect on health whether or not the person experiences major life event stress. The Cascade Model is offered as an alternative to the Diathesis-Stress theory, wherein a more cognitive-interactive view of stress is utilized. In private correspondence with the current author, Peterson (1997) stated “My sense of a cascade is that it is different from a diathesis-stress approach, which treats the stress as ‘independent’ of the diathesis, not something set into motion by it”.

Dykema et al (1995) state that the diathesis-stress theory, as used in the learned helplessness reformulation, views stress as an objective reality which is imposed upon the person from without. This is contrary to the cognitive theory where stress is seen as the result of a cognitive appraisal of the demands of the situation in comparison to the perception of ability to meet those demands. The Cascade Model implies the possibility that people can still experience stress in the absence of objectively occurring events- a pessimistic attributational style on its own, without negative life events may be disturbing enough to produce more stress and poor health outcomes. Research conducted by Dykema et al (1995), with 121 undergraduate students, concluded that there was no support in their study for the Diathesis-Stress model, however their data did support the Cascade model, i.e explanatory style on its own was enough to lead pessimistic subjects to experience more stress, which in turn produced poorer health outcomes.
“...pessimistic explanatory style cascaded into increased reports of hassles, which led people to view major life events as having greater negative impact, which in turn increased the likelihood of illness. The mere occurrence of such events did not interact with explanatory style to predict subsequent illness. However, major life events did influence the cascade of psychological states starting with pessimism and ending with illness”. (Dykema et al (1995.p 365-366).

This finding is consistent with Monroe et al's (1991 p411) suggestion that the nature of the diathesis will actually have an influence on the nature and course of a stressful life event.

“Most people are, at least in some part, the creators of the circumstances they endure...a diathesis influences the manner in which the person negotiates life’s course, and consequently the nature of the stressors to which he or she is exposed...stress is not a random process, but rather part of a developmental sequence systematically influenced by the diathesis. Whereas the construct of stress may still play an important role in the evolving scheme, it is generated to a considerable degree by the person’s behaviour, which in turn is likely to be influenced by the diathesis.”

Monroe et al (1991) go on to state that the diathesis-stress model has experienced a long standing appeal, however the ways in which the diathesis and the stressors interact to produce the disorder has not been well specified, i.e is there a simple additive relationship, a simple synergistic interaction, or a complex synergistic interaction between the variables?

Cooper & Faragher (1992; 1993) have conducted extensive quasi-prospective studies with large populations in relation to psychosocial risk factors and breast cancer, a condition of special interest as it is without “...any known major environmental precursors” (Cooper & Faragher 1993. p 653). The psychosocial risk factors which they have studied include:- the perception of stressful life events (Cooper, Cooper & Faragher
1989), personality characteristics associated with breast cancer (Faragher & Cooper 1990), and coping strategies used by women to cope with stress (Cooper & Faragher 1992).

From a quasi-prospective study with 2163 women, Cooper & Faragher (1992) concluded that when the influence of age had been statistically controlled, women with breast cancer were more typical of the Type B behaviour pattern, however with some Type A behaviours as well. They concluded that women diagnosed as having breast problems tended to:-

• have more ‘laid back’ lifestyles, being less rushed, more deliberate and more patient than the controls
• were much less competitive, ambitious and hard driving than the controls
• had a slow and casual approach to activities, combined with an increased desire to gain personal satisfaction
• showed a tendency to suppress their feelings
• have few interests involving personal relationships, outside their home and work environments. (Faragher & Cooper -1990. p 669).

Some of Cooper et al’s (1989) research focused on the stress side of the diathesis-stress formulation. Again utilizing a quasi-prospective study design, this time with 1596 patients attending breast screening clinics and 567 controls, they reported a significant relationship between breast cancer and the incidence and perception of stressful life events. There was found to be a relationship between breast cancer and the incidences of the following:- death of a close family member or friend; personal illness, especially if requiring hospitalization and/or surgery; retirement and redundancy. Unlike other similar studies which found correlations for a broader range of life-events, this study found no correlations with other life events and breast cancer. Other, more minor life-events were found to only have a relationship with cancer when occurring in clusters rather than on their own, eg. personal relationship/work related problems, property related problems, marriage/birth, serious illness of a close family member.
Apart from severity, the major difference between the latter presented group of problematic life-events and those positively related to breast cancer is the degree of control which the individual feels they have over the incidence or severity of the problem. For example, stress concerning property or renovations is usually controllable; relationship problems with family, neighbours and work mates can often be resolved with the appropriate desire and effort. However, control over such major events as death of a spouse or friend, retirement and redundancy is usually minimal if existent at all- these events are usually imposed upon people, rather than being a matter of choice, such as with renovations, marriage, etc.

In another study, Cooper & Faragher (1993) examined the correlations between different coping styles and breast cancer, concluding that denial (bottling up of feelings) is a poor coping style. The coping styles least associated with breast cancer were found to be :-

i) externalizing, i.e let feelings out; talk to close friends; talk things over with lots of friends; treat self to something; felt that you learned something from the experience; talk to someone who can do something about the problem; try to get some sympathy and understanding from someone, and

ii) anger, i.e. get angry with people or things to ease problems; explosive, mostly temper, not tears.

Women who obtained the most healthy diagnoses were more likely to use a wider range of coping strategies. They were also more likely to use strategies described as positive, whether internal or external styles, in that they improved their perceived locus of control by either seeking external supports or building up their internal strengths. In relating Grossarth-Maticek & Eysenck’s work with a study of coping styles, Schmitz (1992) reports that subjects with personalities assessed as being cancer prone and CHD prone utilize coping strategies described as ‘emotion-oriented’ and ‘avoidance-oriented’. Those subjects assessed as being health prone utilized ‘task-oriented’ coping behaviours. In terms of subject’s reactions to stressful events, Schmitz (1992) found that cancer prone personality subjects reacted with ‘depressive reactions’ and ‘relying on significant others’, while CHD prone personality subjects responded with ‘depressive reactions’ and

In researching the ‘stress’ component of the diathesis-stress model, the importance of Cooper et al’s (1989) findings is that the actual incidence of stressful life-events was found to be correlated to breast cancer for only a few events; however, there was a strong correlation found between perception of stress and breast cancer. The actual incidence of a life stressor was perceived as having a greater impact by the group who eventually received a cancer diagnosis when compared to the benign cyst diagnosis group and the normal breast group. The cancer group perceived nearly all of the life-events as having a more severe impact on them than did the healthy subjects who experienced similar events. The effect of the perceptions of impact was found to be independent of the number or actual nature of the life event, or the degree of controllability of the life-event—“The greater the perceived impact, the greater the risk of breast cancer and the severity of the disease” (Cooper et al 1989.p 421). In a later study, they found that women who experienced little excitement in their day to day lives were more vulnerable to the negative effects of a major life-event, as they perceived the impact as being stronger on them when it occurred (Cooper & Faragher 1993). Conversely, a lifestyle which entailed a large number of less severe life-events (constituting exposure to large amounts of stress) was found to be negatively associated with breast cancer. Perhaps this occurs as these women were more used to experiencing stress, so the impact of a major life-event was perceived as less severe.

Cooper et al’s (1989; 1993) findings further suggest that in the diathesis-stress model, stress does not act as an objective factor operating on the person. As stated by Dykema, Bergbower & Peterson (1995), the actual perception of a stressor and the perception of impact of the stressor, is also a function of the predisposing personality characteristics of the individual. Pessimistic people can easily ‘make mountains out of molehills, and molehills out of nothing’. This would suggest support for the Cascade model of illness, as outlined by Dykema et al (1995), wherein explanatory style on its own is considered
Cooper et al (1989) state that to that point in time, almost no studies had examined the inter-dependent and complex relationship between personality and adverse life-events in the causation of cancer. Most studies had either researched pre-disposing personality characteristics and cancer, or the adverse life-events and cancer. Their research on the various psychosocial risk factors, reported in the studies cited above, were drawn together in their 1993 paper in which the nature and strength of the inter-relationships between breast cancer and antecedent stressful life-events, coping skills and personality were reported.

In summary, the findings of Cooper et al (1989; 1993) and Dykema et al (1995) correspond with Grossarth-Maticzek et al’s (1994) reports that a stressful life event (suffering at the hands of the Nazis) was correlated with poorer health outcomes (nearly twice as much cancer and CHD) especially when combined with pre-disposing personality characteristics. The effect of the stressful life event was not equally damaging across the whole experimental group, with personality interacting with the life event synergistically to produce more illness. On the balance of these findings, it would appear that the Cascade Model of disease is superior in predicting health outcomes than the Diathesis-Stress Model. Literature provided in this chapter suggests that Grossarth-Maticzek et al’s (1994) reports are consistent with other reports in finding a place within existing health psychology models and constructs. The next chapter will review these reports in regards to another vital construct within health psychology, Learned Helplessness.
Chapter 6

The Role of Learned Helplessness in Cancer:

If Grossarth-Maticek’s claims are to be given any credence, his results need to be explicable according to standard psychological theory. Seligman’s (1991) cognitive approach provides a working model in regards to the psychological component of the multi-factorial equation leading to chronic disease.

A major component of Eysenck’s (1987) theory is that the immune system is compromised by the experience of stress, in particular the type uncontrollable stress which leads to the experience of learned helplessness and consequently to depression. Eysenck (1987) argues that it is neither environmental factors nor personality characteristics which ‘cause’ stress; rather, there is a large role played by the individual’s evaluation of the environmental challenge which leads to the experience of stress - cognition plays an important role.

Learned Helplessness is a cognitive psychology construct introduced by Seligman & Maier during the late 1970’s which relates to the causation of stress and depression (Seligman 1991). The model suggests that animals and humans experience as stressful situations where they perceive that they have no power to avoid or escape aversive consequences. Extreme stress is experienced in situations where the person perceives no control over the environment, and depression is seen as being a likely outcome. What has been learned in the situation is the expectation that regardless of what the person does, s/he will be unable to avoid or escape the aversive condition. The relevance of this to Grossarth-Maticek’s is shown in Eysenck’s (1987) statement that “chronic helplessness correlated with cancer 0.59, and rational and anti-emotional behaviour 0.51. These are very high coefficients indeed in a predictive study” (p.90).

An important component of this expectation of impotence is the attributional style which people use to understand their experience. A problem in the early Learned Helplessness
model was the question of why people would persistently blame themselves (and become depressed as a result) if they thought their misfortunes were due to external or chance factors. Attribution theory has been added to this model of depression, suggesting that it is when people attribute the aversive condition to internal causes which represent global and stable conditions, they are likely to become depressed. For example, the cognitive set of “This event is all my fault; it happened because I am lousy at everything I do; I will always be lousy at things I do”, is likely to lead to the type of depression which Eysenck (1987) suggests is immune system compromising.

Comer (1995) states that a review of nearly 100 studies has revealed that people who believe that their lives are at the whim of external forces or luck tend to experience higher levels of depression. In addition, he cites animal studies which have demonstrated that uncontrollable aversive events for rats lead to a lowering of norepinephrine, a depletion of which has been found in depressed humans. Sweeney, Anderson & Bailey (1986 p.974) report the findings of their meta-analysis of 104 studies involving close to 15,000 subjects in which “several attributional patterns had reliable associations with depression scores”.

Seligman’s (1991) theory suggests a strong association between a pessimistic cognitive style, learned helplessness and depression- the pessimistic set of expectations is viewed as ‘the great modulator’ of learned helplessness.

“helplessness becomes hopelessness and escalates into full blown depression when a person explains his failures with permanent, pervasive, and personal causes.” (Seligman 1991. p 285).

Sweeney, Anderson & Bailey(1986) conducted a meta-analytic review and concluded that explanatory style (pessimism/optimism) was found to be reliably associated with depression. People with cognitive styles described as optimistic resist helplessness. The components of the pessimistic cognitive style which Seligman (1991) describes include the following:-
1) Personalization- the belief that unfortunate events are caused by personal-internal faults; and/or the belief that fortunate events are caused by environmental-external factors. (The alternative, less problematic cognitive style is to attribute successes to internal causes and failures to external causes).

2) Pervasiveness- the belief that failure experiences can be attributed to universal explanations, ie. the tendency to view a particular problem as symptomatic of an all pervasive larger problem which adversely affects their whole lives. (The optimistic alternative is seeking a specific explanation for a particular problem, which may still lead to a sense of helplessness, but which is however limited to the troubled area of life.)

3) Permanence- the belief that bad events will persist and will always exert a negative influence on the person’s life- the causes of bad events are viewed as being permanent. (The optimistic alternative is to view bad events as having temporary causes).

Contemporary learned helplessness theory states that when people experience negative life events and use the pessimistic cognitive style described above, they are likely to experience learned helplessness and consequently depression- as such, it is also characterized as a diathesis-stress theory of disease. Seligman (1991) states that learned helplessness not only produces the feelings of depression, but a wide range of animal and human experiments have demonstrated that it also affects the body at the cellular level and weakens the immune system. He suggests a possible biological pathway, similar to that of Eysenck:- catecholamines, a set of neurotransmitters, become depleted during depression. This leads to an increase in the activity level of endorphines, which is detected by the immune system, leading to a turning down of the immune system. Seligman (1991) cites experimental evidence from animal and human studies demonstrating the immuno-suppressive effects of pessimism and depression.

Kamen-Seigel, Rodin, Seligman & Dwyer (1991) describe their study with 26 older people which demonstrated a link between attribution style and immune functioning. Subjects who habitually use the pessimistic style to explain bad events were found to show poorer immune functioning as measured by T4/T8 ratio and lower responses to
mitogen challenge. These results held even when other contributory factors, such as depression, current level of health, age, weight change, alcohol intake and sleeping patterns were statistically accounted for.

In regards to health over the lifespan, Seligman (1991, p.173) states

“Across a lifetime, an optimistic person will have fewer episodes of learned helplessness than a pessimistic person will. The less learned helplessness experienced, the better shape the immune system should be in.”

As evidence for the notion of a long term effect over the lifespan, he refers to the longitudinal study begun by the William T. Grant Foundation in the mid 1930's, which researched various health factors of 200 men over the course of the ensuing five decades. Using a technique referred to as CAVE (content analysis of verbatim explanations), statements made by the subjects in diary entries years earlier were analysed and rated according to their pessimism or optimism contents (Seligman 1991). Seligman and colleague, George Vaillant made these conclusions:- health was related to an optimistic cognitive style, while ill health was related to a pessimistic style; the effects of the pessimistic cognitive style on health were not evident in the sample until around the age of 45, after which optimism was significantly related to better health; how fast and how severely the health of the pessimists began to fail was accurately predicted by their degree of pessimism 25 years earlier when the subjects were around 20 years of age (Seligman 1991).

In a study of 238 terminally ill cancer patients Schulz, Bookwala, Knapp, Scheier & Williamson (1996) found that a pessimistic outlook served as an important mortality risk factor among younger patients (30-59), but less so for older patients (over 60). They concluded that psychological factors like pessimism may have less of an impact in terms of cancer mortality for older terminal cancer patients, as it is more normative for that age group, and may in fact represent an adaptive response to their reality. Where as for the younger age group, pessimism is less normative, as is cancer, leading to more negative
behavioural consequences. They make the point that as a younger person with the expectation that much life is yet ahead, the psychological and physiological reality of cancer is likely to be different compared to the older person. As such, their results demonstrated a clear relationship between pessimism and cancer mortality for the younger group only, and they suggest that psycho-social factors become less important to mortality as the person gets older (i.e. over 60).

In a study of 1544 Finnish women undergoing screening for breast cancer Absetz, Aro & Sutton (2002) found that lower optimism among relatives of ovarian cancer patients was associated with higher perceived risk of the obtaining the condition and subsequent distress. They concluded that optimistic coping styles are beneficial in that they are associated with less psychological distress in regards to cancer risk, i.e less anxiety and depression; whereas a pessimistic attributional style was associated with more anxiety and depression about the prospects of obtaining cancer.

In summary of major research findings, Dykema, Bergbower & Peterson (1995) state that previous studies have found links between pessimistic explanatory style and poor health operationalized in the following ways:- by physician examination, self reported symptoms, immuno-suppression, and survival time with cancer and heart disease.

Apart from the immuno-suppressing qualities of pessimism-learned helplessness-depression, other possible reasons for the demonstrated links with ill health include the following. Bad events also weaken the immune system, as demonstrated by Ader et al (1992) and may make a person more vulnerable to conditions like breast cancer (Cooper & Faragher 1993). Seligman (1991) states that pessimists experience more bad events in their lives compared to optimists, as they are more passive and take fewer steps to prevent or deal with the occurrence of bad events. Also, when trouble strikes pessimists, being more passive, they take fewer steps towards gaining social support (which has also been linked with health outcomes- Syme 1998). Finally, when ill health occurs, pessimist are more likely to believe that it is attributable to permanent, pervasive and personal
causes— as such, they are less likely than optimists to seek out medical advice or to stick to health regimens or prophylactic health behaviours.

Beyond the role played by attributional style in chronic disease, another psychosocial risk factor which must be taken into consideration is gender role, the study of which may also add pieces to the ‘jig-saw puzzle’ which medical risk factors alone are not able to answer.
Chapter 7

Gender, stereotypes, personality and disease:

The study of gender differences and major diseases such as cancer and CHD provide clues as to the operation of psychosocial factors in the causation of these conditions, as men and women have such different social experiences based on their genders. There are substantial biological differences between men and women in terms of hormones and chromosomes which are considered important in the causation of these conditions. There are also substantial differences in the psychological and interpersonal styles of men and women, as well as very different experiences of social support and isolation, coping styles, communications styles, gender roles etc. As such, a study of gender in relation to cancer and CHD has the potential to clarify some of the psychosocial factors which may contribute to the causation of these conditions- males and females represent two different cohorts in a naturalistic observation of health outcomes.

Farrell (1993, p 182, 393) reports from figures supplied by the United States Department of Health and Humans Services, that men die of heart disease compared to women at a ratio of 1.9 to 1. (taken from U.S Department of Health and Humans Services, National Centre for Health Statistic, Centres for Disease Control, Monthly Vital Statistics Report, vol 38, no.5, supplement, September 26, 1989, "Advanced Report 2 of Final Mortality Statistics, 1987", p6.Table D, "Ratio of Age-Adjusted Death Rates for the 15 Leading Causes of Death by Sex and Race: United States, 1987"). He adds that men in the U.S die earlier from all fifteen of the leading causes of death, including for example deaths from cancer at a ratio of 1.5 to 1 when compared to women. Australian figures show a similar pattern to those from the U.S. Abraham, d'Espaignet & Steveson (1995) in a survey of Australian health trends from 1983-1993 provide the following statistics.
Table 9. Death rate for CHD per 100,000 population.

<table>
<thead>
<tr>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Males</td>
<td>310</td>
<td>304</td>
<td>310</td>
<td>289</td>
<td>280</td>
<td>268</td>
<td>268</td>
<td>248</td>
<td>235</td>
<td>233</td>
<td>217</td>
</tr>
<tr>
<td>Females</td>
<td>161</td>
<td>155</td>
<td>159</td>
<td>153</td>
<td>150</td>
<td>142</td>
<td>144</td>
<td>136</td>
<td>127</td>
<td>131</td>
<td>115</td>
</tr>
</tbody>
</table>

(note: Coronary Heart Disease is classified according to the International Classification of Diseases (ICD-9) Codes: 410-414).

The figures shown in Table 9 indicate that over the decade 1983-1993, 2974.80 men died of CHD, while 1576.8 women died of CHD. This represents a ratio of 1.89 men dying of CHD for every 1 woman that dies of CHD. The death rate of both males and females has declined over that period (men’s by 3.8%, and women’s by 3.3%), however the death rate of men is nearly twice that of women, as with the comparable American statistic.

Table 10. Death rate for all cancers per 100,000 population.

<table>
<thead>
<tr>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Males</td>
<td>239</td>
<td>234</td>
<td>246</td>
<td>238</td>
<td>238</td>
<td>242</td>
<td>241</td>
<td>238</td>
<td>234</td>
<td>238</td>
<td>235</td>
</tr>
<tr>
<td>Females</td>
<td>141</td>
<td>139</td>
<td>144</td>
<td>143</td>
<td>139</td>
<td>142</td>
<td>140</td>
<td>140</td>
<td>142</td>
<td>139</td>
<td>141</td>
</tr>
</tbody>
</table>

(note: cancers are classified according to the International Classification of Diseases (ICD-9) Codes: 140-208).

The figures shown in Table 10 indicate that over the decade 1983-1993, 2627.50 men died of cancers, while 1554.00 women died of cancers. This represents a ratio of 1.69 men dying of cancers for every 1 woman dying of cancers. This Australian statistic is more extreme than the comparable American one, which has men dying of cancers at only a 50% higher rate than women. More recent statistics, provided by the National Heart Foundation of Australia (Heart Facts 2003) state that in the year 2000, men across all age groups were 2-3 times more likely to die from cardiovascular disease, and were twice as likely to have a coronary event than were women.
In light of the Australian and U.S statistics presented above (Abraham et al. 1995; Farrell 1993), gender must now be considered one of the risk factors which operate in a synergistic fashion with other risk factors. Farrell (1993) attributes these disproportionate death rates to the effects of industrialization and the new adverse roles in life which industrialization brought primarily to men. He points out that the phenomenon of shorter lives for men as compared to women only occurs in industrialized societies— in pre-industrial societies, this is not the case, suggesting that it is not the biology of men responsible for their higher rate of premature deaths, but it is the social role ascribed to them by industrialization. However, being a male is likely to be both a physical risk factor as well as a psycho-social one. As an example of the biological risk factors, Farrell (1993, p184) states that

“women’s double X chromosomes give them a kind of genetic backup system. That is, if a woman has a defective gene along one thread of her X chromosome, the odds are very high that the matching gene on the other X will be perfect. Men don’t have that back up system”.

Eisler & Blalock (1991) state that estrogen appears to exert a protective influence for women against heart disease. However Harrison, Chin & Ficarrotto (cited in Eisler et al. 1991) reviewed the mortality rates of both sexes throughout the lifespan and concluded that after childhood, biological factors alone were unable to account for the women’s superior longevity compared to men, while Waldron (also cited in Eisler 1991) estimates that 75% of the differences in life expectancy can be accounted for by psycho-social factors. In suggesting that not only biology is at play here, Farrell (1993) states that between the ages of 1-4, boys die only slightly more often than girls. During the ages of 15-24, when adult sex roles are first experienced, men die at a rate almost three times that of women.

It has been suggested by various authors (Farrell 1993; Thomas 1993) that the social roles of men, and the interpersonal communication and social experiences of men which are linked with these social roles, pose a range of risk factors for men. The damaging aspects
of the male social role are often stated as being lack of social and emotional support; social isolation; estrangement from the family; lack of choices in terms of traditional or non traditional roles; preponderance of men in the ‘death industries’, eg. the military, construction work, heavy industry, etc; extreme stress due to all of the above.

Eisler & Blalock (1991) state that acquired psychosocial characteristics associated with the masculine gender role can be either directly or indirectly linked with the seven leading causes of premature death in men. For example, men consume more alcohol than women, perhaps as a method of reducing stress without violating male norms while manifesting masculine ‘toughness’. That men die more in accidents of all kinds is difficult to explain biologically- it is a reflection of men engaging in higher risk work and recreational activities than women, encouraged by male socialization messages thereby validating masculinity.

Regarding male role stress in industrial societies, Farrell (1993) suggests that the problem is industry’s need for men to work away from the home, removing them from the social support available from family and local community. In addition, traditional socialization messages still encourage males to subscribe to notions of being the provider for and protector of women and children. While few men now days will be called upon to physically protect others, the provider role is still primarily that of men- more women do contribute to that role now compared to previous generations, however Farrell (1993) points out that this is usually a matter of choice, rather than being expected of the woman. This assertion may have less validity in Australian society of 2006 than it had in America during 1993, although Carroll (1998) states that Australian women, as of the late 90’s, still saw themselves primarily as mothers and as ‘nest makers’, while men still primarily saw their role as that of provider. Farrell (1993) suggests, that men have fewer choices available to them as to whether they want to be the main income provider or not. In the protector role, and perhaps also in the provider role (especially if engaged in reluctantly), rescuing behaviours create ‘emergency hormones’, such as testosterone which weaken the immune system, and adrenaline which makes blood clotting and heart failure more likely to occur.
Farrell (1993) states that patterns of male interaction such as joking ‘put downs’, being the main form of adolescent bonding cause men to experience more isolation and loneliness than do women who bond via mutual emotional support. Drawing on psycho-social research into illness, he suggests that loneliness (lack of social support) is a strong predictor of heart disease. It is interesting to note here that C.B Thomas (cited in Eysenck 1994 p 186), who conducted the longest continuous study of health with a sample of subjects, found social isolation to also be highly predictive of cancer over a 40 year span, in that those designated as ‘loners’ and suppressors of emotions were sixteen times more likely to develop cancer. In addition to social isolation, Farrell (1993) points to men’s tendencies of keeping their problems to themselves, rather than articulating them and seeking support from others- the stress which this creates is assumed to further damage the immune system.

In summary, Farrell (1993) points towards a combination of men’s roles in industrialized society; fewer choices available in terms of such roles when compared to women’s expanded range of choices; men’s interpersonal styles, including on-going criticism towards each other and lack of mutual support; the role of having to appear strong in order to protect others from feeling insecure were a partner to know of his vulnerabilities and stress; and the ensuing sense of loneliness, isolation and stress which this creates as being causal in men’s mortality figures- it is the combination of these factors which, according to Farrell (1993) means that before reaching the age of 50, men are four times more likely to suffer heart disease than women.

Men’s emotional and interpersonal styles, cancer & CHD:
To the above list of psychosocial risk factors for men must be added the emotional, interpersonal and communication styles which are often attributed to men, i.e. there is often a lack of intimacy with other people, leading to chronic isolation; a lack of giving, receiving or seeking emotional support; a tendency to deny the experience of emotional troubles; a lack of ability to communicate one’s negative or disturbing emotions to others; a tendency to be comfortable with emotional expression only in the form of anger.
These gender stereotypic attributes of men can be seen to coincide with the cancer and CHD profiles as described by Grossarth-Maticek & Eysenck.

The role of traditional personality measurement constructs add only a little light to the question of gender and disease. Eysenck, Eysenck & Barrett (1995) report that in both American and British studies conducted to develop standardization groups for the Eysenck Personality Questionnaire, men have always scored higher on the Psychoticism scale (with the primary traits of aggressiveness, impulsivity, coldness, lack of empathy, tough mindedness), while women have always scored higher on the Neuroticism scale (with the primary traits of anxiety, guilt, tenseness, low self esteem, depression)- there is little evidence of any gender difference in terms of Introversion/Extraversion, and women have been consistently measured to be more anxious, less independent and less assertive than men, although it is not established whether these are inherent differences or socially trained ones.

Both cancer and CHD personalities are characterized by Grossarth-Maticek & Eysenck (1990) as a *failure to cope adequately with interpersonal stress*. Beyond that common feature, the cancer personality profile is characterized by -

1) *Suppression of strong negative emotions*, such as fear and anger, and behaviours appropriate to these emotions, eg. assertiveness, aggression, confidence, dominance, selfishness, and

2) *inappropriate coping mechanisms*, leading to failure, feelings of hopelessness and helplessness and finally depression and despair (Eysenck 1994 p169).

The CHD personality profile is characterised by –

*an inability to cope with stressful situations, and the development of strong anger, aggression and hostility responses.*
One of the stereotypic images of men (i.e. freely express anger) has a reasonable fit with the CHD prone personality profile. Grossarth-Maticek & Eysenck (1991) however, make the point that Type 2 personalities often have difficulties expressing their anger – as such, they suggest that the presence of feeling anger, hostility and aggression is the key factor, not its free expression. In regards to cancer, the relationship between gender and mortality is less strong than it is for CHD (1.9:1), with the Australian ratio being only 1.69 male to 1 female deaths– it is a significant and consistent ratio, even if less than that of CHD (Abrahams et al 1995). Again, the possibility is raised, as Grossarth-Maticek & Eysenck (1990) suggest that a different personality characteristic in the interpersonal domain is one of the causal agents in cancer. Because men die of cancer at nearly 70% higher rates than do women, it is reasonable to suggest (if there is substance to Grossarth-Maticek’s 1990 assertions) that the related cancer prone personality characteristic, like the CHD prone personality, is also more male than female. Unlike the CHD personality profile, however the cancer personality profile fits less with the instrumental stereotype image which ‘the male personality’ has in our culture. Perhaps it can be said that while the cancer prone personality is more likely to occur in males, it will also occur in females but to a lesser extent than it does in males, although this is yet to be demonstrated. If there is truth in the stereotypes of men’s interpersonal styles, then this may be reflected in the fact that although men die of both cancer and CHD at much higher rates than do women, men die of CHD at higher rates than they do of cancer. So perhaps both cancer and CHD prone personalities can be seen as more characteristic of men, but the CHD profile is more characteristic of more men, as reflected in the higher amounts of male deaths from CHD than from cancer.

**Depression, gender and cancer:**
As a gender question, it is worth delving further into the role of learned helplessness and pessimism in the causation of cancer. Seligman (1991) states that current research shows that women experience around twice as much major depression as men, however Australian men are around 70% more likely to die of cancer than are women (Abraham et al 1995). Seligman (1991) further states that no research of randomly selected populations has demonstrated that women are more pessimistic that men, however pre-
pubescent boys have been found to be more pessimistic and depressed than pre-pubescent girls. At around puberty, the gender-depression ratios ‘flip’ in that girls begin to become more depressed than boys, heading towards the 2:1 ratio as found in adulthood. Seligman (1991) explains this ratio in terms of women’s tendencies to think (ruminate) and emote in the face of difficulties, which leads them into episodes of major depression at twice the rate of men. Men, on the other hand tend to respond to difficulties and the ensuing sub-clinical depression with a variety of distracting strategies, eg. denial; decision to not concern themselves with their mood; play sport, or engage in other enjoyable activities; and get drunk. The suggestion is that both men and women experience the same amounts of mild, or sub-clinical depression- women respond to this by focusing on and expressing their emotions (leading to higher levels of major depression for women as emoting involves ruminating), while men successfully attempt to distract themselves with action before the depression becomes clinically significant.

According to Eysenck & Grossarth-Maticek’s theories, the prospect that women are more emotion focused and expressive, albeit while experiencing episodes of major depression ensures that the effects of the major depression are not as harmful to immune functioning- an outlet for negative affect is created. Men, on the other hand succeed in preventing episodes of major depression at twice the rate than do women. However, the fact that they employ strategies to combat sub-clinical depression which are part of the supposed cancer prone personality profile (ie. denial, distraction, repression-the non expression of strong negative emotions, etc) ultimately increases their risk of immuno-suppression and consequently cancer. This accords with Grossarth-Maticek & Eysenck’s (1991b) statement that depression is not a homogenous disorder, and his suggestion that sub-clinical depression is the most ‘cancer promoting’ type. Eysenck & Grossarth-Maticek (1991b.p2) make it clear that when they use the term ‘depression’, they are referring to :-

“sub-clinical...hopelessness depression...largely based on the work of Seligman…Theory links (this) one particular type of depression with cancer; it does not predict any connection with other types of depression”.(Eysenck 1994.p203)
In attempting to make sense of this, it is suggested here that it may not be the incidence of depression (major or sub-clinical) which causes the immuno-suppression, but the coping strategy employed in response to the depression (of not expressing strong negative emotions) which causes the immuno-suppression. The possible link between sub-clinical depression and cancer (higher in men) is perhaps a reflection of the possibility that sub-clinical depression is likely to be responded to with the range of health damaging yet ‘effective’ coping strategies which men are more inclined to employ compared to women. This would be in accordance with Eysenck & Grossarh-Maticek’s theory, as well as the over representation which men have in cancer mortality, despite the higher incidence of major depression in women and in men. It appears that cancer prone personalities (higher in men) could be less severely depressed but more emotionally repressed. As Eysenck (1994 p201) states,

“The long established theory in relation to neuroticism and depression, strongly supported by many studies already reviewed, is that it is the *suppression* of emotion that is associated with cancer.”

This is obviously a gender issue, particularly if men are more inclined to suppress their strong negative emotions as a coping strategy to uncontrollable stress than are women.

The male gender role and disease:
Eisler & Blalock (1991) offer a comprehensive discussion of the emotional and interpersonal constraints which the masculine gender role imposes upon men- the following section will draw on their observations and research.

Gender, as opposed to biological sex, is a term which refers to the social construction of what it means to be a male or a female in our culture. Within the concept of masculinity are a range of cognitive masculine schemata which, through a thorough indoctrination-socialization process, individual men are encultured with to greater or lesser degrees. There is evidence that through successfully learning the prescribed masculine behaviours,
individual males are able to obtain highly valued social approval and avoid social condemnation. As these prescriptions are so pervasive across age, educational, racial, ethnic and political belief boundaries, there is a common assumption that they must be biologically inherent in men, as opposed to being socially learnt. These cognitive masculine schemata are prominent in guiding the emotional and behavioural repertoires of men as well as contributing to the experience of stress.

Eisler et al (1991) are not suggesting that all masculine behaviours and values are damaging to health, however they are hypothesizing that the imposition of masculine values at times does produce greater gender role stress for men, with consequent unhealthy coping behaviours. A strong commitment to masculine gender role cognitive schemata acts to restrict the type of coping responses which an individual male might employ in the face of stress. Research cited by Eisler et al (1991) demonstrated that stress inducing tasks, when perceived by a highly masculine man as being a challenge to his masculinity, resulted in significantly higher reports of stress (measured by systolic blood pressure reactivity) compared to tasks which were not perceived as a challenge to masculinity. This demonstrates that a rigid adherence to a masculine gender role actually determines what situations men will experience as stressful- often very different situations compared to women, or to men with less adherence to masculine gender roles. How men respond to stress is also heavily influenced by the masculine gender role, internalized as the gender schemata.

A prominent gender cognitive schema is a commitment to masculinity, resulting from a fear of being associated with ‘inappropriate’ feminine values, attitudes and behaviours. Masculine scripts of toughness and self reliance ensure a reluctance towards self disclosure of vulnerabilities, and act to prevent the seeking of advice or support from others. Masculinity requires the presentation of the man as being highly rational, non introspective, and capable of managing all problems. This leads to a difficulty with admitting problems or vulnerable feelings to others, naturally mitigating against the expression of negative emotions. As such, a coping strategy which women successfully employ, ie. expressing one’s negative feelings to others and thereby gaining emotional
and practical support, is restricted out by the masculine gender schema. Males learn that emotional expression is considered a feminine trait at the time when they are learning to distance themselves from femininity in general. Rather than just being a stereotype, studies (cited by Eilser et al 1991) demonstrate that men are more socialized to avoid the expression of emotions compared to women.

“Self report and behavioural measures show that women reveal more intimate details about themselves, and are more expressive of emotions like fondness, pleasure, love, happiness and sadness” (Eilser et al 1991.p 55).

In relationships, the lack of emotional expressiveness of men appears to be one of the reasons for their difficulties in maintaining supportive relationships. Women become overburdened with the sole responsibility for emotional expression, and men find it hard to have their emotional needs met to the extent that they deny these needs and/or are unable to express them. Even if men were as willing to disclose their strong negative emotions to others, they would require a social support network to do this with, which they are less likely to have compared to women, again because of masculine gender schema. Social support acts as a buffer against the effects of stress, and its availability during stressful times is dependent upon the quality of the relationships prior to the crisis. Studies cited in Eilser et al (1991) demonstrate that women tend to have more close and intimate friendships than men- women value the affective aspects of their friendships, eg. mutual understanding, intimacy and closeness; while men have fewer friendships, and tend to value the more instrumental aspects, such as shared interests and activities. Consequently, women foster more supportive relationships with each other, and are able to draw upon them during times of crisis when they need more social support; while men do not foster this element in friendships to the same degree, and are less able to access social support during stressful periods. Eisler et al (1991. p57) states that

“social support has been found to be universally related to coronary artery disease in Type A individuals...(and)...it is possible that social support reduces harmful neuroendocrine activity”.

122
Women’s willingness to express strong negative emotions may be one of the psychosocial reasons why they are less susceptible to cancer and CHD; and men’s suppression of strong negative emotions as well as their lack of expression, may be one of the major contributors to their over represented mortality from cancer and CHD. The male instrumental coping style and rational appraisal of situations is not in itself especially harmful- the harm emerges when this coping style is used as an inflexible goal unto itself to the exclusion of other, more ‘feminine’ coping styles such as emotional expression and gaining support.

One of the few emotional expressions which the masculine cognitive schema will allow is that of anger, hostility and aggression. The socialization of boys encourages them to utilize highly competitive and aggressive strategies in challenging situations, which they learn to internalize in order to feel secure and respected in their quest to be men. Where there is a rigid adherence to this style of coping strategy across all situations, the man is likely to experience a greater degree of stress. For example, while desiring a degree of control over one’s environment is adaptive, if it is rigidly insisted upon in a contemporary relationship where there is an expectation of egalitarianism, then the relationship will suffer, and the man will experience more stress.

In summary, Eisler et al (1991) argue that there is a sound empirical basis to gender differences in terms of their perceptions of situations as being stressful; and in terms of men’s smaller range of choices available in coping strategies. Rigid adherence to traditional masculine gender schemata and behaviour limits the range of coping strategies available to individual men during times of stress. The coping strategies available to men appear, according to Grossarth-Matricek and Eysenck’s (1990) conclusions, to be the most unhealthy options, reflecting both the Type 1 & 2 behaviour patterns.

**Type A and sex role identity:**
Psychological researchers over the past 20 years have investigated the relationship between high Type A scores and what has traditionally been referred to as ‘masculinity’.
It is worth noting again here that while there are problems associated with the Type A construct, researchers in the field still consider it to possess *some* useful components in terms of predicting heart disease risk. Booth-Kewly and Friedman (1987), after conducting a quantitative review of the literature of psychological predictors of heart disease, concluded that anger and hostility, and to a lesser extent depression are predictive of heart disease.

Kopper (1993) states that past sex role research has concluded that subjects who were identified as having a masculine sex role (like the Type A profile) were more prone to anger, and more likely to express it externally towards objects or other people; they were less likely to attempt to control or manage the expression of their anger compared to subjects with a feminine or undifferentiated sex role. In these studies, sex role has usually been assessed by a tool such as the Bem Sex Role Inventory (BSRI), or the Short Bem Sex Role Inventory (SBSRI). The BSRI consists of 60 descriptive adjectives that the respondents rate on a 7 point Likert scale as to how descriptive of them the words are—there are 20 masculine items, 20 feminine items, and 20 fillers which are gender neutral.

Hunt (1993) suggests that the researcher would be mistaken to assume that the sex of the respondents would always concur with the masculinity or femininity rating on the BSRI, i.e. there is no reason to predict that any particular man would necessarily score high on the BSRI masculinity scale, and any particular women would score highly on the BSRI femininity scale. She suggests that rather than talk in the traditional terms of masculinity and femininity, the discussion is currently in terms of instrumental (eg. independent, assertive) and expressive traits (eg. nurturant, sympathetic), which can be found in a subjects of either gender. Kimlicka, Sheppard, Sheppard & Wakefield (1988) suggest that by the late 1980’s the trend in sex-role identity research was to assume that the characteristics which our society calls masculinity and femininity were likely to be found in varying degrees in all individuals. When the scale was devised by Bem in the 1970’s, perhaps it could have been used as a valid measure of sex role per se, but the social changes of the past 25 years, according to Hunt (1993) means that it is best thought of as a measure of these two adaptive personality traits of instrumentality and expressiveness.
In order to retain the use of terms by most of the researchers referred to here, it can be understood that ‘masculinity’ is referring to that adaptive personality trait known as ‘instrumentality’, and ‘femininity’ is referring to that adaptive personality trait known as ‘expressiveness’.

In a study with 217 subjects, testing the relationship between Eysenck’s personality dimensions and Bem’s factors, Kimlicka et al (1988) found there to be a positive correlation between Bem’s Masculinity/instrumentality factor and an assertive type of Extroversion, rather than with the ‘coldly assertive and dominant’, or Psychoticism in Eysenck’s dimensions.

De Gregario & Carver (1980), Nix and Lohr (1981), and Batlis & Small (1982) all found subjects high on Type A behaviour patterns to also be high on scores of masculinity, and an inverse relationship to femininity scores- these findings were true for male as well as female subjects. In a study comparing the results obtained from 40 female and 41 male college students, Hellbrun, Wyders & Freidberg (1989) concluded that women who scored high on the Type A measure also scored high on the masculinity measure. Similar results were obtained by Kopper (1993), who compared the data from 407 female and 222 male college students- she found that sex role identity was significantly associated with anger expression (a Type A component), ie. masculine sex role types were more prone towards anger; more likely to express anger outwards, and less likely to control or suppress the expression of their anger. Conversely, she found that feminine sex role types were more likely to attempt to control the expression of their anger, suppress anger, and less likely to express it outwards. Interesting to note was the finding that Type A people showed higher levels of trait anger and anger proneness, but they also showed a greater tendency to hold in or suppress angry feelings. If Type A people are also high in masculinity, their profile begins to appear as:- high levels of trait anger/anger proneness, and a high likelihood of expressing anger outwards, and less suppression of their anger. If Type A people are high in femininity, then they are likely to have a higher trait anger/anger proneness, but unlike their high masculinity counterparts, are more likely to suppress angry feelings, not giving them expression. According to Grossarth-Maticek and
Eysenck’s research, this would suggest a proneness more to cancer than to CHD, even though they are Type A responders.

In regards to women, Type A behaviour patterns and health outcomes, Wright et al (1994) state that while there is evidence for predictability of heart disease with high Type A men, this has not been established prospectively against any major health outcomes for women- the differences in physiological reactivity which have been noted between Type A and Type B men have been found to be less reliable and less pronounced in comparing Type A & B women. They suggest that this means that Type A behaviour patterns manifest themselves differently in men and women, with the picture emerging that Type A women are physically and psychologically healthier than Type A men- less prone to heart disease. While Hellbrun et al (1989) found that Type A women have an abundance of masculine Type A traits (such as argumentative, cynical, fault finding, opinionated, self centered, tough), Wright et al (1994) found that Type A men have higher anger scores compared to Type A women- these results imply that high Type A women follow a different subcomponent route compared to high Type A men in achieving their high Type A status- men, unlike women, achieve the status through the subcomponent route of outwards expression of anger with fewer attempts at anger suppression or control.

Kopper (1993) reports that Type A people with a feminine sex role identity have higher proneness to anger as a personality trait, so it would appear that the extreme expression of anger (seen in aggressive and hostile behaviour), rather than the existence of anger per se, is a possible factor in the psychological profile of CHD proneness.

The BSRI femininity factor, or as Blanchard-Fields, Suhrer-Roussel & Hertzog (1994) prefer to term it nurturance (or communion), can be seen to have some common features with the Interpersonal Reactions Inventory Type 1 (cancer prone) personality. The IRI cancer prone personality is characterized by the following behaviours:- unassertive, non-aggressive, unconfident, non-dominant, unselfish. This interpersonal style suggests a preference for communion with other people, rather than an exertion of will over others. In its more neurotic manifestation, a high need for communion with others may show itself in a lack of emotional autonomy- Grossarth-Matick & Eysenck (1991b .p2) use
Mowrer’s definition of neurosis as being an “emotional dependence which prevents such people from making independent decisions in the light of their own best interests”.

Grossarth-Maticek & Eysenck (1995. p781), in reviewing theories of the healthy personality characterize it in the following terms,

“autonomy, emotional independence, and self regulation, ie. the ability to actively regulate one’s own life, without a degree of emotional dependence on other people”.

Is this more characteristic of men or women? If Grossarth-Maticek & Eysenck’s (1990) reports are correct, the higher rates of deaths from cancer in men would suggest that it is a less common feature for men, despite the stereotype- however, that is also assuming that personality factors acting alone are responsible for cancer and the validity of Grossarth-Maticek’s research. That communion, as a personality characteristic measured by the BSRI, is more correlated with women suggests that it may be a cancer risk pattern only when it goes to such an extreme as to be at the expense of emotional autonomy. It may go to this extreme more in males than in females, although as a general personality characteristic it may be more typical of females than males, as demonstrated by the research.

Because the cancer prone personality, as described by Grossarth-Maticek & Eysenck is more multi dimensional than is the CHD prone personality, it is possible that men may show a similar need for communion taken to a neurotic degree as women (lack of emotional autonomy), however are more vulnerable to cancer because they are generally more emotionally and socially isolated, and tend to repress the expression of their negative feelings more (apart from those who over indulge in the expression of anger, more typical of the Type 2 CHD prone personality). Being lower scorers on the Neurotic scale of the EPQ than women (Eysenck et al 1995), men have less neurotic expression of their negative emotions, even if those negative emotions may be a neurotic dependence on another person and lack of autonomy. It is worth noting as well that an important facet
of the cancer prone personality as posited by Grossarth-Matick (1980) is a tendency towards rationality and ‘anti-emotionality’, which again, as men score lower on Neuroticism, is a more common characteristic of men.

In regards to the stereotypes of women, Kopper & Epperson (1991) state that the experience and expression of anger in women has been a topic of central importance in the study of the psychology of women. One of the main assertions in the literature has been that women experience and express anger differently to men, specifically that they ‘have greater difficulty recognizing and expressing their own anger, resulting in a variety of mental health difficulties’ (Kopper & Epperson 1991. p7). As anger is said to be ‘unfeminine’, it is suggested that women refrain from expressing their anger so as to avoid a clash with their internalized cultural concepts of femininity as well as social disapproval. In regards to the psychological profile for cancer proneness, one would expect that if women were more likely than men to hide or suppress their anger, then they would be more vulnerable to cancer than men, as the non expression of strong negative emotions is a key Type 1 personality characteristic.

Kopper & Epperson (1991) make the point that while speculation on women suppressing anger has been abundant, empirical foundations for these views are scarce. To test the assumption that women are more likely to suppress their anger than are men, Kopper & Epperson (1991) studied 455 subjects, administering a range of relevant questionnaires, including the BSRI. They concluded that, contrary to the stereotype, gender was not an accurate predictor of the expression or suppression of anger; that is men were just as likely to suppress anger as were women. Consistent with other research in this field, they found that sex-role identity was a more robust predictor of the experience and expression of anger; that is, subjects scoring high in Masculinity were more prone to anger and more likely to express it outwardly, while Feminine sex-role types were more likely to control the expression of their anger. It would appear then that being a female does not place one in a higher risk group for suppressing anger.
Perhaps being a person with a ‘Feminine’ sex-role identity (higher anger suppression) is part of the cancer risk factor, and for men, in combination with other psychological risk factors such as rationality and anti-emotionality leads to more vulnerability to cancer. Also, the male tendency of avoiding emotional expression must be considered an extremely important factor here.

There is another possible explanation of the different death rates from cancer and CHD for men and women which is inferred by Grossarth-Maticke & Eysenck’s (1990) suggestions. It may be that while men may consistently show more cancer prone or CHD prone behaviour patterns, women may also show high rates of these behaviour patterns, but in a less consistent, or a more mixed form as per the Type 3 profile which according to Grossarth-Maticke & Eysenek (1995) appears to inoculate the person against both cancer and CHD. Type 3 has been described as an hysterical type by Grossarth-Maticke & Eysenck (1995). While there is no detail provided by these authors in terms of what they mean by ‘hysterical’, the following clues are offered by the current author. With knowledge of Eysenck’s rejection of most things psychoanalytic, it is safe to assume that he is not using the term as did Freud, referring to what is now called a ‘conversion disorder’. It would make sense that he is in fact referring to some of the conditions which, before DSM III, the American Psychiatric Association used to refer to as Neuroses, now referred to by the A.P.A DSM-IV as ‘Personality Disorders’. Perhaps he chose to not use those terms as Eysenck was known to object to the lack of scientific rigour evident in the DSM system and terminology, and in psychiatry more generally (Eysenck, Wakefield & Friedman 1983). That the A.P.A abandoned the term Neurosis was due to recognition that psychiatry’s use of it had made it such an all encompassing term, describing so many personality disorders, that on its own it had become somewhat meaningless- the preferred option for the APA was to introduce into the DSM the category of Personality Disorders with a range of variations.

As Eysenck et al (1995) states, research conducted for developing normative data for his personality tools have consistently shown women to be significantly higher on Neuroticism than men. Borderline Personality Disorder (which prior to DSM III was
considered characteristic of Neurosis) is described by Comer (1995 p. 596-587) in the following ways (with the proposed corresponding Type according to Grossarth-Maticek’s scheme presented in brackets for comparison):

- a pervasive pattern of instability in interpersonal relationships, self image and moods, and marked impulsivity (Type 3)
- major shifts in moods, swinging in and out of intense depressive, anxious, and irritable states that last anywhere from a few hours to a few days (Type 3)
- prone to bouts of anger and hostility (Type 2)
- they are engaged in a conflict between the world around them and the expression of their emotional needs (Type 2)
- physical aggression is just as often directed outwards as inwards (Type 3)
- social behaviour is often confused- they form intense conflict ridden relationships in which their feelings are not necessarily reciprocated (Type 3)
- fearing abandonment, they have difficulty maintaining appropriate interpersonal distance (Type 1)
- quickly become disappointed or enraged when others fail to meet their expectations (Type 2)
- yet they remain intensely attached to their relationships, paralyzed by their fear of being left alone (Type 1)

Comer (1995) states that around 75% of people who receive this diagnosis are women.

From the above, it can be seen that in this personality disorder, there is a high degree of oscillation between extremes- this feature is also characteristic of Grossarth-Maticek’s & Eysenck’s (1990) Type 3 pattern which they suggest inoculates one to cancer and CHD, producing ‘relative health’ rather than optimal health. They propose that the Type 3 person does not spend excessive time engaging in the extremes of either Type 1 or Type 2 profiles, and therefore the oscillation between them neutralizes the effects of each to a certain extent.
In Eysenck’s terms, this personality type is highly neurotic. If there is substance to Grossarth-Maticek & Eysenck’s (1990) reports, the fact that women are more likely to oscillate in the fashion described above by Comer (1995) may explain one of the psychosocial differences between men and women which lead to women contracting far less cancers and CHD’s when compared to men. The Borderline or hysterical personality type is not reluctant to give free expression to strong negative emotions, and as they behave in an erratic oscillating manner, their behaviour is consistent with Grossarth-Maticek & Eysenck’s (1990) description of the Type 3 pattern.

It may be that while most women do not display this oscillation enough to warrant a diagnosis of Borderline Personality Disorder, there is more of a tendency in that direction for women than for men; whereas for men there is more of a tendency towards Psychoticism. Grossarth-Maticek & Eysenck (1991) have also characterized the Type 3 profile as showing tendencies towards psychopathy- Comer (1995) states that when men receive a diagnosis of Borderline Personality Disorder, they are more likely than women to have co-existing conduct, attention deficit and anti social personality disorders (also termed psychopathy); while women are more likely to have co-existing mood disorders and display more self destructive behaviours. There appears to be a difference in how men and women manifest this particular personality disorder, or ‘neurosis’ as Eysenck would refer to it.

In summary, this discussion suggests that women appear to be more versatile in their forms of interpersonal and emotional behaviour than are men, i.e while men may behave in a consistent way characteristic of Type 1 or Type 2 profiles, it is plausible that women are more likely to easily move between the excesses of these two extremes, and while still displaying these unhealthy behaviours, actually inoculate themselves from both cancer and CHD as a function of this versatility. An attempt has been made here to relate findings in gender roles to Grossarth-Maticek’s conclusions. However, not all researchers ascribe this level of credibility to his claims, as will be detailed in the following chapter.
Chapter 8

Empirically based challenges to Grossarth-Maticek & Eysenck:

Apart from the dramatic findings reported by Grossarth-Maticek & Eysenck (1990), Amelang (1997) states that there has only been moderate support in the literature for the cancer prone personality hypothesis. Amelang’s own cross sectional research (Mathews et al 2003) demonstrates that emotional labiality was related to the experience of multiple disease, but not to positive health or to a particular disease state. Mathews et al (2003) conclude that the associations between psychological factors and disease are statistically significant but small. Amelang (1991) suggests that it is due to the size of Grossarth-Maticek’s extraordinary claims that they have come under special scrutiny, and now attempts at independent replication (Amelang, Schmidt-Rathjens & Mathews 1996).

Many of the critiques of Grossarth-Maticek and Eysenck’s reports that appear in the 1991 special edition of Psychological Inquiry express concerns over methodological factors and queries in relation to statistical treatments of the data. At that time there had been no large scale prospective replications of these studies, which would constitute the ultimate form of supporting evidence or dismissal. Amelang & Schmidt-Rathjens (1992) embarked on a large scale replication study, attempting to independently test the findings of Grossarth-Maticek & Eysenck, the results of which will be discussed in a later section. They state that the conclusions reported by Grossarth-Maticek & Eysenck demonstrate a range of problems related to theoretical confusions, obscurities of data analysis and inconsistencies within the material published. Amelang et al (1996) have published findings from a cross sectional analysis in which they were attempting to ascertain the psychometric properties of Grossarth-Maticek’s assessment tools, this being an important topic as so much of the latter’s conclusions are based on the claim of being able to psychometrically predict health outcomes.

Terada, Kawakami, Inaba, Takatsuka & Shimizu (2000) report the results of a cross sectional study conducted with all residents over the age of 35 in the Japanese city of
Takayama, Gifu prefecture (n=37,287). Specifically, they were attempting to clarify the relationship between the rationality/antiemotionality personality characteristic (R/A) as described by Grossarth-Maticzek with the past histories of seven chronic diseases: high blood pressure; heart attack or angina; stroke; diabetes; gastroduodenal ulcer; allergy; and stomach cancer. The R/A personality characteristic is defined in terms of ‘lack of hysteria’ and a conscious suppression of emotion in interpersonal communication, and has been linked by Grossarth-Maticzek and Eysenck to conditions such as cancer, CHD and stroke (Eysenck 1985). Instead of finding an association between this characteristic and disease profiles in their sample, Terada et al (2000) found that for males, this characteristic was linked to fewer incidences of stroke, diabetes and allergy; and for females it was linked with fewer incidences of stroke, allergy and stomach cancer. The authors conclude that their findings were contrary to those reported by Grossarth-Maticzek, and that cultural differences may play a role in this discrepancy. The role of cultural differences are crucial, as this may turn out to be one of the non-specific extraneous factors which could have influenced Grossarth-Maticzek’s outcomes- it points to a unique characteristic of his sample, perhaps based on cultural grounds, however subsequent studies conducted in Germany should be able to at least partly answer this possibility.

Amelang’s studies:
Utilizing a sample of 5133 subjects drawn also from the German city of Heidelberg, Amelang (1997) presented the cross sectional analysis findings- this constitutes the first stage in the larger 10 year prospective study with 5000 subjects. As part of the study into the psychometric properties of the constructs used, Amelang (1997) also obtained data from people who had experienced cancer or CHD and those who had experienced neither. In two separate studies with these populations, he obtained data from cancer patients (study 1 n=104, study 2 n= 50) and from CHD patients (study 1 n= 53, study 2 n= 60) and an orthopedic group as a control (n= 54). He developed an 140 item personality assessment questionnaire, referred to as the R(Revised)-Scales as a composite tool from the pool of 277 items published by Grossarth-Maticzek. This was considered important as over all of Grossarth-Maticzek’s publications, there have been a wide variety of items.
referred to as the items by which he has sorted probands into their typology, ie. Types 1-6. On the basis of some previous factor analysis (Amelang & Schmidt-Rathjens 1992), these researchers had eliminated items from the larger Grossarth-Maticek pool that displayed an insufficient item-scale correlation, as well as redundant items. The remaining 140 items were found to be internally consistent and highly intercorrelated with each of the original Grossarth-Maticek scales, all of them having correlations of greater than 90% (Amelang 1997).

Amelang et al (1996) report two main findings from their initial cross sectional analysis on an earlier subsample of 1858 subjects:-

i) the measures of personality type used by Grossarth-Maticek were not psychometrically distinct from other personality tests.

ii) personality scores of the disease group significantly differed from those of healthy subjects, but not in the direction predicted by Grossarth-Maticek’s publications.

These findings were later confirmed by Amelang’s (1997) analysis of the full sample, constituting 5133 subjects.

There was found to be a significant overlap between the Neuroticism and Depression scores of the subjects and the R-Scale 1 and 2 scores, to the extent that “it is probable that nothing additional is measured with Type 1 and Type 2 that is not already covered by the construct of Neuroticism” (Amelang et al 1996; p 202). Types 1 & 2 also loaded on one factor with Anger In and Jealousy, along with Neuroticism and Depression. As with their earlier research Amelang et al (1992; 1996) found that rather than Types 1 and 2 (measured by the items in the R-Scale 1 and R-Scale 2) being distinct categories of unhealthy risk factors, both R-Scale 1 and 2 load on the one factor, on which Type 4 (healthy, measured by R-Scale 4 items) loaded highly negatively. They referred to this loading of Type 1 and Type 2 onto the one factor as Factor 1, which was highly correlated with Depression, Anger In, Jealousy and Neuroticism. The opposite pole of Factor 1 (Type 4) was defined by high positive correlations with Optimism, Social Support and Sense of Coherence. They found that R-Scale 1 and R-Scale 2 items showed a positive correlation of .81, concluding that Types 1 and 2 do not constitute different
factors at all. This implies that the type of assessment items utilized by Grossarth-Maticek do not actually discriminate between proneness for cancer and proneness for CHD- in Amelang et al’s (1996) study, the R-Scale 1 and 2 items appear to assess the same construct.

In regards to the expected pattern of assessments of subjects with cancer and CHD, Amelang et al (1996) found very limited support for Grossarth-Maticek’s proposed relationship between health and personality. Contrary to the theory, they found that subjects with CHD did score higher than those without CHD on the Type 2 measures, but they also scored higher on Type 1, 3, 5 and 6 measures. Subjects with cancer did score higher than those without cancer on Type 1 measures, but also on Type 2 measures. According to the conclusions offered by Grossarth-Maticek and Eysenck, subjects with cancer should have scored high on Type 5 measures, but when compared to subjects with CHD, they scored significantly lower. Overall, Amelang et al (1996) concluded that the R-Scales failed to discriminate between subjects with cancer and CHD, but they did partially differentiate between healthy and unhealthy subjects. Those subjects with health problems (either cancer or CHD) were found to relate to both Neuroticism (and with Type 1 and Type 2 items), as well as with ‘Social Deviance’ (Type 3 and Type 6 items), but neither dimension was able to significantly discriminate between those subjects with cancer and those with CHD.

Using logistic regression analysis, Amelang (1997) reports that risk factors such as gender, passive smoking and neuroticism led to significant discrimination between subjects with cancer or CHD and those without, while Grossarth-Maticek’s scales accounted for little of the health/illness variance.

“No significant contribution of the two R-Scales to the discrimination between cancer and healthy groups was found. Consistent with the Grossarth-Maticek hypotheses, there was a significant contribution of R-Scale 2 to the discrimination of the CHD and the healthy groups.” (Amelang 1997:p 337).
This positive result is less impressive in light of the fact that the same result was obtained when R-Scale 1 was substituted for the R-Scale 2. In addition, Grossarth-Maticek’s personality constructs added only little incremental validity over other health relevant personality factors such as neuroticism, which explained more of the health-illness variance than did the R-Scales. A limitation on this study design is that even if subject’s scores on the R-Scales had followed the predicted directions, then the fact of already being sick with either cancer or CHD could not be excluded as a confounding factor in terms of the R-Scales results. However, considering the very strong findings reported by Grossarth-Maticek & Eysenck (1990), one would nevertheless expect to find cancer patient scores falling primarily in the Type 1 profile and CHD patient’s scores falling primarily in the Type 2 profile- that this did not occur is a concern which requires explanation or further attention.

Obviously, these results pose quite a challenge to the ability of Grossarth-Maticek & Eysenck’s (1990) conclusions to stand up to independent replication. One criticism which Eysenck made of Amelang’s research (cited in Amelang 1993) is that the latter obtained his data from the administration of questionnaires as opposed to interviews with subjects. Amelang (1993) makes the point that nowhere in Grossarth-Maticek’s publications does he state the necessity of obtaining data via interviews rather than via questionnaires; and that obtaining data via interviews actually creates more possibility of interviewer bias polluting the results than does the administration of questionnaires (Amelang et al 1996). Amelang (1997) states that he knows of no other field of research where the use of questionnaires rather than interviews has taken the level of prediction from near perfect to near zero.

Further research on Amelang’s cross sectional sample, reported by Yousfi, Mathews, Amelang & Schmidt-Rathjens (2004) found that small but ‘theoretically meaningful’ correlations were found between some diseases and personality measures. These were Emotional Labiality; Type A Behaviour; Behavioural Control; Locus of Control over Diseases; and Psychoticism.
Cross sectional analysis does pose some of its own problems. Cozby (1981) makes the point that a cross sectional analysis design does not allow the researcher to eliminate the possibility that some factors other than those under study have created the outcome— as with prospective studies compared to experimental studies, correlations are being obtained and no causal relationship can be assumed. As such, it is possible that another factor has been involved in influencing Amelang’s outcomes, rather than the results being due to a lack of influence of specific personality variables as proposed by Grossarth-Maticek. However, Amelang (1997) does not present his cross sectional analysis as providing the definitive answer. Amelang, Hasselbach & Sturmer (2004) report the results of the 10 year prospective study. Of the 5,133 subjects (aged 28-74) who were sampled 10 years earlier, 257 had died and 82% of those still living participated in the follow up. One hundred and twenty of these reported incidences of CHD and 180 reported incidence of cancer. The incidence of CHD could be significantly predicted by ‘Type A Behaviour’, ‘Emotional Labiality’, ‘Behavioural Control’ and the ‘Rationality/Antiemotionality Scale’. After controlling for gender, smoking and age, their findings demonstrate that only the factor of ‘Emotional Labiality’ had any predictive power for CHD. None of the personality factors had any predictive power for cancer at all. Perhaps partly as a result of these finding, Amelang & Schmidt-Rathjens (2004) state that the significance of psycho-social factors to the causation of cancer and CHD has been decreasing over time, while the contribution of the standard medical risk factors has remained constant. They now state that no more than 1-2% of the health-disease variance can currently be explained by personality variables, and as such hypotheses about causal relationships between these conditions and psychological factors are of ‘little value’ (Amelang et al 2004. p.12). It is suggested that methodological procedures such as better diagnostic instruments, differences in sample composition, and increased strictness for the control of extraneous variables could account for the apparent decrease in significance of psychological predictors.

Amelang (1991:p 233) questions whether Grossarth-Maticek’s findings may be more the result of “non-typical but favourable conditions”. One gets the sense from reading his
articles that he would like to accuse Grossarth-Maticek of scientific fraud, however he does not publicly make this allegation.

Schmitz (1992), from two studies utilizing data provided by 192 subjects who were administered a range of psychometric tests, concluded that Type 1 and Type 2 respondents were similar in that subjects scoring high on these types also scored high on measures of neuroticism:- Type 1 and N score $r=0.43$; Type 2 and N score $r=0.68$ (both significant at the $p<0.001$ level). Both Type 1 & 2 were also negatively correlated with Extraversion ($r=-0.30$; $r=-0.25$, $p<0.01$ and $p<0.25$ respectively). The main difference between them were that Type 2 subjects displayed a significant correlation of $r=0.27$ ($p<0.01$) with Psychoticism. Type 3 subjects also correlated highly with Neuroticism, with $r=0.48$ ($p<0.001$), and correlated negatively with Extraversion, with $r=-0.22$ ($p<0.05$). These results would also suggest less of an ability to distinguish between cancer and CHD prone personalities than do Grossarth-Maticek’s reports.

The anomaly of Neuroticism and ill-health:
There are several theoretical difficulties and inconsistencies which arise in relation to the construct of neuroticism and cancer proneness. An anomaly between Eysenck’s statements regarding neuroticism and Seligman’s construct of pessimism is that according to Eysenck’s (1965) model of personality, pessimism is a feature primarily of Introverts, especially of the more neurotic kind. Extraverts and emotionally stable people (the exact opposite to introverted neurotics), however, are said by Kissen & Eysenck (1962) to be more cancer prone, while introverted neurotics (for whom pessimism is a feature) were found to be uncorrelated to cancer. This is in contradiction to Seligman’s (1991) reports that pessimism (which according to Eysenck (1965) is a feature of neuroticism) is related to poor immune functioning and consequently to cancer, and to Eysenck’s (1987) later assertions that learned helplessness-depression is linked to cancer.

In the 1980’s Eysenck (1985) stated that there is a negative relationship between anxiety/Neuroticism (higher in women) on the one hand, and cancer on the other. However, Psychoticism (higher in men) also appears to be negatively correlated with
cancer. Eysenck (1985) suggests that scorers high in Neuroticism or Psychoticsm are more likely to have an emotional outlet for their distress through emotional expression. He also asserts that as they are more likely also to experience elevated ongoing chronic stress, they appear to be ‘inoculated’ against cancer, arguing that it is acute stress rather than chronic stress which is more likely to lead to cancer.

While Eysenck suggests a negative relationship between cancer and neuroticism, Schmitz (1992) states that there is an affinity between both Type 1 and 2 behaviour, and neuroticism. Schmitz’s (1992) research with 192 subjects found a strong correlation between Type 1 scores and neuroticism. On reading the profiles of both Type 1 and Type 2 patterns, it is difficult to avoid the conclusion that they are in fact quite neurotic. In an apparent turn around in the 1990’s Grossarth-Maticek & Eysenck (1991,p3) refer to the cancer prone personality as the ‘cognitive aspects of the neurotic disorder’, and the whole tone of their description equates with a description of a neurotic pattern- they make repeated references to the Type 1 and Type 2 behaviour patterns as being neurotic. Over the course of several decades, Eysenck appeared to utilize the construct of neuroticism in contradictory fashions in order to suit his particular argument at the time. This confusion is somewhat characteristic of much of the research with has been reported in regards to personality features and conditions such as cancer and CHD.

An additional problem with attempting to use the construct of neuroticism in this field is that neuroticism is such a multi dimensional construct, that to talk in terms of it as though it has one meaning is misleading. The various aspects of neuroticism, rather than neuroticism as a whole, may be expected by some health psychology researchers to have different effects on health. For example, people experiencing a lot of neurotic interpersonal stress may not be in a high risk group for cancer as a result of freely giving vent to their neurotic feelings. On the other hand, another person who experiences similar interpersonal stress, but for equally neurotic reasons does not give vent to his/her feelings, may be expected to have their immune system and health more compromised. Different forms of neuroticism can be implicated in both behaviour patterns, so it appears to not be neuroticism per se which is the crucial factor in terms of cancer prediction.
Constructs such as neuroticism may be of little relevance or value, other than to say that both cancer and CHD prone patterns appear to be quite neurotic (Schmitz 1992; Grossarth-Matichek & Eysenck 1991), although Amelang (1996) reports that Grossarth-Matichek’s constructs offer no further information than does the construct of neuroticism in explaining results.

Comer (1995) states that people high in neuroticism do show poorer health outcomes when compared to the emotionally stable, however this may have less to do with the dynamics of the personality characteristic. It may have more to do with the fact that highly neurotic people are more often socially isolated and lonely as a result of the associated interpersonal difficulties compared to emotionally stable people. Syme (1998) states that social support acts as a buffer against stress, and can assist people in developing a sense of control over adverse life circumstances which is health enhancing. Social isolation has been implicated as a major predictor of cancer in the longitudinal study initiated by C.B Thomas (cited in Eysenck 1984), and as a major risk factor for CHD as reported by Bunker, Colquhoun, Esler, Hickie, Hunt, Jelnick, Oldenburg, Peach, Ruth, Tenant & Tonkin (2003).

The role of repression:
While not replicating a prospective study design, and thereby not directly challenging Grossarth-Matichek’s conclusions, Kreitler, Chaitchik & Kreitler (1993) conducted research with relevant implications for the entire psycho-oncology field. They researched the effects of knowledge of a cancer diagnosis on levels of ‘repression’- this construct was considered important as much of the earlier research has concluded that emotional repression is one of the key features of the cancer prone personality- Grossarth-Matichek also has repression as an important feature of the Type 1 profile. Kreitler et al (1993) cite several publications arguing for the importance of repression in the causation of cancer, ranging from studies conducted in the 1950’s through to the 1990’s. It has been a central and re-occurring theme in this arena of study, with many researchers concluding that a poor ability to express especially negative emotions like anger, hostility and depression is highly correlated with the incidence of cancer.
There have been a range of problems in studies investigating repression, not the least of which has been an inadequate clarity of what the term actually means - is it the same as suppression, or denial? Kreitler et al (1993) report that there have been recent advances in the operationalization and measurement of repression, however few if any of the earlier studies were measuring repression with reliable or valid tools; they suggest that their studies are amongst the first to utilize a measure of repression which has adequate reliability and validity properties.

For their study, Kreitler et al (1993) measured repression using the Manifest Anxiety Scale and a social desirability scale assessing defensiveness. They suggested that repression implies “that defensiveness has been applied successfully in order to keep anxiety down to a reasonable or tolerable level” (Kreitler et al 1993 p.51). As such, they conceptualize repression as being a defense mechanism utilized for the purpose of managing anxiety. In their study, high repressors were subjects who scored low on anxiety and high on defensiveness. All of the other possible combinations, eg. high on both anxiety and defensiveness, low on both, etc, were considered as low repressors.

They conducted two testing occasions after mammograms had shown a need for exploratory surgery and/or an excision. The first testing occasion was conducted 2-3 days before the surgery was to occur; the second testing occasion was conducted 1-2 days after surgery which is also the time in which the subjects learnt of their diagnosis. At the time of the first testing, no women knew if they had cancer or not- the exploratory surgery would answer that. After the surgery, they divided the sample into those women who were found to have breast cancer and those who were found to not have breast cancer. They also used a control group of women who were undergoing surgery for non cancer reasons. After the surgery, they again re-tested all the women on the measures. There were 40 women in the group whose biopsies showed they were healthy, i.e free from breast cancer; there were 32 women in the group whose biopsies showed that they had breast cancer; and there were 26 women who were matched for various psychological factors who underwent surgery that was unrelated to cancer.
The main hypothesis of the study was that women would utilize repression as a coping strategy to help them in dealing with the anxiety aroused as a result of a cancer diagnosis—those with a cancer diagnosis were expected to demonstrate higher levels of repression than those without such a diagnosis. Kreitler et al (1993) found that before the surgery, there were no differences between the women, i.e. all three groups were roughly equal on levels of repressiveness, anxiety and defensiveness. After the surgery (which was the diagnostic test), when the women knew their diagnosis, they re-tested the subjects and found that those with cancer had scored highest on repressiveness. The differences were modestly significant (p<0.05). As such, they concluded that rather than repressiveness being a cancer prone personality characteristic, it was a state dependent coping strategy utilized by cancer patients to help them minimize anxiety. This finding is relevant in light of the other researchers who have found cancer patients to be high in emotional repressiveness and concluded therefore that repression must be a personality characteristic making them vulnerable to cancer.

Kreitler et al (1993) suggest that the degree of repressiveness may still have some implications for the progress of the disease, i.e. this psychological factor may not be important pre-disease, but may be important post diagnosis in terms of how the disease unfolds, survival times, etc. This is yet to be studied. There are problems with this study: the levels of significance obtained were only moderate, never better than the 0.05 level; the results were obtained very soon after the surgery and diagnosis, so they may not reflect the reality for cancer patients after the effects of trauma have lessened; the researchers relied on group means to establish who were repressors and who were not. Despite these limitations, Kreitler et al's (1993) findings join Amelang's (1991) in throwing some doubt onto the main tenants of the proffered psychological contribution to cancer.

Holland (in Goleman & Gurin 1993), in agreement with Kreitler et al's (1993) conclusions detailed above, states that denial is one of the standard reactions to learning of a cancer diagnosis- as well as the response of denial, the feelings elicited are stated as
being restlessness, anxiety and at times hopelessness accompanied by poor sleep and concentration, repetitive thoughts about the diagnosis, which she refers to as symptomatic of depression (Holland 1996). These feelings generally last for around a week or two, with most patients returning to a more normal psychological condition when treatment begins, usually within a few weeks of the diagnosis. Holland (1996) states that the degree of ongoing distress caused by a cancer diagnosis is dependent upon the patient’s ordinary psychological state prior to the diagnosis—those with pre-existing psychological problems are more likely to experience acute feelings of anxiety and depression which are more likely to last longer than those without such problems. She cites a recent study conducted which found that,

“47% of people diagnosed with cancer had a level of distress equivalent to that seen in a true psychiatric disorder. By far, the most common problems were anxiety, depression or a combination of the two” (Holland 1996. p 159).

In addition to the above mentioned findings of Kreitler et al (1993), there have been several studies that have found negative results in terms of the association between cancer and emotional repression. Greer & Morris (1978) found that rather than the repression of emotional expression as being a contributor to cancer, it was either very little or very much expression of anger which was related to cancer. In their study, 160 women who were admitted for breast tumor biopsy were interviewed and assessed on the Hamilton Rating Scale for Depression, the Eysenck Personality Inventory, and the Hostility-Direction of Hostility Questionnaire. In the course of the operation, 69 Ss were diagnosed as having breast cancer and 91 were diagnosed as having benign breast disease, with this group serving as controls. They found a significant correlation between the diagnosis of breast cancer and a persistent behavioral pattern of abnormal release of anger. This abnormality was in most cases extreme suppression, however there were subjects whose abnormality consisted of an over expression of anger.

In a prospective 5-year study of 69 female patients with early breast cancer Greer, Morris and Pettingale (1979) found that a post diagnosis attitude of a fighting spirit or the use of
denial in women with breast cancer was associated with an increased chance of being alive 5-10 years later compared to women who displayed stoic acceptance or helplessness-hopelessness. Patients’ psychological responses to the diagnosis of cancer were assessed 3 months after the diagnostic operation- these responses were later related to outcomes 5 years after the operation, with recurrence-free survival being significantly common among patients who had initially reacted to cancer by denial or who had a fighting spirit.

This finding confirms those of Suls and Fletcher (1985) who conducted a meta analytic review of the research literature on coping strategies. They concluded that strategies involving avoidant tactics (such as denial) are effective in reducing pain, stress, and anxiety in some cases, whereas non-avoidant (attention) strategies appear to be more effective in others. Results of an overall analysis of studies providing tests of attention versus avoidance indicated little evidence for one strategy’s superiority over the other.

Denial may have some psychological and physical health benefits under certain circumstances- avoidance was associated with more positive adaptation in the short-run.

“If attention involved an emotional interpretational set or no explicit set, then it was associated with more negative outcomes than avoidance...However, attention was superior to avoidance if the former involved a focus on sensory schemata rather than emotional processing.” (Suls & Fletcher 1985 p.249).

They also found a temporal factor, as avoidance indicates better outcomes initially, but in the long run attention was associated with more positive outcomes. One could conclude from these studies that the role of repression or denial of strong negative emotions in the causation of cancer is at least unclear.

In summary, there are sufficient empirical and theoretical challenges to Grossarth-Maticek’s claims to cast their veracity into some doubt. As with other scientific enterprises, support for a claim can only be considered in the context of independent replication studies. The results of Amelang’s replication study (Amelang et al 2004) does
cast serious doubt on Grossarth-Maticek’s claims, however these do not provide an absolute end to all speculation about his results. As Amelang et al (2003) speculate, differences in methodology and sample composition may explain some of the apparent decrease in contribution of psychological factors to these conditions. In addition, Eysenck (cited in Amelang 1993) does cast doubt on Amelang’s data collection method, suggesting that the latter’s approach was unlikely to obtain the results which Grossarth-Maticek reported.

Is it possible that differences in sample composition could create such radically different results between Grossarth-Maticek and Amelang? Are Grossarth-Maticek’s claims based purely on scientific fraud? Or are there methodological issues, related to data collection techniques, as suggested by Eysenck (in Amelang 1993) which may throw some light onto the question of how Grossarth-Maticek obtained such startling results? The answers to these questions may hold several important implications for health practice and psychological research with human subjects. The goal of the current research enterprise is to cast light onto these compelling questions, thereby further elucidating the role that psychology may play in the provision of health care and the prevention of chronic disease. If there is a large psychological contribution to chronic disease, as well as the potential for prevention, then it behooves psychology to clarify this role and articulate its contribution. While Grossarth-Maticek and Eysenck (1990) suggest a relatively straightforward role for psychology, it is possible that their research indicates the importance of psychosocial experience in the causation of cancer and CHD, but not in the manner which they have suggested. In fact, their research may provide evidence of a crucial psychosocial variable which was an unintended causal factor in Grossarth-Maticek’s studies. This possible factor is heretofore unexamined and will be postulated in the following chapter.
Chapter 9

Pointing the Bone in a Scientific Society:

“an error can be the unrecognised bearer of truth”
Umberto Eco, *Foucault's Pendulum*, 1997, p.337

Did Grossarth-Maticek aid belief to become biology?
An interesting phenomenon of traditional Australian Aboriginal culture pertains to the practice of pointing the bone. This was a form of condemning to death the recipient of the bone pointing by a suitably qualified ‘magic man’. Folklore, anecdotal and eyewitness accounts testify to the effects of such a practice, usually being the rapid death of the recipient who was otherwise healthy and not expected to die (Lockwood 1962). This outcome is only possible within a cultural context, wherein the predominant view is a subscription to certain magical beliefs. The possibility of a similar phenomenon in our ‘scientific culture’ will be explored in this chapter. Within a scientific culture, predictions of health or illness that are presented from a scientific paradigm may demonstrate the same power of belief over biology.

It is contended here that the remarkable results published by Grossarth-Maticek and Eysenck (1991) may not be the result of either accurate predictions as espoused by Grossarth-Maticek, or scientific fraud which is alluded to by some commentators. A third explanation is possible in which the following factors are suggested to have combined, producing the remarkable results claimed by Grossarth-Maticek & Eysenck (1991)- these phenomena are:-

- Experiment effect
- Interview bias
- Self fulfilling prophecy
- The relationship between stressful life events, learned helplessness depression and excessive releases of stress hormones such as cortisol.
- The relationship between excessive cortisol levels and cancer/CHD.
In Grossarth-Maticke’s interviews, the full interview condition required that interviewers provide all information required by the proband and sought to answer all questions posed. It is possible that Grossarth-Maticke’s interviewers were actually asked by the probands for their risk status, and were compelled by the interview conditions to reveal this impression. Grossarth-Maticke failed to respond to the current author’s attempts to obtain an answer to the question of this possibility- as such, it must remain conjecture. Another possibility is that the impression of risk status was inadvertently conveyed to the probands by the interviewers via an interviewer bias- these possibilities will be further explored in this chapter.

None of Grossarth-Maticke’s critics appear to have made the leap from viewing his data collection method as inadequate, to viewing the interview/experiment itself as being a health damaging life event. Personal communication with Fox (1999) indicated that although he raised the query of non standardized data collection methods, he had not considered the treatment effect possibility when he contributed to the Psychological Inquiry special edition (Fox 1991). However, one only needs to consider the amount of anxiety that exists in our culture concerning life threatening illnesses such as cancer and CHD for this notion to appear plausible.

In more detail, the proposal to be investigated in this study is that Grossarth-Maticke’s interviewers conveyed to the study subjects their supposed pre-disposition to cancer, CHD, or to positive health, thereby adding to the long term possibility of these outcomes via a treatment effect. People who were initially cancer prone according to psychological characteristics in only a modest fashion may have become much more susceptible as a result of the effect of learning of their elevated risk status, ie. a modest psychological factor may have been elevated to a major psychological factor, thereby damaging the functioning of their immune systems. As such, this effect is proposed to be similar to the effect of pointing the bone in traditional Australian aboriginal cultures- a hex or curse has been placed on the recipient, producing deleterious biological changes and an untimely death.
Learning of belonging to a risk group for cancer or CHD may have lead some of Grossarth-Maticek’s subjects to an alarm reaction or a psychological crisis with associated surges of stress hormones including cortisol- the anxiety may have been maintained over time for a particular sub-set of subjects, leading to ongoing elevated levels of cortisol production, with consequent effects on the immune system. It is also possible that a ‘sleeper effect’ was initiated in the interviews. Lang and Lang (in Rosenberg & Turner 1981) describe the sleeper effect as being when the amount of change which occurs immediately after the exposure to the stimuli is smaller than the change noted some time later. Alexander & Wiley (in Rosenberg & Turner 1981 p. 286) make the point that,

“The immediate effects of an event do not exhaust its influence…. Subsequent events in a sequence are structured, in part, by the events that precede them. The implications of an event that falls early in the sequence may not be evident until later response possibilities emerge. Everyday life is no stranger to events that seemed inconsequential at the time of their occurrence, only to be regarded as crucial later. These effects are no less real for being unrecognized at the actual moment the original event took place.”

As such, if there was not an initial alarm response at the point of perceiving an at-risk status, it is also possible that Grossarth-Maticek’s designated at-risk subjects may have experienced a sleeper effect with the psychological, emotional and physiological consequences of this news increasing over time rather than decreasing. As a result of the stress related hormones excreted over the course of years, Grossarth-Maticek’s subjects may have become more susceptible to cancer and CHD than they otherwise would have been. This would be in effect a self fulfilling prophecy on a large scale- a prediction that was at the outset a false definition of the situation evoking behaviour, but was however a widely shared belief in vulnerability taken to be true by the interviewers and subsequently by many of the subjects. This prediction may have been fulfilled, not because it was true from the beginning, but because enough vulnerable people took it to
be true, thereby becoming anxious enough to compromise the functioning of their immune systems and producing the outcome that otherwise may not have occurred.

This possibility would explain the extraordinary findings from Grossarth-Maticzek's studies, and the observed relationship between the beliefs of the interviewers and the health outcomes. Those interviewers who did not believe in his theory, according to Grossarth-Maticzek’s view, failed to obtain a high level of prediction of illness. What may have happened is that those interviewers:-

- failed to convey to the subjects a belief that they were susceptible to cancer/CHD, and thereby
- failed to create a perceived health crisis and heightened cancer/CHD anxiety condition in their subjects;
- failed to create a stressful life event which would add to excessive secretions of cortisol, and thereby
- failed to assist in the compromise of the subject’s immune systems;
- failed to add to ongoing rumination of pessimistic cognitions about the future, and thereby
- failed to convert a modest psychological factor into a major risk factor.

This may have been then recorded as ‘poor validity’, i.e their assessments failed to correlate with the incidence of ill health. The rest of this chapter will make the case in detail for each of the aspects required of this proposal.

Methodological issues:
The question of a possible role of experimenter bias in explaining the disparate findings of Grossarth-Maticzek (1980) and Amelang (1997) is raised by several authors such as Schuller & Fox (1991), but appears to not have been thoroughly explored, and not tested in an empirical manner. Fox (1999) stated in personal communication to the author that he had not considered the possibility of a large treatment effect when suggesting the possibility of experimenter bias (Schuller & Fox 1991). A range of other possibilities are offered to explain how Grossarth-Maticzek arrived at such compelling results. To many of
the contributors of the 1991 special edition of Psychological Inquiry dedicated to this topic, Grossarth-Maticek’s reports are simply “too good to be true”. There are allegations of data tampering, however this possibility is examined by an independent statistician Heller, with the conclusion being that there is no evidence of this occurring (Eysenck 1991). The only errors in data entry or statistical analysis that were found, when corrected, actually strengthened Grossarth-Maticek’s claims. Many of the critiques subtly imply that the authors would like to accuse Grossarth-Maticek of scientific fraud, however they seem to stop just short of this, perhaps for legal reasons. Amelang (1991:p 233) questions whether Grossarth-Maticek's findings may be more the result of “non-typical but favourable conditions”, with little clue as to what these conditions may be. In private email communication to the author, referring to Grossarth-Maticek's research, Amelang (1998) states,

“Without any doubt there is a great deal of potential creativity and subjectivity in that stuff, but I wonder if there are- to bring it directly to the critical issue- real persons to the data filed in the Grossarth-Maticek archives. Several independent people have raised this question, and I for myself will resign to the statement that the Department of Psychology would, must have taken notice of a research project addressing to nearly the whole Heidelberg population of people in the age between 40 and 60. On the other side, we know that it cannot prove that these studies really have not been done and that the decision must be done on the basis of independent new research”.

While no researcher’s reputation places them above the possibility of incorrect analysis or interpretation of data, the inclusion of Eysenck into Grossarth-Maticek’s research enterprise added a greater degree of credibility to the claimed results. Before entering the international psychology literature in the early 1980’s, Grossarth-Maticek was largely unknown, however Eysenck was perhaps the most prominent of British psychologists. He is stated as being the third most cited intellectual in history, following only Marx and Freud, and as being the most cited psychologist in history (‘The Australian’ Newspaper Monday 8th September, 1997). Eysenck (1991b) describes his involvement with
Grossarth-Maticke as occurring well after the data collection phase, meeting him in the early 1980’s and offering to help with his data analysis. He was somewhat critical of many aspects of Grossarth-Maticke’s research, referring to several methodological problems, however Eysenck (1991b) considered that overall, despite these problems he was able to help Grossarth-Maticke salvage some important findings from the masses of data. Grossarth-Maticke’s basic theory of psychosocial factors in the causation of major ill health corresponded neatly with Eysenck’s own views; with Grossarth-Maticke reporting findings of large scale long term prospective studies, the opportunity arose for Eysenck to evaluate his own hypotheses with a valuable and unrivaled data base.

The novelty and importance of Grossarth-Maticke’s reports is not that he claims to have found psychosocial correlates with cancer and CHD- the Health Psychology literature testifies to empirical findings of a modest role for psychosocial factors, as detailed in earlier chapters. It is only Grossarth-Maticke, in collaboration with Eysenck that has claimed an extremely powerful causal relationship between such factors and ill health. Their reports of an 81% accuracy of prediction of incidence and death by cancer and CHD on the basis of psychosocial factors is by and far the largest claim yet made in the Health Psychology arena. Amelang et al (1996) make the point that where the relative risk rate for a cigarette smoker to die of cancer or CHD is 2.0, the relative risk rates presented by Grossarth-Maticke for Type 1’s to die of cancer is 50, and the relative risk rate for a Type 2 to die of CHD is 30 (ie. the Type 1 person has a relative risk of dying of cancer at a 50 times higher rate than a non Type 1 person).

The current issue in question is not to dispute the modest role which psychosocial factors may play in ill health, but to question how Grossarth-Maticke arrived at such extraordinarily large findings, i.e to explain the gap between the well demonstrated modest role and the extraordinary role of psychosocial factors as reported by Grossarth-Maticke. To rely on insinuations of scientific fraud is not only uncharitable to both Grossarth-Maticke and Eysenck, but as importantly is not empirically based. As in any allegation of misconduct, the onus is on the accuser to provide evidence of fraudulent activity- none has been forthcoming, although some methodological and procedural
problems have been articulated and by and large acknowledged by Eysenck (1991b) and Grossarth-Maticek. While Grossarth-Maticek is a mostly unknown quantity, Eysenck’s honesty has never been questioned before. Although at various times he attracted controversy, there is no evidence of prior misconduct. In fact, the evidence is more to the contrary, that he has been willing to publish controversial and unpopular findings in relation to race, gender and intelligence at an enormous personal cost to himself and his family (Eysenck 1991a). A less honest researcher may have been more inclined to ensure his own and his family’s safety and comfort, and not articulate such reports.

The Clue:
Referring to a 1985 article co-authored by Grossarth-Maticek and written in German (not available in English), Schuller & Fox (1991 p.258) state the following.

“In their section on "Interview Methodology" (pp 15-16), the process of inquiry does not fulfill the necessary requirement in epidemiologic studies of maximum standardisation and consequent replicability and generalizability to other cohorts. As the authors explicitly acknowledge, the results depended on the interviewer and not on the interview instrument. The authors give their purpose in doing the interview experiment:

“By means of the interview experiment we wanted to answer the question whether, for successful prediction of specific organic diseases, defined system conditions are necessary in the process of data accumulation, that is whether the kind of prediction depends on the kind of data accession. It appears that interviewers who did not fulfill the system conditions did not arrive at successful prediction...”(p16)
One notes with incredulity that the first system requirement was that the interviewers believed in a relationship between psychosocial factors and the development of cancer". (p.15, italics added)

**Experimenter Bias:**

Most psychology texts list experimenter bias as a source of error in experimental research- it has been clearly demonstrated to operate in a wide range of research situations. Cozby (1981p.169) states that

“No matter how many precautions are taken to keep extraneous variables constant, it is still possible that the experimenter may somehow treat subjects in each condition differently. This problem is called experimenter bias or experimenter expectancy effect (Rosenthal, 1966, 1969). Experimenter bias refers to any intentional or unintentional influence that the experimenter exerts on the subject in an attempt to confirm the hypothesis”.

Unintentional experimenter bias poses a problem to the validity of research. Experimenters may emphasize certain words or terms when reading instructions to subjects, with changes in tone of voice or volume; they may also subtly influence a subject by smiling or showing minimal signs of relief when the subject responds in a way confirming the experimenter’s hypothesis (Cozby 1981). It is unlikely that an experimenter who is entertaining particular hypotheses will completely fail to subtly communicate these to the subjects.

The classic studies in which experimenter bias was well demonstrated were conducted by Rosenthal (1994) in the nineteen sixties. Experimenter bias was later to be referred to as the ‘Pygmalion effect’, or the ‘Rosenthal effect’ referring to his finding that he could produce an increase in school children’s IQ score simply by telling the teacher that s/he will intellectually bloom over the next year. (Walker, Burnham & Borland 1994). This information led the teachers to think about and treat the ‘intellectual bloomers’ differently to how they were treating the non bloomers, or to how they were initially treating the
children. The crucial factor in creating this outcome was the expectation on behalf of the teacher (created falsely by the Rosenthal) that certain students were ‘bloomers’ as such, the teachers began to subtly treat these students differently, which conveyed to these students that a certain expectation had been created, leading to a certain expected outcome. Cozby (1981) makes the point that rats and horses have been found to respond to subtle environmental cues in the form of experimenter bias, so it seems reasonable that humans will also be able to perceive subtle cues. Rosenthal (1976) conducted a meta-analysis of over 300 studies which clearly demonstrated the power of one’s expectations to influence another person’s behaviour in the direction of the expectation.

The ‘Rosenthal effect’ is an example of a self fulfilling prophecy, a term first articulated by sociologist Robert Merton in the late 1940’s. Merton (cited in Sztompka 1986 p. 234) states:-

“the self fulfilling prophecy is, in the beginning, a false definition of the situation evoking behaviour which makes the originally false conception come true....The initially fallacious expectation makes for a seemingly confirmed outcome.... In that pattern, an initially false but widely shared prediction, expectation, or belief is fulfilled in practice not because it was at the outset true, but because enough people took it to be true and, by acting accordingly, produced the outcome that would otherwise not have occurred”.

Demand characteristics are another important source of error in research, related to experimenter bias (Cozby 1981). The entire experimental context can be viewed as containing a range of environmental cues that help the subject solve the problem of how to behave in that context. The types of questions or demands placed on the subject will give them clues as to the hypothesis being entertained by the experimenter. The experimental context can be viewed as an unfamiliar one to most people, creating a need to know what it is all about. Questions such as ‘what is being tested here? Why? What does the experimenter think of me, or how do they evaluate my answers?’ all represent an urge which humans have to make sense of their experience. Both experimenter bias and
demand characteristics may combine to inform the subject about the true nature of the research, and of the experimenter’s hypothesis. These can lead to changes in the subject’s cognitions such as interpretations, self evaluations, expectations, and actions, as well as consequent emotions.

Interviewer bias operates in a similar fashion to experimenter bias, however refers to interview situations where data is being obtained from subjects in the form of a face-to-face interaction. As with experimenter bias, the possibility is raised that the interviewer can, in a non deliberate manner communicate to the interviewee a range of information such as:– the study hypothesis, and the interviewer’s hypothesis of that particular subject. These subtle communications can affect the outcome of the interview. Again, subtle and inadvertent non verbal communications may be conveyed by the interviewer to the interviewee, such as smiles, silences, nods, signs of relief, eye contact or avoidance, tone and volume of voice, etc.

In conclusion, the role of experimenter bias is well established by psychological research. The possibility of it effecting the results of an experiment are tackled by a range of methodological strategies, such as conducting double blind studies where in the person having contact with the study subjects does not know of the hypothesis being tested or the group in which the subject is located until the data has been collected (Wade & Tavris 1998). Standardisation of data collection is also considered to be highly important in psychological research, with non standardised data collection methods being viewed as inferior. Referring to psychometric testing in research, Anastasi (1982. p.24) states:–

“Standardisation implies uniformity of procedure in administering and scoring the test. If the scores obtained by different persons are to be comparable, testing conditions must obviously be the same for all. Such a requirement is only a special application of the need for controlled conditions in all scientific observations.”
With a standard method for obtaining study data, there is less possibility that the experimenter or interviewer will inadvertently treat each of the subjects differently, according to his/her own hypothesis or view of the subject. As such, the chance of the experimenter communicating these cues to the subject is lessened.

Making sense of Grossarth-Maticke’s results:
It is clear from Schuller & Fox’s (1991) quotation from a German language article co-authored by Grossarth-Maticke in 1985 that the data collection method employed in the prospective studies was not of a standardised form. The use of students as trained interviewers, a requirement of whom was that they endorsed Grossarth-Maticke’s psychosocial hypothesis, introduces into the experimental situation a source of error, the effects of which need to be accounted for. In response to the possibility of fraudulent data collections, Eysenck (1991b) reports the investigation conducted by Dr W.D Heller of the Institute for Statistics of Karlsruhe. He states:-

“Dr Heller also interviewed a random sample of the students who had collected the data for Grossarth-Maticke, to look at methods used, reliability of testimony, and adequacy of training; he found nothing to complain about.”
(Eysenck 1991b p.299).

It is unclear from the literature presented how Dr Heller evaluated for the possibility of experimenter bias, if he did at all. Eysenck (1991b) appears to credit Schuller & Fox’s (1991) critique with more importance than he does the other 19 authors responding to his and Grossarth-Maticke’s reports in the 1991 special edition of Psychological Inquiry. For example, he states,

“Schuller and Fox represent the more scientific part of the group of critics of Grossarth- Maticke’s work who have remained unconvinced, and without doubt their concerns carry the most weight”. (Eysenck 1991b p.310)
Grossarth-Matichek (cited in Eysenck 1991b p.310) responds to the possibility of experimenter bias with the following statement:

“One typical misunderstanding relates to the interview experiments, which have not been extensively documented. This experiment had as its aim to research the conditions more closely which would enable proper prediction as well as meaningful intervention. We found that, as expected, prospective research results relying on filling in of questionnaires depended very much on the kind of relation established between interviewer and subject. Interviewers with a high degree of empathy, who took seriously individual differences in behaviour, as relevant to the origin of diseases, and who managed to choose a proper moment for the beginning of the questionnaire-related interview, after a friendly preliminary discussion, achieved a more reliable and valid relation between personality variables and mortality. Less empathic interviewers who denied a synergistic relation between organic and psychological factors, and only believed in physical causation of disease produced low retest-reliabilities and poor validities”. (italics added)

What is being stated here simply confirms the claims of Schuller & Fox (1991) referred to earlier. The interviewers who believed in Grossarth-Matichek’s psychosocial theory “achieved a more reliable and valid relation between personality variables and mortality”, i.e. there was a statistically significant relationship between death by cancer or CHD 10-15 years later, and the beliefs (and presumably consequent behaviours) of the interviewers. Those interviewers who are defined as denying a ‘synergistic relation between organic and psychological factors’ obtained only poor validities, which means that few of their predicted cases of cancer or CHD obtained these diseases. When there is a statistically significant relationship between the beliefs of an experimenter/interviewer and the health outcomes, surely the possible role of experimenter must be considered.

Suggestive of this assertion, Grossarth-Matichek, Eysenck & Barrett (1993) report that in one of their studies comparing the accuracy of predictions according to test
administration style, a poor accuracy of prediction (ie. fewer deaths and incidences of cancer and CHD) was obtained when the data was gathered by the usual method in psychometric research, ie. questionnaires with standardised instructions from the test administrator. The extraordinary results were only obtained when interviewers introduced a condition wherein

“trust and understanding had been increased by interviewers’ suitable participation; worst results were achieved for subjects when no special effort was made to increase either” (p. 943)

The ‘worst results’ referred to here are actually referring to the outcome of subjects not contracting cancer or CHD- those who had participated in an administration procedure in which there was minimal contact between the subject and the interviewer had outcomes which were not significantly related to cancer or CHD. In addition, those subjects who were tested by an interviewer who did not believe in the Grossarth-Maticek theory had outcomes which were not significantly related to these diseases. Does this not suggest a possible experimenter bias operating? Eysenck, Grossarth-Maticek & Barrett (1993) explain the relationship between beliefs of the experimenters and the health outcomes in terms of accuracy of predictions, however there is another viable alternative explanation which entails the role and subsequent effects of experimenter bias.

Was it possible for Grossarth-Maticek’s interviewers to not convey to the subjects whether they were in a disease prone group or a health prone group, either deliberately or inadvertently? As stated above, the psychological research community has long been aware of various sources of experimental error including experimenter and interviewer bias and demand characteristics of the study. Was it possible for Grossarhth-Maticek’s subjects to not know that they were participating in a study investigating a wide range of causes of cancer and CHD, including psychological ones? The subjects were being asked to respond to questions concerning all of the known physical risk factors, such as cigarette smoking, alcohol consumption, family members with cancer or CHD, diet, levels of exercise, etc. The general topic of the research, if not actually stated to them
would appear to be evident from the nature of these questions alone. What else could be
the topic of study? Also, it is stated by Grossarth-Maticzek (in Eysenck 1991b) that the
purpose of the interviews with the best predictive powers was to establish conditions of
trust and understanding by explaining to the subjects as much as they needed to know
about the research study.

In addition to this factor, the interviewers, at least those who were able to ‘predict’ cancer
and CHD, are known to have believed in the role of psychosocial factors in the causation
of these diseases. Considering the wide range of situations in which experimenter bias
has been demonstrated to operate (including rats running though mazes: Coszby 1982),
why would the interviews conducted by Grossarth-Maticzek’s students not be similarly
effected by experimenter bias, especially when one remembers that the level of belief was
considered important to obtaining positive results?

One may wonder how the interviewers could fail to convey to the subjects two things:-
i) the psychosocial theory of disease as it related to cancer and CHD
ii) the interviewer’s assessment of the respondent in terms of the Grossarth-Maticzek
typology.

In a cross sectional study comparing scores on the Grossarth-Maticzek items between
healthy people, those with cancer and those with CHD, Amelang (1997) failed to find
results supportive of Grossarth-Maticzek’s claims in that subjects with cancer or with
CHD did not significantly differ in their typologies. He did, however find a modest
relationship between Type 1 and Type 2 profiles with ill-health in general (not
specifically to cancer or CHD), but this is much the same as the modest relationship
reported by other researchers. It is already known that standard measures of neuroticism
(factorally related to Type 1 and Type 2) are modest predictors of ill-health in general-
high neurotics display two to three times as many somatic complaints compared to non
neurotics (Sarafino 1994). Amelang’s (1997) research also demonstrated a relationship
between Type1 & 2/neurotic scores and the incidence of cancer and CHD.
The hypothesis being suggested here would explain both Grossarth-Maticke’s findings and those of Amelang’s (both cross sectional and prospective), primarily because Amelang did not gather his data with interviews- he used standardised questionnaire administration, therefore there was no interviewer bias operating- the subjects did not experience the ‘benefits’ of the interviewer bias to shape their fears of the future.

Grossarth-Maticke, Eysenck & Barrett (1993 p.945), in describing their interpretation of the function of different methods of questionnaire administration to the reported health outcomes, detail the following in regards to methodology.

“The method of administration was always by a trained interviewer, who spent one hour explaining the purpose of the questionnaire administration, assured the subject of confidentiality, answered questions both general and specific, and tried to win the trust and confidence of the subject by showing interest and concern, being friendly and courteous, giving information, and being responsive to questions. Independent replications have usually simply administered the questionnaire without such preparation by interviewers to groups of well and ill subjects in an attempt to relate types to cause of death or diagnosed illness”.

The assessment tool which they used was described as the Short Interpersonal Reactions Inventory consisting, in the original German form of 71 questions (dropped to 70 items in the English version, giving 10 items for each of the 6 typologies and 10 items for a lie scale). Trained interviewers were used to collect the data.

“Our main interest here is the demonstration (in agreement with several large scale prospective studies using a four type scale but omitting Types 5 and 6) that Type 1 predicts cancer, Type 2 predicts coronary heart disease, and Type 4 the absence of both. We are less concerned with the other types which are largely irrelevant to cancer and coronary heart disease...”. (Grossarth-Maticke et al 1993 p.945).
As such, the items of interest are the 40 items related to the Type 1-4 constructs, as seen in the Short IRI. Selection for the study begun in 1975 was based on a ‘fairly random sample’ from population records, approaching 1958 males aged between 55-57 years old, all living in Heidelberg. The subjects “were invited to take part in a scientific study of psychosocial factors in health and illness” (Grossarth-Maticek et al 1993 p.946). A sample of 1721 accepted the offer to participate, in which all subjects were asked to complete the Short IRI, “but this administration was carried out differently using randomized assignment to four groups” (Grossarth-Maticek et al 1993 p.946).

“Group A- In this group (n=338) the questions were read aloud by the interviewer, and explanations were given after each question as to the precise meaning of the question following any queries by the subject. This group is designated the explanation group...

Group B- In this group (n=348) we tried to manipulate the variable of trust. Participants were invited to talk with the interviewer for 45- 60 minutes, discussing in the first part positive and negative events in their lives and their typical reactions to these situations. Following this part of the interview, participants were asked in the second part if they trusted the purpose of the questionnaire administration or if they still had some questions to ask. The interviewer did his best to answer such questions as were raised, and only began the administration of the questionnaire when the subject stated that the interviewer as well as the purpose and also design of the questionnaire administration were trusted.

Group C- For Group C (n=348), the explanatory method for Group A was combined with the trust evoking method for Group B so that, following the discussion devoted to gaining the trust of the subject, the interviewer would continue with the explanation of all the items in the questionnaire. This group enjoyed both the explanatory and the trust producing paradigms.
Group D- This group (n=687) constituted the control group, receiving neither the explanatory nor the trust producing interaction with the interviewer. Instead, they were given the questionnaire and asked to fill it in without prior discussion or explanation of the meaning of the questions.” (Grossarth-Maticzek et al 1993 p.946).

As can be deduced from the above descriptions, more opportunity for a treatment effect would be created in the conditions in which the subject was able to describe negative events in their lives and their reaction to those. This occurred in groups B and C. As was expected, the results demonstrated that,

“There were significant differences in the accuracy of the predictions, depending crucially on the method of administration of the questionnaires. Best predictions were achieved for subjects when both trust and understanding had been increased by interviewers’ suitable participation; worst results were achieved for subjects when no special effort was made to increase either. Intermediate results were found for procedures which increased either trust or understanding.” (Grossarth-Maticzek et al 1993 p.943)

In terms of raw numbers and percentages, the following results are given.

**Table 11. Personality Type and Mortality: Four groups given different administration of the Questionnaire** (Grossarth-Maticzek et al 1993 p.949).

<table>
<thead>
<tr>
<th>Type</th>
<th>Deaths</th>
<th>%</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cancer</td>
<td>N</td>
<td></td>
</tr>
<tr>
<td>Group A (explanation only)</td>
<td>6</td>
<td>40% of all Group A Subjects who died of cancer (15)</td>
</tr>
<tr>
<td>Group</td>
<td>Type 1</td>
<td>Type 2</td>
</tr>
<tr>
<td>------------</td>
<td>--------</td>
<td>--------</td>
</tr>
<tr>
<td>Group B (trust only)</td>
<td>7</td>
<td>4</td>
</tr>
<tr>
<td>Group C (trust and explanation)</td>
<td>12</td>
<td>3</td>
</tr>
<tr>
<td>Group D (neither trust nor explanation)</td>
<td>10</td>
<td>12</td>
</tr>
</tbody>
</table>

As can be seen by comparing the relative percentages of deaths by cancer between the different administration style groups, those subjects who received the ‘full treatment’ during the test administration, died at more than twice the rate of those who simply filled in the questionnaire, i.e. 30.36% compared to 70.59%. Furthermore, Type 2 subjects in
the Group D, who should have been more susceptible to CHD and less susceptible to cancer compared to the Type 1 subjects, actually died of cancer at a greater rate than the Type 1 subjects; while the deaths by CHD for this Group D were exactly the same for Type 1 and Type 2 subjects at 34.29%. This may suggest that because disease specific cues were not given in the course of the test administration, no real differences were noted in terms of deaths by either disease; whereas in the other conditions, disease specific cues may have been given, leading to the observed differences in deaths, i.e Type 1 subjects were given more cancer prone cues than Type 2 subjects- this could have interacted with their personality types to produce more deaths by cancer. It must be asked if disease specific cues may have been as overt as the interviewer’s responses to direct questions by the subjects? For example, did probands ask questions such as “Does this mean that I am going to get cancer?”, or “what does this tell you about me?”.

In a similar article by Grossarth-Maticke, Eysenck and Boyle (1995), the authors provide what appear to be an expanded set of results from the same study, however with a larger sample (n= 3563 men and women followed over 15 years). The test administration conditions are the same as described above, with the results as described below. The number in each group was not equivalent due to refusal rates being different with each of the interview conditions. Grossarth-Maticke, Eysenck and Boyle (1995) state that the refusal rate was highest for interview condition 4, with the other 3 conditions having much lower refusal rates.
Table 12. Proportion of S's in different groups dying of Cancer (extract from Grossarth-Maticek, Eysenck and Boyle (1995 p.707).

<table>
<thead>
<tr>
<th>Type</th>
<th>Group 1 Expl. &amp; trust (%)</th>
<th>Group 2 explanation (%)</th>
<th>Group 3 trust (%)</th>
<th>Group 4 quest. only (%)</th>
<th>$X^2$</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Type 1 (cancer)</td>
<td>25% (69/276)</td>
<td>14.7% (38/258)</td>
<td>11.6% (23/199)</td>
<td>7.4% (4/53)</td>
<td>31.36</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>Type 2 (CHD)</td>
<td>10.7%</td>
<td>8.7%</td>
<td>8.6%</td>
<td>3.14%</td>
<td>NS</td>
<td></td>
</tr>
<tr>
<td>Type 4 (health)</td>
<td>1.4%</td>
<td>3.0%</td>
<td>11.2%</td>
<td>10.6%</td>
<td>25.57</td>
<td>&lt;0.01</td>
</tr>
</tbody>
</table>

Again, as with Table 11, it is clear from these figures that those subjects who ‘enjoyed’ the full treatment died from cancer at much higher rates compared to those who self administered the questionnaire. The full raw figures are available in the articles.

A Chi squared test of the comparative death frequencies of subjects by membership to groups 1 or 4, and typology as per Grossath-Maticek’s assessments suggest a very significant interaction effect between typology and interview condition with deaths. The following table shows the cells used in this analysis.

Table 13. Deaths by cancer according to membership to interview condition groups.

<table>
<thead>
<tr>
<th>Interview condition</th>
<th>Full Interview condition Observed deaths/expected</th>
<th>Self Administered Quest. Observed deaths/expected</th>
</tr>
</thead>
<tbody>
<tr>
<td>Type 1</td>
<td>69/49.12</td>
<td>4/23.88</td>
</tr>
<tr>
<td>Type 4</td>
<td>2/22.88</td>
<td>31/11.21</td>
</tr>
</tbody>
</table>

This analysis demonstrates an interaction effect between interview condition and typology (vulnerability) with deaths, with a significance level of 5%.
The corresponding table for deaths by CHD is presented below, with the same trend being apparent, i.e. those who had the full treatment in the interviews died of CHD at much higher rates than those in group 4.

Table 14. Deaths by CHD according to membership to interview groups (extract from Grossarh-Maticek, Eysenck and Boyle (1995 p.707).

<table>
<thead>
<tr>
<th>Type</th>
<th>Group 1 Expl. &amp; trust (%)</th>
<th>Group 2 explanation (%)</th>
<th>Group 3 trust (%)</th>
<th>Group 4 quest. only (%)</th>
<th>X²</th>
</tr>
</thead>
<tbody>
<tr>
<td>Type 1 (cancer)</td>
<td>4.3%</td>
<td>4.7%</td>
<td>5.0%</td>
<td>3.8%</td>
<td>0.5</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>NS</td>
</tr>
<tr>
<td>Type 2 (CHD)</td>
<td>11.9% (28/235)</td>
<td>12.3% (30/243)</td>
<td>7.8% (18/230)</td>
<td>5.2% (3/58)</td>
<td>p&lt;0.05</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Type 4 (health)</td>
<td>0.9%</td>
<td>1.1%</td>
<td>5.2%</td>
<td>4.8%</td>
<td>19.71%</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>p&lt;0.001</td>
</tr>
</tbody>
</table>

People who were assessed as being prone to health in Grossarh-Maticek’s full interview condition appear to have been inoculated against both cancer and CHD by the more in depth interview, as in the interview the message being communicated to them was that they were basically prone to positive health- this may have boosted their already high rates of optimism, self esteem, self efficacy, internal locus of control, etc. This suggests that the pernicious effect of receiving a poor prognosis could be matched by a health promoting effect of receiving positive health news in the course of an interview with conditions of trust and understanding. The immune system could be either enhanced by life events, in this case the interview and the associated cognitive, affective and endocrine processes, or it may be damaged by them.

Grossarh-Maticek, Eysenck and Boyle (1995 p.707) state,

“The analyses were complicated since the number of S's differed across each group and Type. Analyses were conducted on the raw scores adjusted to standard

166
raw scores in each instance so that direct, legitimate comparisons could be made. (It is of course not legitimate to undertake Chi-squared analysis on percentage scores, but these are given to facilitate appreciation of the trend of the data).”

In terms of explaining the different strengths of the observed relationships between interview condition and death by cancer and death by CHD, a plausible explanation is that Type 1 people (cancer prone) are referred to as neurotic introverted, while Type 2 people (CHD) prone are referred to as neurotic extroverted. There is a difference in terms of the amount of external cues which introverts and extroverts perceive and interpret from their environment, and how they respond to these in terms of arousability.

Monte (1995) in describing Eysenck’s personality theory, states that levels of cortical arousal are what differentiates between introverts and extroverts, as well as between neurotics and emotionally stable people. It is plausible that extroverts are less sensitive to subtly communicated messages (as their central nervous systems are generally less sensitive and arousable- their initial cortical arousal is low) and therefore were not as susceptible as the Type 1 introverts to the subtle communication of proneness to ill health. Monte (1995 p.811) cites research conducted by Levey in which “the most important parameter manipulation separating introverts from extroverts was stimulus intensity. Extroverts conditioned better with strong UCS (air puff), and introverts were superior with the weak UCS”. According to this theory of personality, introverts may be more likely to perceive subtle communications- this is supported by the fact that introverts are more conditionable (Monte 1995).

The primary personality characteristics described by Eysenck as neurotic are:- anxious, depressed, guilt feelings, low self esteem, and tension; the secondary characteristics are:- irrational, shy, moody, and emotional (Monte 1995p.823). Neurotics of both introverted and extroverted types can be expected to panic more over negative communications compared to non neurotics, while the introverts are more likely to pick up subtle environmental cues than are extroverts.
It is proposed here that the hypothesised self fulfilling/damaging prophecy effect of Groosart-Maticek’s interviews occurred as a result of an interaction between information gained in the interview (stressful life event in which a risk was perceived) and the subject’s predisposing personality characteristic of pessimistic attributional style, which then interacted synergistically over the subsequent years to produce biological outcomes.

A reasonable response to the above hypothesis could be to question:- i) whether such an interview, and learning of a cancer risk status, could constitute a stressful life event, and ii) question the ability of such a stressful life event to damage immune functioning- how would such a process operate on the physiological level suggested?

**The effects of stressful life events:**
Part of Cooper & Faragher’s (1989) research focused on the role of stressful life events as correlates to breast cancer and has already been reported earlier in this paper. They found a relationship between breast cancer and the death of a close family member or friend; *personal illness, especially if requiring hospitalization and/or surgery*; retirement and redundancy. A sense of control over such major events as serious personal ill health, death of a spouse or friend, retirement and redundancy is usually minimal if existent at all- these events are usually imposed upon people rather than being a matter of choice. Grossarth-Maticek, Eysenck & Boyle (1994) also researched the effects of stressful life events in their study with Jewish Nazi concentration camp survivors (detailed earlier). High Type 1 & 2 scores (the diathesis) was found to interact synergestically with the stressful experiences, producing higher mortality.

As the research reported by Grossarth-Maticek, Eysenck & Boyle (1994) is the subject of validation studies, it can hardly be regarded as its own evidence. In their review of research focusing on stressful life events and breast cancer Butow, Hiller, Thackway & Kricker (1997) identified methodological problems with most of the published papers, including those of Cooper et al (1989) above. In two limited prospective studies Geyer (1991; 1993) found that the most severe life events (those associated with loss), were more common in the cancer group, and that there was an interaction between severe life
events and family history in determining breast cancer risk. The adjusted regression coefficients, presented without standard errors or tests of significance, demonstrated a stronger association between life events ($r = 0.28$) and breast cancer than between age and breast cancer ($r = 0.19$). The study population used by Geyer (1991; 1993) were ninety-two women, thirty-three of whom were diagnosed with malignant disease, fifty-nine diagnosed with benign disease, and a control group of twenty women admitted for non cancer surgery.

Inspiring Butow et al’s confidence was the research program of Chen, David and Nunnerley, Mitchell, Dawson, Berry, Dobbs & Fahy (1995), in which the Brown & Harris Life Events and Difficulties Scale (LEDS) was the tool utilised. This is described as a measurement tool which attempts to capture a more complex conceptualisation of life events than do simple checklists. Chen et al (1995) examined seventy-two women referred for examination of breast lesions following mammographic screening and a second group of forty-seven symptomatic women undergoing biopsies; forty-one were diagnosed with cancer and seventy-eight with benign disease. Moderately threatening events that were experienced as important by the study participants were associated with increased risk of breast cancer, an association that was seen in both the screened and symptomatic samples. Butow et al (1997) state that these studies suggest severe or severely threatening life events may be associated with increased breast cancer risk.

A conclusion that can be drawn from these studies is that stressful life events may play a role in the onset of serious illnesses such as cancer. The effects of stressful life events may interact with the person’s psychological predisposition in terms of interpretation of events, interpretation of the event’s impact, and degree of controllability as well as degree of social support (Cooper & Faragher 1989).

Protheroe (1999) studied 333 women who had just had a biopsy or a needle aspiration for a suspicious breast lump. Using a similar quasi prospective methodology to Cooper & Faragher’s (1989), the results of the tests were not known at the time of sampling the subjects. Protheroe (1999) found no link at all between any of the measures of stress or
stressful life events and the likelihood that a woman would be diagnosed with a cancer. Unlike Cooper & Faragher’s (1989) study, Protheroe (1999) asked the women if they thought they had cancer before the test results were known. They found that the women who guessed that they would receive a positive cancer diagnosis were more likely to report a severe stressful life event compared to the women who believed they had a benign lump. As such, it can be concluded that the notion of stressful life events causing cancer has been incorporated into the culture’s risk perception equations. This finding renders retrospective studies as an inadequate approach, and semi-prospective (also meaning semi-retrospective) studies as being of limited use.

**Grossarth-Maticek’s interview and Risk Status as a Stressful Life Event:**

According to the cognitive appraisal paradigm of stress (Sarafino 1994), individuals undergo two cognitive processes in order to experience a given situation as stressful. Firstly, they must assess whether a demand placed on them from the environment threatens their well being; and secondly, the must assess whether they possess, or have access to the resources necessary to meet the demand. In the initial primary appraisal, the event is appraised for implications of the threat of future harm. The secondary appraisal refers to an individual’s sense of possessing the resources needed to respond to the demands of the situation. If a person judges the ‘fit’ between the demands and the resources available to be close, little or no stress ensues. If, however the resources are perceived as being inadequate to meet the demand, stress is experienced. For people to experience stress around the issue of cancer, they would have to perceive their well being to be threatened; and they would have to perceive the demands of cancer on them as overwhelming their abilities to cope with it (Brown & Lees-Haley 1992).

The amount of cancer worry experienced after learning of an elevated risk status is also a function of the amount of pre-news anxiety, which in neurotics is elevated above the norm. Easterling & Leventhal (1989 p.787) state that,

“In the case of worry or fear responses, abstract cognition corresponds to the underlying recognition that a threat exists. Such a cognition is instilled by some
stimulus that the individual believes to be informative, e.g being told by a health professional that one has a high risk of cancer due to family history”.

It is proposed here that the effect on probands of learning that they are cancer or CHD prone in the course of Grossarth-Maticek’s interviewees would have much the same as learning that they were cancer prone due to family history. Grossarth-Maticek’s interviewers are reported by him as believing in the psychosocial theory of illness; in the course of creating an interview climate of trust and understanding, they are likely to have conveyed this intuitively appealing theory to the subjects; and are likely, as such, to have appeared as well informed social scientist, ie. credible authorities on such issues.

Although Grossarth-Maticek has been repeatedly unclear as to exactly which items were used in his studies (according to Amelang 1997, he has used many), what is clear is that there were many items used in each interview - the Short Interpersonal Reactions Inventory (with 50 items) is a much abbreviated form of the full Interpersonal Reactions Inventory containing hundreds of items. It is more than plausible that after asking the respondent, say 10 items from the Type 1 cancer prone scale, the interviewer could have formed an impression of the subject in terms of their cancer proneness. In the full interviews, this may have occurred only 1/4 of the way into the interview, leaving around 3/4 of the interview remaining in which to inadvertently convey the subject’s typology to them.

Apart from this possibility, Grossarth-Maticek, Eysenck & Barrett (1993) state that in the most predictive interview condition, the interviewers created a climate of understanding and trust by discussing with the subjects for 45-60 minutes such things as their previous life experiences, good and bad, and their typical reactions to them. It is likely that an interviewer who believes in Grossarth-Maticek’s theory of psychological contributors to cancer would have had ample opportunity to decide on the subject’s typology even before the IRI was administered solely on the basis of the subjects reporting previous bad life events and their reactions to them. As demonstrated in other studies of experimenter bias, this could have been conveyed via non verbal communication such as: silences, smiles,
nods, eye contact or aversion, volume and tone of voice, signs of relief or concern, etc. It is also possible that Grossarth-Maticke’s interviewers may have submitted to pressure from the subjects and revealed to them their Type status; or that they thought it as part of their role, in creating trust and understanding to volunteer the risk status to the subject. Although this would be difficult to demonstrate, it makes sense that an interview condition that is trying to create understanding and trust would also entail explaining the IRI results to the subject—after all, who would not want to know the results? Why would an ordinary subject not ask the interviewer what his/her answers mean, and why would the interviewer not provide the answer when they are explicitly trying to establish conditions of understanding through answering all queries?

Miller (1996) reports that not all people in a situation of potential threat to their health will actually seek out more information. A particular stable coping dimension, referred to as 'Monitoring', describes those people who are likely to seek out more information and ask what the interview results mean for them. The opposite to monitoring, referred to as 'Blunting', represents those people who, as a coping strategy in response to potentially threatening information, are unlikely to seek out more information. More discussion of this important individual difference will occur in a later chapter.

It is suggested here that by the end of the lengthy interview, the subjects could have at the very least developed an impression of their risk status for cancer or CHD as conveyed by the interviewer in a manner standard to experimenter bias, if not having actually learnt of their risk status from the interviewer on asking ‘what does this tell you about me?’ From this situation, it is hypothesized that a risk perception was formed with an associated alarm reaction experienced. A sleeper effect is another possible result of the described situation with the perception of risk being ‘planted’ at the time of the interview with the risk perception and associated anxiety growing in strength in the following years, also leading to an eventual increase in stress hormones. Again, one would expect either of these responses to occur with the less psychologically well adjusted individuals who were prone to trait anxiety— one would not expect this outcome with more emotionally stable subjects.
The possibility of a large scale treatment effect (via interviewer bias and self fulfilling prophecy) as an explanation for Grossarth-Maticke’s results is proposed here as plausible according to well demonstrated psychological phenomena as treatment effects, experimenter/interviewer bias, and self fulfilling prophecies. This assertion is made on the basis of sufficient experimental evidence which clearly demonstrates the operation of these phenomena. The case for this possibility will be further explored in the following chapters.
Chapter 10

The power of expectations:

Evidence suggestive of self fulfilling prophecies:
Madon, Jussim & Eccles (1997) state that although throughout the 1980’s many researchers believed that the self fulfilling prophecy was a pervasive phenomenon, neither meta analysis nor naturalistic studies supported this conclusion. The evidence, they suggest points more towards self fulfilling prophecies as being generally rather small effects, ranging from around .1 to .2 in terms of standardized regression coefficients on target behaviours and achievements. However, rather than dismissing the notion of self fulfilling prophecies, Madon et al (1997) enquire about the conditions under which this phenomenon will occur, and with what types of targets and perceiver expectations. They suggest that in experimental settings, the moderators of self fulfilling prophecies have been established in terms of goals, personality characteristics, power relationships and situational factors, but that these moderators have not been so well researched under naturalistic conditions. In a naturalistic study involving 98 teachers and 1539 sixth grade students, Madon et al (1997) demonstrated that self fulfilling prophecies are more powerful for low academic achievers, i.e this population was more vulnerable to the effect of others’ expectations. They concluded that while on average self fulfilling prophecies may be small effects, “under certain conditions and for certain types of targets, self fulfilling prophecies may be more powerful than average sizes suggest” (Madon et al 1997 p.807).

As stated, the vulnerability of the subjects is one such factor. In Madon et al’s (1997) study, vulnerability was in terms of academic performance, but it is fair to suggest that vulnerability in a health research context could include factors such as trait anxiety. High achievers (or more emotionally stable people) have more psychological resources enabling them to combat negative expectations as they have had more successful experiences to draw upon, and have greater background coping abilities.
Madon et al (1997 p792) review the reasons why negative expectations may be more powerful in creating self fulfilling prophecies than are positive expectations. The following reasons are offered:- people often consider negative information more useful than positive information; people react more strongly to negative rather than positive feedback; and they often weigh the costs more heavily than rewards when making decisions. Madon et al (1997) concluded that there is evidence of powerful self fulfilling prophecies operating under certain conditions within their study.

In Grossarth-Maticek’s studies, Type 1 and 2 people could be described as psychologically vulnerable, and the expectation created can be described as negative, i.e the risk of cancer or CHD.

Bolletino and LeShan (in Watkins 1997), referring to the power of beliefs with people who are already sick with cancer, state that patients’ healing abilities are strongly affected by their expectations and beliefs, suggesting an immunological reaction to expectations:-

“Practitioner’s expectations, attitudes, beliefs and words strongly influence the expectations of their patients. Most cancer patients have clear expectations about the likelihood of recovery. Their expectations arise less from their medical prognosis than from their longstanding metaphysical expectations about life. Those who learned early that life can bring them good things generally expect to recover. Those who learned early that life disappoints them generally expect to die. Expectations can mobilize or weaken the powers of self healing”. (Bolletino and LeShan in Watkins 1997, p.102)

The powerful effects of prognosis created expectations are discussed by Bolletino and LeShan (in Watkins 1997) in detail. When a clinician informs the cancer patient of the diagnosis with worse case scenarios; or when a physician states or implies that there is nothing more that can be done for the patient; or when a prediction is made in terms of how long the person is likely to live for,
“the patient may well accept the practitioner’s ‘truth’ so that it becomes a prediction which the patient fulfills... How could any patient have hope if her own doctor conveys hopelessness?” (Bolletino and LeShan in Watkins 1997. p.103)

Bolletino and LeShan (in Watkins 1997. p.102) refer to a famous case study demonstrating the health enhancing and health damaging effects of positive or negative expectations. The case was reported by Bruno Klopfer in 1957.

“His patient, ‘Mr Wright’, had advanced widespread lymphosarcoma. His body was riddled with tumours, some the size of oranges. All standard treatments had failed, and he was expected to live no more than a few weeks. A new drug, Krebiozen, touted as a potential cancer cure, was being tested at the clinic where Klopfer worked. At Wright’s pleading, Klopfer included him in the trial. Shortly after he administered one injection of the drug, the patient’s tumour masses, Klopfer wrote, melted away like snowballs on a hot stove, and in only these few days were half their original size. Wright was soon released from the hospital, apparently free of malignancy. Two months later, when newspapers printed reports of the worthlessness of Krebiozen, his tumours quickly returned. Suspecting that this was due to Wright’s expectations, Klopfer decided to use him as a control patient. He told Wright that he would give him a double strength dosage of a new, more active form of the drug- and treated him with distilled water. Again the tumours melted away, and for the next 2 months Wright lived without symptoms. Then the newspapers published a report from the American Medical Association stating beyond doubt that Krebiozen was worthless. Wright appeared at the hospital a few days later. His tumours had reappeared, and he died within 2 days.” (Bolletino and LeShan in Watkins 1997. p.102)

As further confirmatory evidence, Bolletino and LeShan (in Watkins 1997) cite the observations conducted Walter Cannon, the researcher who identified the ‘fight or flight’ response in the 1920’s. Cannon was interested in studying the effects of ‘voodoo medicine’, specifically questioning “how it is possible that one human being can point at
another human being and say that at a specific time that person will die- and the prediction is fulfilled?” (Bolletino and LeShan in Watkins 1997. p.102). The phenomenon suggests the role that fear might play in terms of bodily responses to frightening expectations. To this end, Cannon observed people in hospital who had been hexed in a voodoo context, and watched them die of no determinable cause. “He discovered that what happens is that the individual accepts the truth of the witch doctor’s prediction. That ‘truth’ can lead to death... In voodoo death... belief becomes biology” (Bolletino and LeShan in Watkins 1997. p.102).

The parallels between the expectation of death created by a cancer diagnosis and the witch doctor’s hex are obvious- the negative cancer prognosis can operate as a modern day scientific curse. Archerberg (cited in Watkins 1997. p.102) writes,

“Images are so readily translated into physical change that dying from having been given a feared diagnosis by a credible physician is just as feasible as a hex death by a cursed Haitian”.


“Even though the physician dutifully describes the prognosis as ‘survival’ statistics, the curse frequently has already done its work. Even though patients may be told they have a 50% chance of living another 5 years, their interpretation is frequently that they have a 50% chance of dying. Like the accursed individual in voodoo societies, they may cooperate by succumbing ‘on time’ ”.

In a study of 238 terminally ill cancer patients Schulz, Bookwala, Knapp, Scheier & Williamson (1996) found that a pessimistic outlook served as an important mortality risk factor among younger patients (30-59), but less so for older patients (over 60). They concluded that psychological factors like pessimism may have less of an impact in terms of cancer mortality for older terminal cancer patients, as it is more normative for that age
group, and may in fact represent an adaptive response to their reality. Where as for the younger age group, pessimism is less normative as is cancer, leading to more negative behavioural consequences. They make the point that as a younger person with the expectation that much life is yet ahead, the psychological and physiological reality of cancer is likely to be different compared to the older person. As such, their results demonstrated a clear relationship between pessimism and cancer mortality for the younger group only, and they suggest that psycho-social factors become less important to mortality as the person gets older (ie. over 60). It is worth keeping in mind here that at the point of the assessment interviews in the two large Heidelberg studies, Grossarth-Maticek’s subjects were on average around the age of 50, whereas in the Yugoslavian study, the average age of participants was 60, at the highest end of the younger age range as described by Schultz etal (1996). As such, according to Schultz etal’s (1996) findings in regards to age, a pessimistic outlook could be expected to have exerted an important mortality risk for Grossarth-Maticek’s subjects in both studies.

If it is demonstrated that expectations can act on the biological system to either enhance or damage the health status of people with cancer, it is only a small step to question whether expectations can operate in a similar fashion over time on people who are currently healthy. Grossarth-Maticek & Eysenck’s (1989) article on the effects of health warnings related to cigarette smoking has already been reviewed. The worst outcomes were found for those subjects who believed smoking was going to cause them harm, especially when they obtained this view from the media.

It is possible that in Grossarth-Maticek’s larger studies, the stress induced from obtaining a perception of being in an elevated risk group added to stress levels in the same way as the ‘smoking= elevated risk’ information, and also produced a self fulfilling prophecy in the directions of cancer and CHD, thus the 81% accuracy of predictions.

Phillips, Ruth & Wagner (1993) provide more evidence which is suggestive of the power of self fulfilling prophecies to adversely effect health outcomes. They studied the role which cultural beliefs in Chinese astrology may have in the amount of life years lost due
to a range of illnesses. In order to test the hypothesis that a belief in the power of year of
birth in the Chinese astrological system is related to amount of life years lost, the
researchers obtained death data of 28,169 Chinese-Americans and 412,632 Americans
whose death certificates nominated them as being ‘white’. In the Chinese astrological
system, certain years are believed to be ill fated in terms of health, while other years are
believed to be lucky in terms of health. Phillips et al (1993) found that the more these
traditional beliefs were subscribed to, the more life years the Chinese-Americans lost
when compared to white Americans born in the same years and dying of the same
diseases. The range of differences in life years lost varied from 1-5 years. These results
were found to be independent of extraneous variables such as how well the patients
looked after their health and the treatments which they were given for their diseases. This
study demonstrates the possible role which powerful beliefs, on this occasion cultural
beliefs, may play in issues of health and mortality. Those subjects who were closer to the
traditional Chinese astrology belief systems were the most susceptible to shorter lives
compared to those Chinese-Americans who did not subscribe to traditional beliefs, or to
white Americans who did not subscribe to these beliefs at all.

A similar role for the power of health beliefs is being hypothesised in this thesis to
explain Grossarth-Maticek’s results. Traditional Chinese-Americans who are unlucky
enough to have been born in certain years, and who are unlucky enough to adhere to
beliefs in their bad luck, must place themselves in a higher risk group for poor health and
mortality. This creates a set of expectations about health outcomes which appear to have
had physiological and immunological correlates. There may be a similar elevated anxiety
level for Grossarth-Maticek’s most vulnerable subjects, with similar immunological
correlates. Phillips et al’s (1993) results are difficult to explain on any grounds other than
a vague reference to the power of expectations. The expectations referred to here are
pessimistic ones, involving a belief that fate has dealt these people inevitable poor health.
With this in mind, it is worth re-examining some of Seligman’s (1991) conclusions
concerning the power of pessimism to produce ill health.
A possibility that cannot be excluded in explaining the results described above, as well as those of Grossarth-Maticek may be that people with negative expectations of health outcomes (eg. Chinese Americans born on bad luck years, Grossarth-Maticek’s Type 1 and Type 2 subjects, Seligman’s pessimists) may be more prone to an external locus of control and poor self efficacy in regards to health behaviours. That is, they may conclude “well, I’m likely to die from cancer/CHD disease anyway (by virtue of being assessed as Type 1, ‘bad luck’ birth year), so what’s the point in giving up cigarettes, alcohol, unhealthy food, or exercising and attending the physician for regular check ups?” From within a pessimistic cognitive set, this is a reasonable conclusion, with consequent unhealthy behaviour patterns for a person with little self efficacy in regards to their potential for health.

The issue of control is of central importance in Marmot’s Whitehall studies (Stafford, Hemingway, Stansfeld, Brunner & Marmot 1998), and it will be argued here that it also has implications for what may have happened to Grossarth-Maticek’s subjects. It is possible that Grossarth-Maticek’s results were obtained from a combination of negative health behaviours supported by the interview induced risk perception, and a powerful self fulfilling prophecy which produced sustained anxiety and associated physiological consequences. Grossarth-Maticek’s interviews had the power to generate fear of cancer/CHD in the subjects- these conditions are so feared, at least in part because they are perceived as being uncontrollable.

Josten et al (1985) state that fear is a powerful factor in predicting if a person will engage in health promoting and illness preventative behaviours. For example, people who are very fearful of contracting colorectal cancer, and women fearful of contracting breast cancer are less likely to have colon examinations or to practice regular breast self examinations- fear has the power to discourage people from engaging in constructive health behaviours. Kash et al (1992) confirm that a more optimistic sense of self efficacy in women with a genetic risk for breast cancer produces more preventative health care behaviours, while women who perceive themselves as high risk and have high levels of distress are the least likely to engage in such behaviour. For Grossarth-Maticek’s
subjects, obtaining the impression either directly or indirectly that they were viewed as being cancer/CHD prone could be expected to create a sense of extreme powerlessness in the most vulnerable subjects. A chronic sense of lack of control has been shown by Marmot and colleagues to have extremely damaging effects on health, in fact to be the factor which explained the gradient between health outcomes according to position in the organizational hierarchy.

In summary of the major research findings Dykema, Bergbower & Peterson (1995) state that previous studies have found links between pessimistic explanatory style and poor health operationalized in the following ways: by physician examination, self reported symptoms, immunosuppression, and survival time with cancer and heart disease. An examination of Grossarth-Maticek’s Short Interpersonal Reactions Inventory demonstrates that the items in subscale 1 (cancer prone) and subscale 2 (CHD prone) are primarily pessimistic attributions of one form or another which Type 1 and Type 2 respondents endorse. As such, there is a close link between Seligman’s (1991) construct of pessimistic attributional style, immunosuppression measures, poor health outcomes and Grossarth-Maticek’s Type 1 and Type 2 personality profiles (related to an elevated death by cancer and CHD in his sample). The internalization of a poor health prognosis (eg. cancer/CHD prone types) and a subsequent self fulfilling prophecy is more likely in a person who can be characterized as possessing a pessimistic attributional style. A perception of lack of control in regards to cancer and CHD proneness may have been a key factor in the mortality outcomes for Grossarth-Maticek’s vulnerable subjects- again, certain types of subjects could be expected to respond to the perception of risk with characteristic pessimistic expectations, believing that they were powerless to prevent the onset of the feared condition.

What conditions are required for a currently healthy asymptomatic person to view themselves as being at risk of cancer/CHD? This question will now be examined as no proposed self fulfilling prophecy could occur without there first being a perception of risk with subsequent anxiety.
A Psychological Model of Expectations:

To this point, a range of empirically demonstrated findings have been brought together to suggest the hypothesis of this study that a large scale interviewer bias may have created self fulfilling prophecies amongst the most psychologically vulnerable of Grossarth-Maticek’s subjects. In addition to this, a psychological model articulating the process or ‘mechanism’ by which the above suggestion may arise must be possible. Kirsch (1990) proposes that in explaining both therapeutic psychological and medical change, and thereby its opposite- ‘therapeutic harm’, expectancy theories contained within the broader Social Learning Theory are most able to account for such outcomes.

Social learning theory can be seen in its many facets influencing psychological constructs since the 1930’s when Tolman first emphasized the role of expectations in his cognitive learning theory (Kirsch 1990). Although referring to himself as a behaviourist, Tolman argued that behaviour could only be understood in regards to mental processes, especially expectations, which by necessity must be inferred. Julian Rotter also made the role of expectations central to social learning theory in the mid 1950’s, leading to the many later variations of this general psychological approach to learning, such as Bandura’s, Mischel’s and Seligman’s modifications and extensions (Kirsch 1990).

One primary feature which all approaches to social learning theory share is the proposition that most important human behaviours are a function of learning. Learning itself is viewed as primarily a cognitive process by which new information is acquired through direct experience (such as classical and operant conditioning), or indirectly through observation, modeling or other forms of vicarious learning. In Rotter’s 1954 social learning theory, expectancy and reinforcement value are the central constructs (Kirsch 1990). The other main aspect of Rotter’s approach is the notion of ‘situational specificity’, making it compatible with behaviour modification (Rappaport 1977).

Expectancy, the facet which introduces cognitive considerations to social learning theory, is defined as being the person’s estimate that a certain event will occur, while reinforcement value is the subjective value of the anticipated event- they are subjective
probability statements. Any particular behaviour is viewed as being a joint function of these variables, i.e. a behaviour is performed if there is an expectation that it will lead to a desired outcome. Outcomes which have no value to the person are unlikely to affect their behaviour, as are potential outcomes that are seen as being entirely improbable. Conversely, highly valued outcomes which are viewed as being probable are seen as factors in the production of certain behaviours. Complicating the picture is the reality that any behaviour is likely to have more than one possible consequence, so people need to consider all salient expected outcomes and their reinforcement values in making decisions which lead to behaviour. Most situations also offer a range of alternate choices, so in order to predict behaviour it is important to know the available alternatives and each of their associated expectancies and reinforcement values.

Kirsch (1990) suggests that Rotter’s locus of control construct, a belief about the cause of outcomes, is the most well researched generalized expectancy. This topic of study continues to the present in the form of Seligman’s (1991) approach to attribution theory, with its many implications for health outcomes. Seligman’s construct of ‘learned helplessness’ added a focus on emotions to social learning theory, as did the cognitive-behavioural emphasis on the role of cognitions in emotions. Apart from affecting behaviour, expectancies are seen to have emotional consequences. Bandura (1977) also contributed to social learning theory in distinguishing between expectancies that were based on beliefs about one’s own capabilities (self efficacy), and expectancies about environmental contingencies (outcome expectancies).

Kirsch (1990) suggests that most expectancy theorists have researched the role of stimulus expectancies, or the anticipated outcomes which are external events or objects such as money, school grades, social acceptance, promotion, etc. He adds to the spectrum of social learning constructs with the notion of response expectancies. These are the outcomes which are anticipated from certain responses, or behaviours. The basic model of social learning theory states that behaviour probability (BP) is a multiplicative function of expectancy (E) that the behaviour will lead to a particular outcome, and of the reinforcement value (RV) of that outcome. (Kirsch 1990). From particular situations, we
form sets of broad generalized expectations about broad classes of situations and behaviours, particularly applied in new situations. The focus of behaviour change is on changing expectations as reinforcement values tend to be very stable, e.g certain behaviours are almost always reinforced while others are almost always punished.

Response expectancies are important in that they tend to be self confirming.

“When we expect to experience something strongly enough, we find ourselves actually experiencing it. Response expectancies can alter our experiences of pain, nausea, tension, anxiety, depression, sexual arousal and relaxation. They also tend to affect the physiological processes with which these subjective experiences are associated, for example pulse rate, blood pressure, gastric motility, penile tumescence, and the release of endorphins in the brain.” (Kirsch 1990 p.10).

Subjective experiences, such as those listed above tend to be non-volitional in character— we cannot directly control how much pain, fear or anxiety we feel, although we can do things to indirectly alter internal states such as these. Response expectancies are able to affect subjective experience (such as anxiety) and associated physiological states as, for example, it is frightening to seriously consider that one is going to experience a life threatening illness such as cancer or CHD. When excited or frightened by a response expectancy the heart will beat faster, or slower when relaxed by a non alarming response expectancy. Kirsch (1990) points to the case of Klopfer’s cancer patient (referred to earlier) as evidence of what a response expectancy can do to a person’s immune response- the patient, based on what he had been falsely told of the ‘medication’ went into remission as he developed a response expectancy that the placebo was in fact a cancer curing agent.

‘Self-fulfilling prophecies’ are viewed by Kirsch (1990) as being another form of self-confirming expectation. For a student to be affected by a teacher’s expectations, or a subject to be affected by the experimenter’s/interviewer’s expectations:-
the expectation has to affect the behaviour of the teacher, experimenter, or interviewer.

the student, subject or interviewee must perceive the difference in the other person’s behaviour (research has clearly demonstrated the ability of subjects in experiments, or interviewees in interviews to perceive the changes in experimenter/interviewer behaviour.)

and finally, that perception must generate behaviour that is consistent with the expectation (subjects leaving an interview with a new or heightened sense of risk would be expected to behave differently according to this perception of risk.)

All of these pre-conditions appear plausible events in Grossarth-Maticek’s full interview condition, acting as indirect contributors to the internal state and behaviour of the subjects. Response expectancies, however, more directly affect behaviour and internal states as they are anticipations about one’s own experience. In Kirsch’s (1990) terms, it is plausible that Grossarth-Maticek’s most psychologically vulnerable subjects left the full interview condition with the response expectancy that they were likely to experience cancer or CHD.

The discovery of the placebo effect in the 1950’s, with the obvious role of expectancies, led to it being treated as an artifact needing to be controlled experimentally, whereas Kirsch (1990) is suggesting that as a psychological variable it needs to be treated as a main effect. Of relevance to the current study, in addition to the beneficial effects of placebos, they have been shown to also mimic symptoms of unwanted side effects of genuine medications, such as:- drowsiness, weakness, confusion, headache, nervousness, insomnia, nausea, constipation, dizziness, dry mouth, cramps, anorexia, vomiting, delirium, diarrhea, nightmares, tremors, skin rashes, sweating, fatigue, and constriction of pupils (Kirsch 1990, p.14). That is, these symptoms have been found in subjects who ingested harmless agents, but were told they were medications with the above side effects. As such, it can hardly be said that response expectancies cannot have an effect on a recipient’s physiological processes- these reported side effects all occurred as the result of an idea, or an expectation that they would occur. The changes brought about by
placebos have been corroborated by corresponding physiological changes such as pulse or heart rate, skin temperature, systolic and diastolic blood pressure and gastric motility (Kirsch 1990).

The above cited negative physiological changes which have been observed arising from the administration of placebos suggest the role of fear and anxiety. Kirsch (1990 p73) states that “People fear and avoid situations that are dangerous- in other words, fear is produced by the expectation of harm”. The amount of fear generated is dependent upon the amount of harm that is expected and the perception of likelihood of that harm occurring. As stated in an earlier section of this paper, cancer and CHD are amongst the most feared medical conditions in our culture (Holland 1996). Our cultural understanding is that both of these conditions can cause a great deal of harm to the organism (ie. pain, disability, disfigurement, and death). It is reasonable to suggest that when people develop a subjective expectancy of these conditions occurring, fear will result from this expectation.

Social learning theory, and in particular expectancy theory provides the structure by which this process can be better understood. What are the psychological effects of learning of a cancer/CHD diagnosis, or of the prospect of being prone, thereby developing a set of expectations for illness? This will be explored in the next chapter.
Chapter 11

Psychological sequelae of disease risk

Risk perception and response to risk notification:
The perception of risk or threat is central to the current study as it is proposed here that a process which either created or heightened perception of chronic health risks (i.e cancer and/or CHD), leading to an alarm response was initiated in the course of Grossarth-Maticek’s full interview condition. It is proposed that subjects entered these interviews with either little or no perception of cancer/CHD risk, and those who were assessed as being cancer or CHD prone left the interviews with a new perception of risk as a result of an interviewer bias or direct information. Further, it is proposed that this new risk perception and associated worry made psychologically vulnerable people even more vulnerable to a health damaging psychological effect via long term immuno-suppression.

As demonstrated elsewhere, cancer elicits an enormous amount of anxiety in our culture, and an actual diagnosis is often experienced as a life crisis. But what of the effects of finding oneself in a group which is currently healthy but is predicted to contract cancer at a later time?

Frost, Walsh Vockley, Suman, Greene, Zahasky & Hartman (2000) state that several factors influence the psychological outcomes for women at increase risk of breast cancer. These include:- age (women less than 29 years old have the highest levels of global distress about the prospects of breast cancer); the use of denial; levels of support; higher levels of perceived cancer risk; and a perception of there being many barriers to effective screening have all been found to increase psychological distress in relation to breast cancer risk (Frost et al 2000).

It is also reasonable to expect that people who are characterised as neurotic (in Grossarth-Maticek & Eysenck's Type 1 & Type 2 terms, more prone to feelings of helplessness and hopelessness, pessimistic, more easily upset, etc) will respond with more internal distress
than the emotionally stable personality. As such, people who are assessed as being Type 1 or Type 2 (both considered to be neurotic) could be predicted to be just those people who will respond to such news with more anxiety, pessimism, dwelling on their bad luck, considering the negative impacts, etc. The more neurotic a person is (reflected in higher Type 1 and Type 2 scores) the higher would be the expected impact on their cognitive and emotional state, leading in some or many to a crisis in which they perceive that their very existence is now threatened by a highly feared and uncontrollable disease.

While this suggestion has not been empirically tested, there are many people in the community who have been given a heightened risk perception by virtue of having first order relatives with cancers that are known to have hereditary components. The findings concerning this group of people will provide clues as to how one would expect Grossarth-Matick’s subjects to respond on learning of or perceiving the view that they were people at risk of cancer or CHD. Before presenting these findings, it is important to understand the cognitive processes involved in developing a risk perception.

Cognitive processing models of risk perception:
As with other stressful life events, the response to finding oneself in an at-risk group will depend on the interaction effect between the event and the person’s diathesis, or personality factors. Absetz, Arja & Sutton (2002) state that a perception of risk to cancer is associated with psychological distress, but research does not support the view that this life event would be equally stressful to all people- its effects will be mediated by cognitive/affective factors such as level of emotional stability, optimism/pessimism, locus of control, self efficacy, etc. Absetz et al (2002) state that individuals differ in their coping styles in response to stressful events. The health psychology literature has discussed coping styles in terms of:-

- **attentional styles** in information seeking (eg. Miller);
- **situation specific coping strategies** (eg. Folkman and Lazarus);
- and **dispositional coping styles** (eg. Carver et al, Endler and Parker).
Despite these different conceptualizations, the proponents have accepted the notion that there are two basic dimensions of coping.

1) Coping directed at reducing the threat - eg. monitoring, problem focused coping, approach coping, and

2) Coping directed at reducing the emotional reaction caused by the threat - eg. blunting, emotion focused coping, avoidance coping.

While adaptiveness depends on the circumstances of the situation, problem focused coping is usually seen as less associated with depression and more effective in the long term. Emotion focused coping and avoidance are usually associated with greater psychological distress and as such are probably less functional (Absetz et al 2002).

‘Perceived susceptibility’, as a factor in the Health Beliefs Model (HBM), refers to an individual’s personal estimate of the chances that he/she will contract a particular disease (Rice 1998). A central component of this estimate is the person’s degree of belief in the medical diagnosis or prognosis. If the individual accepts the ‘expert opinion’, then a greater perception of risk is likely to ensue.

Characteristics of the risk communicator:
Covello (1992) provides a detailed discussion of what the research literature suggests are the most important factors in successfully communicating a message of risk to the recipient, ie. successful in terms of the recipient absorbing and internalizing the risk information. He states that trust and credibility are the key factors in successful risk communication- only when these factors are established can other goals such as education and the sharing of information follow. More broadly, there are four factors which influence perceptions of trust and credibility. These are:-

- perceived caring and empathy
- perceived competence and expertise
- perceived honesty and openness
- perceived dedication and commitment.
The communicators who are most trusted in our society are health professionals, university professors, professional organizations, and nonprofit volunteer health organizations (Covello 1992). Research has demonstrated that of the above, physicians and university professors are the viewed as being the most credible in conveying risk communication for the following reasons- 1) they are perceived as being motivated by higher goals than ordinary commercial ones, e.g physicians are seen as dedicated to healing, and university professors as being dedicated to truth and knowledge. 2) both are seen as being financially independent from the organizations which may hire them and therefore not beholden to their views. As such, physicians (or more broadly health professionals) and university professors (and more broadly those who are working towards this status) do not have to work as hard as other people to establish their trust and credibility in risk communications- there is an assumption that by virtue of their positions they are trust worthy and credible (Covello 1992).

Risk communication is effective to the extent that the recipient perceives the communicator as possessing the characteristics of credibility and trust worthyness. Caring and empathy is demonstrated to the recipient when the communicator demonstrates good listening skills, giving the recipient the sense that they have been given the opportunity to be heard. Perceptions of competency and expertise are influenced by the communicator’s ‘merit factors’, i.e education, experience, knowledge, professional recognition, and presentation skills. Perceptions of honesty and openness are fostered when the communicator’s actions and words convey truthfulness and candidness. Perceptions of dedication and commitment are influenced by a perception of the communicator being committed to hard work and diligence in the pursuit of such goals as health, safety and environmental concerns.

One can estimate the extent to which Grossarth-Maticzek and his team of interviewers were perceived as being trustworthy and credible by the study subjects, and thereby evaluate how likely was the proposed risk communication to be believed. Firstly, simply by virtue of being an academic researcher and a PhD of a health profession, Grossarth-Maticzek himself fulfills an equivalent to the role of a health professional and a university
professor— the credibility attributed to these professionals is easily transferable to Grossarth-Maticzek. His team of interviewers were PhD candidate psychology students—while their immediate credibility may not be as high as Grossarth-Maticzek’s, there could still be expected to exist some vicarious credibility by virtue of their association with Grossath-Maticzek and his research project. They would also have been perceived as being ‘fledgling’ university professors and/or health professionals, thereby earning some immediate credibility by virtue of their merit factors leading to perceptions of competence and expertise. It is also likely that the need for perceptions of dedication and commitment would have been fulfilled by both Grossarth-Maticzek and his interviewers as the research enterprise was seen as being a pursuit of health goals, and not commercial goals.

Secondly, the conditions of the full interview condition explicitly fostered the stated requirements for maximizing trust and credibility. The behaviours of the interviewers were designed to foster trust and understanding in the researcher as well as the research goals. This was achieved by the interviewer listening to the subject describe positive and negative life events and their typical reactions to these; and providing as much information as was necessary for the subject to gain a sense of understanding. Via these behaviours, the interviewers would have established a perception of caring and empathy as they were using standard psychological interview micro-skills such as active listening, empathic responses, etc. Further, the goal of establishing a condition of understanding by answering any questions pertaining to the research would have created a perception of honesty and openness.

As such, it can be seen that Grossarth-Maticzek and his interviewers, as well as the full interview condition in general, fulfilled all of the necessary conditions for trust and credibility to be established in the interviews. Consequently, it is highly probable that if the perceptions of risk according to Grossarth-Maticzek’s theory was communicated either overtly or unintentionally, the risk message is likely to have been effective in having the recipient internalize the perception of risk.
Characteristics of the message recipient.
The following models are presented as depictions of the type of cognitive processes operating in the recipient of the message in the creation of risk perception.

C-SHIP Model
The Cognitive-Social Health Information Processing (C-SHIP) Model, developed by Miller and Diefenbach (in Krants et al 1998) is concerned with the cognitive and affective processing in which people engage in relation to health issues, medical risks and treatment options, as well as the outcomes in terms of health enhancing or health defeating behaviours. The structure of relations between the range of cognitive-affective mediating units which process cancer information have been extensively studied by Miller and colleagues. Drawing on other cognitive-social approaches and constructs, the cancer threat relevant mediating units in the C-SHIP model are:-

- the individual’s encodings and constructs
- outcome expectancies
- self efficacy beliefs
- health goals and values
- affects that become activated by new or changing cancer threat information and treatment options.

According to the C-SHIP model, information which conveys a cancer threat, such as an abnormal screening result, genetic testing or feedback, worrying sensations or symptoms, is first encoded and integrated into the individual’s existing knowledge and memory system. Both schematic and conceptual memories are important in terms of referring to past illness episodes, as well as making inferences, abstractions and judgments about cancer and its emotional consequences. These memory systems determine subsequent health decisions and behaviours. The Monitoring Process Model (MPM) is offered by Miller et al (1996) as a component of the C-SHIP Model and is guided by it. Gattuso, Litt & Fitzgerald (1992) suggest that Miller’s MPM represents an interactional approach,
incorporating both trait and situational elements, taking into account the person/situation interaction.

Miller et al (in Krantz et al 1998) refers to extensive research in proposing that there are stable differences between individuals in terms of the encoding patterns which are used in response to information about cancer risks and management options. People appear to differ in terms of the coping styles/encoding patterns which they use in response to cancer threat information. These differences are evident in terms of distinctive patterns of information searching and characteristic ways of responding cognitively and emotionally to threatening information. Miller (in Shoda, Mischel, Miller, Diefenbach, Daly & Engstrom 1998. p8) states,

“Every individual is characterized psychologically by a relatively stable structure of relations among cognitions and affects that are likely to be activated when encountering a certain type of information, such as the results of a genetic test for cancer susceptibility.... A second key principle is that relationships among an individual’s cognitions and affects are not necessarily logical or under conscious control, and the individual is frequently unaware of them.”

Miller & Diefenbach (1998) state that some people, which they refer to as ‘high monitors’ are more likely to attend to cancer risk information, scan their environment for threat and amplify the cancer threat or risk perception. When presented with risk information, these people typically focus on the threatening aspect of the situation (eg. the person perceives themselves to be at great risk for cancer on learning that their mammogram is abnormal). As a consequence to learning of risk information, it is the high monitors who can be expected to experience greater concerns, more prolonged distress and a greater sensitivity to diagnostic regimens (Miller 1996). People who are characterized as low in monitoring utilize the coping style of distracting themselves from and not attending to risk information. As a result, they tend to be less distressed and
anxious than high monitors. The coping style referred to as ‘Monitoring’, and its opposite referred to as ‘Blunting’, are essential components of the C-SHIP model.

In more detail, Monitoring refers to the tendency of the person to scan for and to amplify threatening information to the point of obsessing about potentially negative outcomes. Miller & Managan (1983, p236) states that,

“High monitors seek out and attend to threat relevant information when confronted with threat, which generates a high degree of intrusive and repetitive ideation about the stressor”.

Miller et al (in Krants et al 1998) state that high monitors typically focus selectively on the threatening aspect of the situation when faced with cancer risk or disease information. They view themselves as being vulnerable and fragile and are very sensitive to cues about potential illnesses, especially those that are perceived as being uncontrollable and potentially chronic, such as cancer (Miller 1996). In addition, they tend to encode neutral or ambiguous information (both internal and external) as being highly threatening, to ruminate on this information and to view it as evidence of the perceived threat (Schwartz et al 1995). Consequently, these people experience greater anxiety and concern, more prolonged distress and a greater sensitivity to diagnoses compared to low monitors. In addition, due to their elevated anxiety levels and an obsessive focus on the threat, these people ultimately tend to cope with their distress by developing an avoidance pattern, attempting to avoid cues which prompt cancer related thoughts or feelings. An outcome of this strategy is that, as with other anxiety conditions, the attempts to avoid threatening cues acts as an interference to processing and coping with the cancer threat. While trying to avoid external cues to the threat, high monitors respond to internally generated threat cues, and appear to be unable to ‘switch off’ this intrusive and repetitive ideation. These people fail to de-sensitize to the threat as they prevent the process of acceptance and adaptation- consequently, they remain highly anxious and continue with their extreme vigilance of threat cues, some of which are internal somatic cues.
**Monitors** are more likely to engage in the following strategies:-

- ask about what exactly is going to be done
- want to be warned of imminent pain
- vigilantly watch procedures for threat
- look for internal signs of injury
- stay alert in the situation
- scan the environment for any relevant information
- look for possible ways of escaping the threat
- try to mentally work out ways of escaping the threat
- ask questions that increase chances of escaping the threat
- talk to others in an attempt to obtain any relevant information.

Miller (1996) states that monitors have been found to have lower thresholds for detecting bodily cues and tend to amplify them. As a result, they detect or over interpret new or changing physical symptoms and experience more arousal and negative internal states when compared to people low in monitoring. These responses to risk threat are similar to people’s responses when they experience traumatic events which are intense and prolonged, and which violate the expectations of being able to control important aspects of life (Miller 1996). As such, the high monitor response of elevated anxiety and dysfunction, combined with an attempt to deal with intrusive ideation by avoidance, is similar to the psychological profile of PTSD in response to a traumatic event.

Phipps & Zinn (1986) state that Monitors have also been referred to as ‘sensitizers’ and ‘vigilants’, while Blunters have been referred to as ‘repressors’ and ‘distracters’. In contrast to Monitors, Blunters are characterized by a coping response which utilizes distraction from the threatening information- Monitors want to know more about the threat, while Blunters want to know less and do not seek this information out. This strategy is effective in the sense that Blunters experience less anxiety and distress than high Monitors as they actively distract their attention from such information. Blunters also report to experiencing more self efficacy in relation to their ability to cope with the
threat, are less concerned with trying to suppress and contain intrusive ideation, and consequently feel less victimized by such thoughts and images (Miller 1996).
Conversely, Monitors are more sensitive to their own ruminative nature, and as such focus on and exaggerate even minor potential health threats, turning them into catastrophes.

**Blunters** are more likely to engage in the following strategies when confronted with a feared situation:-

- take a tranquilizer or have an alcoholic drink
- think about pleasant memories
- try to sleep
- do mental puzzles
- daydream and fantasize
- talk with others as a distraction
- distract self with watching movies, reading books
- control one's negative thoughts
- deny anything is wrong and proceed as normal.

The differences between Monitors and Blunters are not evident under ordinary, non threatening conditions- they only become apparent when under threat conditions, that is when ‘the heat is on’. The prototypic example is when they are confronted with the prospect of a potentially major or life threatening illness such as cancer (Miller 1996). Schwartz, Lerman, Miller, Daly & Masny (1995) state that among women who are at familial risk of ovarian cancer, Monitors are more likely to display exaggerated risk perceptions, intrusive thoughts about cancer, and general psychological distress.

From the above, it may be gathered that both Monitoring and Blunting have their advantages and disadvantages in terms of psychological and physical health, depending on the particular situation. For example, Monitors tend to seek out further diagnostic information, and due to this vigilance are more likely to seek early health screening and
intervention, while Blunters are less likely to seek early screening or treatment. Obviously, health benefits can follow from early detection of abnormalities, however this needs to be weighed up against the cost of chronic heightened anxiety, especially in light of research conclusions from psychoneuroimmunology. Also, such chronic anxiety tends to interfere with effective problem solving and adaptive coping (Miller 1996). Blunters are generally less anxious and ruminative, leaving them with a more sanguine disposition and able to cope with and adapt to adverse information, however due to this, they tend to avoid health protective behaviours that can objectively reduce their health risks.

As Monitors seek out bad news, see it pervasively internally and externally and exaggerate its threatening significance, it stands to reason that they will also experience high levels of distress and negative affects. Miller (1996) states that while Monitoring may be correlated with measures such as generally negative affect, depression, or anxiety as theory would suggest, it appears to not be a redundant construct nor is it explicable in terms of these other constructs. Monitoring appears to be a coping strategy/encoding pattern that is factorially independent from general negative emotionality, trait anxiety and depression. Although it could be expected to correlate with neuroticism, it is not the same factor. In regards to other cognitive psychology constructs, Miller & Managan (1983, p223) state that a high Monitoring response pattern plays a part in the learned helplessness/hopelessness syndrome, which “...been shown to be related to cancer, perhaps even more consistently than depression”. Learned helplessness, they suggest, is a reflection of negative encoding (“I am vulnerable”), pessimistic outcome and poor self efficacy (“there is nothing I can do about it”), and negative affect states. The Monitor, by virtue of their vigilance, excessive replaying and rumination over the feared information, experiences extreme anxiety which may be sustained over a protracted period of time.

Like negative emotionality which appears to be an enduring characteristic with a portion of the population, high Monitors can also experience long term intrusive negative feelings and cognitions (Miller 1996). Their tendency to ruminate and rehearse the ‘bad news’ which they have received in terms of a risk factor or diagnosis, ensures that the
long term psychological and somatic costs of this elevated anxiety are substantial, depending on the duration and the intensity of the threat to their life or well being as well as the other cognitive factors alluded to in the C-SHIP model (Miller 1996). Monitors are more likely to re-live the stress generated by the threat information via intrusive thoughts, images and memories, and to respond with intense distress to reminding cues of the threat. Miller (1996) states that Monitors and Blunters do not differ on initial psychological or physiological measures of arousal when exposed to threatening information, however Blunters tend to diminish their anxiety over time by either not seeking out information or cues concerning the threat, and/or by habituating to the threat. In contrast, Monitors sustain higher levels of arousal over time as they are seeking more information and they show less habituation to the threat. Even in the absence of ongoing threat cues, Monitors will still experience threatening signals while remaining unaware of safety signals.

As with other dispositional constructs within psychology, most people are neither just a Monitor nor a Blunter, but lie somewhere along the continuum. In addition, this tendency will interact with other personality, behavioural, and situational variables to produce particular outcomes- no predictions of individual responses is possible without taking into account the interactions between the following personality variables. As suggested by the C-SHIP model, factors such as the person’s self concept, their typical affective states (eg. the ease with which s/he becomes depressed, a factor of neuroticism), their outcome and efficacy expectations, self regulatory and problem solving capacities as well as their values and major goals all interact with the demands of the situation to determine effective problem solving and adjustment. Miller (1996) states that a sense of risk and distress are likely to be acute for the subset of Monitors who resort to a pessimistic attributional style, expecting the worst possible outcomes for themselves in terms of their health, having very little self efficacy for dealing with the situation.

In regards to the relationship of monitoring to attributional style Audrain, Schwartz, Lerman, Hughes, Peshkin & Biesecker (1997) report from a study of 256 women with at least one first order relative with either breast or ovarian cancer. Women with higher
levels of distress were more likely to be less optimistic and higher in Monitoring. Monitoring may be a strategy with functional value for individuals in coping with cancer who are also high in self efficacy, however the distress is highest for Monitors that are low in optimism and self efficacy (neurotic tendencies). High Monitors who score low on a measure of avoidance eventually show a decrease in anxiety as they have more opportunity to fully process the threat and adapt to it with effective coping and problem solving strategies. The high Monitors who are also high on measures of avoidance are the individuals who fail to de-sensitize to the threat, fail to adapt, and consequently maintain elevated levels of sustained arousal and anxiety (Miller 1996).

Monitoring has been found to be a productive strategy when the situation is perceived as being controllable, but unproductive when perceived as being uncontrollable, as with the genetic component of cancer. How the demands of the situation are perceived in terms of whether the threat is encoded as being of a controllable or uncontrollable nature is a contributing factor in regards to outcome. Monitoring may also be a useful coping strategy when the information being monitored can be used to adaptively change the person’s health protective behaviours, ie. the information translates easily into protective behaviour change strategies. Influencing how the situation and information is encoded is the predispositional nature of the person in terms of their tendency towards optimism or pessimism, helplessness or mastery orientation; their emotions such as calmness or anxiety and depression; and the problem solving strategies and abilities that are available or accessed by the person (Miller 1996).

Table 15. Comparison of factors interacting with Monitoring/Blunting and anxiety.

<table>
<thead>
<tr>
<th>Coping style</th>
<th>Attributional style</th>
<th>Self efficacy</th>
<th>Avoidance</th>
<th>Situation Percept.</th>
<th>Anxiety outcome</th>
</tr>
</thead>
<tbody>
<tr>
<td>Monitor</td>
<td>Pessimistic</td>
<td>Low</td>
<td>High</td>
<td>uncontrollable</td>
<td>On going</td>
</tr>
<tr>
<td>Monitor</td>
<td>Optimistic</td>
<td>High</td>
<td>Low</td>
<td>controllable</td>
<td>Short term</td>
</tr>
<tr>
<td>Blunter</td>
<td>Optimistic</td>
<td>High</td>
<td>High</td>
<td>controllable</td>
<td>Short term</td>
</tr>
</tbody>
</table>
In summary, Monitoring is likely to produce extremes of sustained levels of anxiety and distress when the situation and threatening information is perceived by the person as being unchangeable or hopeless, or when in fact the situation refers to a long term, intensely threatening and uncontrollable threat, such as a high genetic risk for cancer; and when the high monitoring characteristic is joined by a generally pessimistic attributional style, and the use of high avoidance strategies (Miller 1996).

Phipps & Zinn (1986) studied a sample of pregnant women who were to undergo a diagnostic procedure referred to as amniocentesis, and found that those who scored high on Monitoring experienced greater mood disturbance before and during the procedure than did women who scored high on Blunting. Miller et al (1983) report a study examining children’s reactions to aversive dental treatments. Eighty two children between the ages of 7-12 were measured on the Miller Behavioural Style Scale (MBSS) and independently observed during the dental procedures for signs of anxiety and distress. Those subjects who were measured as high Monitors on the MBSS reported more anxiety than Blunters and were rated as being more anxious, especially when they also engaged in avoidance. Miller, Siejak, Schroder, Lerman, Hernandez & Helm (1997) studied the responses of 101 women with human papillomarvirus-related precancerous cervical dyslpasia who were undergoing long term medical follow up and management. In the same study, they also examined the responses of 75 HIV infected gay men. For both samples, those who scored on the MBSS as high Monitors experienced higher levels of disease-related intrusive ideation which lead to avoidant ideation for the purposes of preventing panic. The efforts to avoid these obtrusive disturbing thoughts were found to be ineffective, as these people required increasingly defensive strategies to cope, such as denial and mental and behavioural disengagement. Miller et al (1983) explored the interacting effects of personal dispositions measured by the MBSS, and situational conditions on the stress response of 40 gynecologic patients who were about to undergo a coloscopy for diagnostic purposes. They found that on arrival at the hospital, those assessed as Monitors were more depressed, hostile and tense than were Blunters. In addition, results showed that Monitors took more time to decrease their levels of anxiety.
and tension after the procedure than did Blunters. In a study of 239 women with a family history of breast cancer, Lerman, Sands, Balshem, Lustbader, Goldstein, James & Engstrom (1993) found high monitors to be more distressed than blunters over time. With a similar population, 103 women who had a first order relative with ovarian cancer, Schwartz et al (1995) found that high monitors perceived themselves to be at higher risk than did Blunters, regardless of their true risk. This higher perception of risk was associated with heightened levels of intrusive ideation, and therefore with higher levels of anxiety and distress.

It is suggested here that although Monitoring has been demonstrated to operate as an independent trait from neuroticism, the combination of high Monitoring with neurotic tendencies such as high avoidance and pessimism is just the combination which would have seen an unknown amount of Grossarth-Maticzek’s subjects respond to the interview with excessive and sustained cancer and/or CHD anxiety. The type of information that may have been either overtly or covertly conveyed to the subject (i.e. elevated cancer or CHD risk) is the type of information and situation which Miller and colleagues have extensively studied. From their research, it is safe to predict that certain types of people (i.e. pessimistic neurotics who are also high Monitors) will predictably respond with acute anxiety on perceiving that they were in an elevated risk group for serious illnesses which are life threatening. Further more, this combination of personality characteristics and risk information has been demonstrated to lead to chronic sustained levels of anxiety in comparison to people who receive the same information but who do not share this profile. This results from the tendency of a subset of high Monitors to engage in high avoidance responses after the initial risk learning situation and an ongoing vigilance towards somatic and social cues for ill-health cognitions.

Within the C-SHIP model (Miller et al 1998), individualized illness representations are formed as a result of perceiving, encoding, and integrating information in available memory structures. These illness representations, or schemas include beliefs about:

- the health threat (‘am I really at risk because this expert says so?’)
• its cause (the origin of the health threat, eg. ‘I am vulnerable to cancer because of my personality type/family history of cancer’)
• consequence (minor v’s life threatening, e.g. ‘cancer is life threatening and I may die from it’)
• time line (ie. whether the illness is acute, chronic or cyclic)
• controllability (ie. whether treatment is available, e.g. ‘there is surgery and chemotherapy available for most cancers’).

As stated above, involved in the person’s response to risk information and illness representations are:-

• outcome expectations (eg. the belief that cancer is a likely outcome and will be severe),
• self efficacy expectancies (eg. confidence that one can manage the required lifestyle changes or cope with treatment), and
• treatment expectancies (eg. believing the medical treatments are effective against cancer).

These illness representations reflect the person’s individual view of cancer/CHD threat and are not necessarily medically accurate perceptions. Within the C-SHIP model, affects are viewed as being integral parts of the illness representations. Cancer, as an illness representation, is viewed as being an emotionally laden schema as evidenced by the finding that it is the most feared condition in our culture (Berman & Wandersman 1990), and that patients report increases in levels of anxiety, depression and anger when diagnosed with cancer (Miller et al 1998). Facing one’s vulnerability to cancer, perhaps by receiving risk information news, often elicits a ‘signature affective response’ to threatening events (Miller et al 1998). This can involve intrusive ideation about the source of the stress, mental replaying the health professional’s feedback about risk, and intense psychological distress when exposed to symbolic reminders of the event.

Whether a person responds with the signature affective response described above to risk information is predicted within the C-SHIP model by the individual’s interpretation of the
risk information and how this combines with situational factors (Miller et al 1998). Risk information which confirms expectations by matching prior health constructs, beliefs, goals and values has a greater chance of being integrated into the existing schemas. As such, the risk information is more likely to be internalized if it corresponds with pre-existing beliefs about vulnerability, the desire for and valuing of positive health, and beliefs around self efficacy, or lack thereof. For example, a person who typically has a pessimistic expectancy style is likely to focus on the negative interpretation of feedback received- they are likely to readily activate additional prior information which reinforces and sustains the pessimistic expectation and alarm (Miller et al 1998). Such beliefs may include the notion that ‘I am going to get cancer which has grown undetected and die early from it after undergoing painful, disfiguring and ineffective treatments’. Expected affective correlates to these illness representations and perception of risk are initially alarm, followed by anxiety and depression.

Contrast the above response with a person who has an optimistic outcome expectancy, accessing a different pattern of cognitive-affective schemas. Such a person may not become as alarmed as a result of remembering friends who survived cancer, or stories of people who are reported as having developed more rich and fuller lives as a result of life threatening conditions. The information may also elicit schemas about preventative lifestyles strategies such as exercise and a low fat diet, or may elicit a characteristic habit of responding to threats with stoic determination. The person can still be expected to experience some distress, but it would be less compared to the person who responds with pessimistic illness representations.

**RISP model**
The Risk Information Seeking and Processing (RISP) model has been developed by Griffin, Dunwoody & Neuwirth (cited in Griffin, Neuwirth, Giese & Dunwoody 2002) as a combination of Eagly & Chaikens heuristic-systematic model (HSM) of information processing and Ajzen’s theory of planned behaviour (both cited in Griffin et al 2002). The RISP model accounts for the observation that people respond to risk information differently, with some people processing information about risks intensively, some
superficially, and others not at all. The HSM component of the RISP model assists with understanding how people come to seek, attend to and process risk information.

The RISP model suggests that a strong motivation for information processing is the person’s desire for accurate and sufficient information about the risk. The ‘sufficiency principle’ suggests that people seek a ‘sufficient’ degree of confidence in the information and will exert whatever effort is required in order to attain their processing goal. If the processing goals are set ‘high’, systematic processing is more likely to be engaged in. If processing goals are set ‘low’, heuristic processing is more likely to be utilized. These two styles of processing may be engaged in simultaneously, until the information has been processed to a point which is regarded as sufficient - the goal has been met and the processing ceases. Griffin et al (2002) state that research has demonstrated that information sufficiency is related to the affective response to risk information.

When the person has ‘low’ processing needs, they are likely to be satisfied with heuristic processing. This is described by Griffin et al (2002) as a limited method of information processing as it requires fewer cognitive resources and less effort than do more systematic processing methods. In using heuristic processing, people employ a range of superficial cues as cognitive short cuts to satisfying their processing needs. People described as high in Monitoring (Miller 1996) may rely more on heuristic information processing in scanning their environment for cues of threat, and thereby arrive at a risk perception and anxiety quicker than do those low in Monitoring. Cues such as the following may all be used by a person to arrive at a decision about perception of risk:- the length of the message, with the more arguments in favour of an assertion, the more believable it must be; the perception of a trusted spokesperson or creditable authority to deliver the message; the use of statistical data to support the assertion. The perception of credibility of Grossarth-Matticek and his interviewers has already been discussed. It is also possible that the interviewers in the Heidelberg study used outcome statistics gained in the Yugoslav study when providing all possible information to subjects in the full interview condition.
Contrasted with the heuristic processing style is the systematic information processing style. In this approach, a more comprehensive effort is made to analyze and understand information with a more extensive evaluation of the available information. Griffin et al (2002) state that this style of processing tends to lead to more permanent conclusions than does heuristic processing which tends to be more volatile in the conclusions reached.

Where there is a ‘gap’ between what a person knows about a risk and what they feel they need to know, motivation for more cognitive effort via systematic processing is generated. Such extra cognitive efforts may include evaluating the message more critically, thinking about the message, and integrating what message-based information with what one already knows. Systematic processing also depends upon existing knowledge structures (eg. education), the perceived ability to obtain relevant information, as well as the perceived credibility and usefulness of available information (Griffin et al 2002).

The cognitive models presented here inform researchers about the processes involved in developing a risk perception for populations that are viewed as having increased chances of cancer/CHD as a consequence of hereditary factors. Inferences can be made from how people with a genetic risk for cancer/CHD respond to this news of being in an elevated risk group to how Grossarth-Maticek’s subjects may have responded to learning or perceiving that they were in an elevated risk group.

First order relatives as a natural risk group:
First order relatives of people with a condition known to have a hereditary component are a naturally occurring group who have a heightened risk perception. As such, they can be viewed as a similar group to Grossarth-Maticek’s subjects who learnt or perceived that they were at risk of cancer/CHD. One of the difficulties for people in a hereditary risk group is that they are attempting to cope with the threat of the condition, rather than with the disease itself- as such, it is more abstract and thereby difficult to control. Considering the large effects of an actual cancer diagnosis, it could be anticipated that being in an
elevated risk group would also increase the level of cancer anxiety, if not to the same extent as an actual diagnosis.

It is known that certain cancers have a strong genetic component, such as breast cancer, colorectal (rectum or colon) cancer, melanoma, leukemia, ovarian cancer, and prostrate cancer (Scientific September American 1996: Fact Sheet). Due to media attention regarding the issue of breast cancer, an enhanced public and professional awareness has developed concerning the familial nature of the disease. Lerman, Lustbader, Rimer, Daly, Miller, Sands & Balshem (1995) state that other than age, the most important risk factor for the development of breast cancer is having a first degree relative with the condition. While the lifetime risk of breast cancer for such women may be as high as 50%, the vast majority of women with a family history of breast cancer have a significantly lower risk. Lerman et al (1995 p286) cite the following figures: the lifetime risk for a 40 year old woman with a family history of the disease ranges from 13.6% to 30.4 %, depending on the presence of other risk factors.

Stefanek & Wilcox (1991) report that the degree of risk varies according to factors such as:- the menopausal status of the effected relative (i.e post or pre menopausal); the laterality of the disease (i.e unilateral or bilateral), and the number of generations affected. Newman, Austin, Lee & King (1988) report that approximately 4% of breast cancer is thought to be due to hereditary factors, while Solomon (1990) reports that around 8% of colorectal cancer is due to hereditary factors. Shoda, Mischel, Miller, Diefenbach, Daly & Engstrom (1998) state that it is estimated that genetic factors contributed to around 25% of the new breast cancer cases in women under the age of 30 for 1996 in the U.S.

“Mutations in BRCA 1/2, for example, appear to be responsible for the disease in 45 % of families with multiple cases of breast cancer only, and up to 90% of families affected by both breast cancer and ovarian cancer” (Shoda et al 1998 p. 4).
As such, people who have first order relatives with these types of cancers in particular are considered to be in a naturally occurring heightened risk group for these cancers. Many such people would have been made aware of their elevated risk status by health professionals on learning of their relative’s cancer- these are people who, by and large, perceive themselves to be at larger than average risk of cancer. Kash, Holland, Halper & Miller (1992 p.25) state that “many high risk women describe themselves as “walking time bombs” awaiting the inevitable cancer to develop”.

**Psychological consequences of being in an ‘at-risk’ group:**
The following research results pertain to people who were currently well and asymptomatic but who were assessed as being at-risk of serious medical conditions. Lerman, Rimer & Engstrom (1991) state that common psychological reactions to genetic cancer risk information are denial, low self esteem, anxiety and guilt- these responses have also been noted in carriers of susceptibility to other genetic conditions such as Huntington’s chorea, Tay-Sach’s disease, and cystic fibrosis (Lerman et al 1991). Josten, Evans, & Love (1985) state that finding oneself in a high risk group of cancer for genetic reasons creates in people an overwhelming sense of vulnerability and heightened levels of cancer worry. Lerman et al (1995 p286) claim “studies have shown that a majority of women with a family history of breast cancer have exaggerated perceptions of their own risk and experience excessive anxiety”. A finding which surprised and disappointed these researchers was the result demonstrating the resilience of these threat cognitions. Their intervention of specialized breast cancer risk counselling with 200 women with familial patterns of breast cancer did show some overall effects in terms of improved risk comprehension of the treatment group as compared to the controls. However, three months later, around 75% of the treatment group still viewed themselves as having a greater than 50% life long chance of contracting breast cancer, while according to an objective risk model, their average risk level was only around 14%. Valdimarstdotter, Boubjerg, Kash, Holland, Osborne & Miller (1995) report that in a study comparing 27 women with a family history of breast cancer with a control group, women in the former group displayed signs of psychological distress both before a mammography, as well as one month after the notification of normal results.
Josten et al (1985) found evidence that 75% of people attending a genetic counselling service who had a first order relative with cancer perceive their level of risk to be higher than what it actually is. Lerman, Lustbader, Rimer, Daly, Miller, Sands & Balshem (1995) report that two thirds of women who have a family history of breast cancer in their sample extremely over estimated their lifetime risk of developing the condition. Lerman & Schwartz (1993) report that women who seek counseling for genetic risk of breast cancer frequently experience a range of psychological problems such as obtrusive thoughts about the condition, impairment in daily functioning due to cancer anxieties, and sleep disturbances- the level of mood disturbance is similar to that displayed by breast cancer patients. Brain, Norman, Gray, & Mansel (1999) confirm that women with a family history of breast cancer do experience a range of psychological difficulties including a heightened sense of susceptibility and resultant anxiety. These researchers studied the levels of state and trait anxiety amongst 883 women who were designated to be at risk of developing breast cancer as a result of having a first order relative with the disease. Brain et al (1999) found there to be moderately significant correlations between breast cancer worry and trait anxiety ($r=.40$) and breast cancer worry and state anxiety ($r=.38$), supporting the notion that general anxiety levels as a personality characteristic are associated with cancer anxiety levels as measured by the State-Trait Anxiety Inventory. The women who were more susceptible to state anxiety were also more generally anxious, as measured by higher trait anxiety scores. Brain et al (1999) also found positive correlations between perceived level of risk for breast cancer and levels of breast cancer worries- women who placed themselves in a high risk group for cancer also experienced more breast cancer anxiety. Rothemund, Paepke & Flor (2001) found that female subjects with family histories of breast cancer were more distressed and anxious compared to control subjects, however, differing to Brain et al (1999), they concluded that trait anxiety was not a predictor of breast cancer worry.

Shoda et al (1999) demonstrated that the psychological impact of genetic testing for breast cancer is even more pronounced for women that have no previous exposure to cancer, as they are less psychologically prepared to deal with threatening feedback-
women who have had some family exposure to breast cancer have already begun their preparation for the results.

McCaul, Branstetter, O'Donnell, Jacobson & Quinlan (1998) studied the levels of cancer anxiety amongst a sample of 65 women with family histories of breast cancer as compared to a sample of 70 women who had no family history of breast cancer. These researchers suggest that most other studies of cancer anxiety in women collect their data in an unusual, clinical context in which one may expect a heightened level of cancer anxiety to occur. Proposing a superior methodology, McCaul et al (1998) had women in their study self monitor their thoughts and feelings over time in a home setting, rather than a one off data collection in a clinical setting. They found that women with a family history of breast cancer reported statistically significant more frequent cancer worry than did women with no such family history, and they reported more distress in relation to how much the cancer worry affected their moods. McCaul et al (1998) also report that there were found to be no differences in amount of breast cancer worry between those with first order relatives with the disease and those with other than first order relatives - to have relatives with the disease, regardless of the degree of relation, was more important in generating breast cancer worry than was the closeness of the relationship per se. Worry levels were viewed as being at least partly the result of the study itself as a cue to worry - these levels did decrease over time for the sample overall (data was again taken at a 1 year follow up), but women with a family history of breast cancer were less likely to report a decrease in worry over time than were women with no such history.

“Indeed, the family history differences observed in this study suggest that women with a family history may be chronically more worried about breast cancer than women without such a history” (McCaul et al 1998. p577).

Lerman et al (1993) found that 30% of women with first order relatives with breast cancer reported worrying about their prospects of contracting the illness so much that it interfered with their daily functioning. Kash, Holland, Halper and Miller (1992) found that 25% of women in this category displayed so much worry about breast cancer so as to
merit the use of counselling to help them cope. Kash et al (1992) report that in their sample of 217 women with a family risk factor for breast cancer, women who perceived their risk as high with little self efficacy in regards to the risk, demonstrated levels of psychological distress similar to survivors of Hodgkins disease and leukemia. Lerman, Trock, Rimer, Jepson, Brody & Boyce (1991) report that from a study of 308 women, subjects with suspicious mammograms (which via further diagnostic tests ruled out breast cancer) were found to exhibit breast cancer worries to the extent that it interfered with their moods and daily functioning three months after the mammogram. McCaul et al (1998) also demonstrated that this cancer anxiety maintains itself over time.

From their study of over fifteen hundred 48-50 year old Finnish women, Absetz et al (2000) report that the majority of women in their study who had family members with breast cancer were well aware that hereditary was a risk factor for the condition. This knowledge, however, was not sufficient on its own to produce cancer risk perception- it had to be combined with experience of having someone close with breast cancer to produce a sense of risk- perception of risk increased with the amount of vicarious experiences one has with breast cancer- the closer was the experience (eg. first order family or close friend), the higher was the perceived vulnerability. They suggest that risk perception is only partly due to the knowledge that breast cancer can be inherited, but it is also due to having experience with people (family or friends) who have breast cancer. The lowest perception of risk was displayed by women who had no vicarious experience with breast cancer combined with an optimistic coping style.

Extending the findings beyond breast cancer anxiety Miller, Diefenbach, Kraus, Watkins-Bruner, Hanks & Engstrom (2001) report that recent findings from a Swedish study showed that 60% of sons of men with prostrate cancer expressed worry about the disease as they viewed themselves as being at risk. In a study of 56 first order relatives of men with prostrate cancer and 100 control subjects, Miller et al (2001) report that the first order relatives display a distinct psychological profile which includes higher perceptions of vulnerability and an over estimate of their risk of prostrate cancer when compared with controls. In a sample comparing 56 men with and 100 men without first order relatives
with prostrate cancer, Miller et al (2001) found that the former group on average perceived their lifetime risk at 48%, where in their actual risk ranged from 13-25%. In addition to seeing themselves as more vulnerable, these men saw prostrate cancer as being less preventable compared to controls. Kinney, Choi, DeVellis, Kobetz, Millikan & Sander (2000) from a sample of 95 first degree relatives of colorectal cancer patients found that the vast majority of them (68%) believed that their relative risk of the condition was increased. Wiggins, Whyte, Huggins, Adam, Theilman, Block, Sheps, Schechter & Hayden (1992) studied the psychological sequelae for 135 people attending genetic screening for susceptibility for Huntington’s disease- all of these subjects had a confirmed family history of the condition so already had some degree of risk perception. After genetic testing, subjects obtained one of three forms of news:- increased risk; decreased risk, or no change in risk status. Subjects who were confirmed to have a genetic predisposition for the condition (carriers) were initially more depressed and exhibited more psychiatric symptomology in the 7-10 days following testing compared to the non carriers. The measures of distress had all declined significantly for both carriers and non carriers 12 months later. However, with subjects who did not receive genetic testing (due to withdrawal of their consent, or because testing was not informative), their measures of distress were higher than both carriers and non carriers- presumably, they viewed themselves as being at risk due to familial factors, however were not able to fully cope with this risk status (as were the carriers) because there was no definitive assessment of vulnerability. The carriers had been able to mobilize their coping strategies, however the non-tested subjects were not, and as such remained distressed by the possibility that they could be vulnerable. As stated, at the 12 month follow-up, carriers were found to have the same level of psychological well being as the non-carriers. The authors point out that this result may be due to the fact that participants in the study received counselling and psychological support in the 12 months following the testing. As such, it can be seen from this study that the initial response to learning of an increased risk status (being a carrier) is a deterioration in psychological well being. For ethical reasons, these researchers were not able to assess the psychological consequences 12 months after receiving the news without the provision of counselling and support.
Horowitz, Hulley, Alvarez, Billings, Benfari, Blair, Borhani & Simon (1980) report that men who were notified of their elevated risk status for CHD in the Multiple Risk Factor Intervention Trial experienced more disturbing obtrusive thoughts and distress than men who received no such notification- being notified of such a risk status was a stressful life event. Similarly, Reelick, de Haes & Sherman (1984) found that women who were notified of abnormal Pap smear tests following mass screening for cervical cancer showed significant psychological distress in the form of tension and depression, even though the results were not diagnoses of cancer. With a similar population of 395 women, Miller, Siejak, Schroeder, Lerman, Hernandez and Helm (1997) report that a full two thirds of the subjects reported that they were afraid that they had cancer. Bloch, Adam, Wiggins, Huggin & Hyden (1992) report that people undergoing genetic screening for Huntington’s Disease typically react to increased confirmation of risk with emotional difficulties such as depression and anxiety. All people who obtain this news, according to Bloch et al (1992) undergo a difficult immediate period of adjustment, however some experience a prolonged period of significant emotional distress and some become suicidal. Kessler (1987) reports that people who learn that they are at risk of Huntington’s Disease are also at risk of psychiatric dysfunctions and major affective disorders, and as a result are more likely to be hospitalized for psychiatric reasons than individuals in the general population. Suicide also constitutes the third or fourth most common cause of mortality in the group at-risk of Huntington’s Disease.

Another group of people in society who learn of having a health risk status are those whose work has brought them into contact with potential carcinogenic agents. Lerman et al (1991) report that denial and minimization has been observed in workers who were at risk of work related asbestos-related mesothelioma, whereas other studies have found workers responding with high levels of distress when exposed to vinyl chloride. They conclude that average levels of psychological distress amongst at risk workers is not elevated, however a small minority of workers are likely to suffer from ‘excessive distress and dysfunction’.
As demonstrated here, the short term consequence of learning of an increased risk status is alarm and increases in depression and psychiatric symptomology. These results confirm the contention that elevated anxiety levels about cancer can occur or be generated without a diagnosis being made of the worried individual - a sense of threat and alarm can be created simply by virtue of being in an at-risk group.

Risk cognitions are obviously tenacious and exaggerations of risk is widespread, however rather than the bulk of the population being predominantly pessimistic in regards to risk, the pessimistic view appears to limited to a subset of people as suggested by Lerman et al (1991). Absetz, Aro, Rehnberg & Sutton (2000) report the presence of a widespread ‘optimistic bias’ which is a tendency to see one’s own risk in more favourable terms than the risk to other people. For most people, this trend even holds also for events such as being mugged or getting divorced. Optimism in risk perception serves the function of decreasing anxiety and as such can be viewed as an adaptive response which the risk perception literature suggests most of the population utilizes in order to cope (Klein 2002).

When the optimistic bias does not operate, resulting in a greater perception of risk, the person may be utilizing the ‘availability heuristic’ as suggested in the RISP model (Griffin et al 2002). Facione (2002) suggests that this cognitive shortcut may operate when, for example a woman may over-estimate the likelihood of obtaining breast cancer as a result of having friends and family members who have experienced the condition. In her community based sample of 770 women aged between 19-99, Facione (2002) found that women with family members who had experienced breast cancer did report a higher perceived risk of the condition, whereas those with friends who had experienced the condition did not perceive themselves as being at high risk. As such, the optimistic bias is less likely to operate for women with a family history of breast cancer - these women are likely to view themselves as being at risk of the condition. Lipkus, Biradavolu, Fenn, Keller & Rimer (2001), on the other hand, report that while people in general do show an optimistic bias, women consistently and ‘dramatically’ overestimate their risk of getting breast cancer.
As such, an optimistic bias will be engaged for many people as a coping strategy to ward off anxiety; however, some people can be expected to respond with ‘signature’ pessimistic expectations and a heightened perception of risk as a result of receiving such news. It is these people who one can expect to be more alarmed on receipt of risk information, and maintain a higher level of anxiety and depression beyond the initial response.

It is also possible that not all life threatening conditions are equally feared even when one is located in a high risk group. Mossey (1981) reports from a study of 230 men participating in a hypertensions compliance study in which the participants were assessed for their risk of hypertension. A range of communications or patient labels were given to the men, including: - a) ‘you have high blood pressure’ associated with newly identified hypertension among those unaware of it; b) ‘you need to take drugs to control your blood pressure’, associated with having drug treatment recommended, and c) ‘high blood pressure is serious- left untreated, it is a risk factor for other conditions (eg. heart attack, strokes, aneurism)- conscientious compliance with prescribed treatment is recommended’. Psychosocial functioning was assessed before and 6 and 12 months after the screening and risk communication, including measures of marital adjustment, perceptions of self and self perceptions of health. Mossey (1981) reports that the data does not support the presence of a strong, deleterious effect due to patient labeling and increased risk perception. There was noted, however, a trend with patients exposed to message b (above) showing a decrement in psychosocial functioning in most measures 6 months later, and to a lesser extent 12 months later. As such, unlike cancer risk, the risk of CHD posed by high blood pressure was not found to produce high levels of long lasting anxiety. Combine this result with the claim by Wilcox et al (1999) that only 34% of women knew that CHD is the leading cause of death among women over 65 years of age; and the fact that cancer is the most feared condition in our society, one may conclude that the impact of being at risk for CHD is not as large as the impact for being at risk of cancer.
As a limitation to the broad applicability of research results concerning the cognitive structures of risk, Vaughan (1993) states that social and cultural factors may limit the relevance of results across different groups. Ethnicity and socio-economic status have been found to influence perceptions of risk and produce considerable variability. Vaughan (1993) suggests that further investigation into the role of cultural and social factors in risk perception is required, as there is some question as to how applicable the current theories are to diverse populations. This is an important consideration in that Grossarth-Maticek’s samples were taken from particular ethnic groups, i.e. Yugoslavia and Germany. It is possible that within these ethnic and age groups, unknown health beliefs, e.g. via traditional folk medicines, could have operated which added to the outcomes reported via the process hypothesized here.

In summary, the question of interest is ‘What happens to people who may be characterised as neurotic when they perceive that they are in an elevated risk group?’ It can be concluded from the research evidence provided in this chapter that most people respond to risk of cancer with perceptions of risk and associated psychological distress. Some people, the least emotionally stable, will maintain this level of distress beyond the initial alarm phase. The ability of CHD risk to promote and maintain a perception of risk and associated anxiety is less clear (Mossey 1981).

The effects on ‘neurotic’ (Types 1 & 2) probands in Grossarth-Maticek’s study of perceiving that they are in an elevated risk group for cancer can be expected to go beyond the ‘normal’ rise and fall of acute cancer worry, and extend into being a chronic level of cancer worry. It is conjectured that Grossarth-Maticek’s subjects in the full interview condition were told of their risk status on request, or may have perceived this as a result of an interviewer effect. The somatic cues to ill-health are likely to be a regular feature of their lives, as researchers have demonstrated more generic bodily symptoms in people assessed as neurotic (high neurotics display two to three times as many somatic complaints compared to non neurotics– Sarafino 1994), and Grossarth-Maticek’s subjects were in an age range where such bodily symptoms are increasingly the norm as a factor of age.
Finally, add to this picture that at the 7 year mark, Grossarth-Maticek's subjects were contacted by his researchers, primarily to ascertain if they were:-
i) alive or had they died from cancer or CHD, and
ii) if alive, had they experienced cancer or CHD?

This could be expected to act as a very powerful reminder of their risk status, and although probably not needing this experience in order to feel anxious about the prospect of obtaining cancer or CHD, it could be expected to act as a very powerful evocation of more intense cancer/CHD worry. This was again repeated at the 13 year mark, and at the 15 year mark, with subjects presumably knowing that they were to be contacted at these intervals to see if they were alive. McCaul et al (1998) found that women participating in their study of breast cancer anxiety reported that simply being a subject in the study acted as the most frequent prompt to thinking about cancer- much of the anxiety measured was cued by the study itself. Is there any reason to think that this factor would not have operated in Grossarth-Maticek’s study as well, which was known by the subjects to be prospective with an interest as to whether they would live or die?

In conclusion, evidence has been presented in support of experimenter effects and self fulfilling prophecies. Evidence has also been presented that finding oneself in an at-risk population leads to perceptions of vulnerability to these conditions, as well as to increased negative affect- being told that you are at risk by a credible authority leads most people to view themselves as being at risk and to be alarmed by this. It can be seen that conditions like cancer evoke a wide range of strong negative affects. While most people are able to place the risk in perspective, some less emotionally stable people can respond with extreme alarm and maintain this level of alarm over a long period of time. Evidence has been presented in earlier chapters that having a pessimistic attributional style does make one more prone to learned helplessness depression and subsequent increases in stress hormones- internalizing a risk perception and viewing oneself as a likely candidate for cancer or CHD is a pessimistic expectation. And finally, excessive secretions of stress hormones have been demonstrated to damage the functioning of the
immune system as well as damage the part of the brain which regulates the release of the stress hormones (Khalsa 1997).

All of these findings combined suggest that a possible explanation for Grossarth-Maticek’s reports is that psychologically vulnerable people were placed in a situation (the full interview condition) whereby they learnt of or perceived that they were at risk of life threatening conditions. It is proposed that these people, as per their ‘signature’ patterns of negative affect, spent the subsequent years worrying about the prospect of contracting cancer or CHD, became chronically distressed about this prospect and were more likely to experience learned helplessness depression and elevated anxiety. One proposed consequence of this psychological cascade is that excesses of cortico-steroids and other stress hormones would be released into their blood supply which, over a long period of time, would reduce the ability of their immune systems to resist the feared conditions. This effect would not occur when the IRI scores were obtained in the self administered manner, as there was no opportunity for an experimenter effect to occur.

Dunn (2001) reports studies which demonstrate that the best predictors of long term psychological adjustment in people with cancer is the linking of bad news (ie. positive cancer results) with treatment options. Patients who are informed that they have cancer, but whose physicians make an effort to detail the treatment approaches available demonstrate better psychological adjustment 12 months post diagnosis. One of the main differences between Grossarth-Maticek’s subjects who participated in his experimental intervention (receiving Creative-Novation Behaviour Therapy) and those who did not is that the latter group may have been left with a perception of vulnerability to cancer and CHD with no treatment options made available. This would make sense as no researcher would be expected to have a detailed discussion with the subjects about treatment options for a condition which had not yet arisen. On the other hand, Grossarth-Maticek’s subjects who underwent his therapy were given, by virtue of receiving the treatment, the belief that they were able to do something about their vulnerability to the predicted condition. As such, they could be expected to have a greater sense of control over the prospects of obtaining cancer or CHD, and therefore to have suffered less of the effect to which Dunn
(2001) refers. More anxiety accrues from having a life threatening condition (or perceiving oneself to be at risk of such a condition) and having little sense of the options available for treating (or preventing) the condition. Treatment or prevention options obviously provide one with a sense of control over a health issue which is usually feared as being extreme and somewhat uncontrollable.

Fredrikson, Furst, Lekander, Rotstein & Blomgren (1993), studied the effects of stress on measures of immune functioning in 27 patients who were currently free from signs of cancer but who were undergoing adjuvant chemotherapy for breast cancer. They considered it important to distinguish between trait anxiety (people who experience anxiety as a general condition in their lives, typical of neurotics) and state anxiety (a situation specific episode of anxiety), utilizing the Speilerberger State-Trait Anxiety Inventory. The stressful events acting as the independent variable was the imminent experience of adjuvant chemotherapy, with blood measures being taken at home two days prior to the therapy, and again at the hospital prior to the therapy. They found that state anxiety did not relate to changes in the immune system measures of decreases in helper/inducer T-cells- there were found to be no changes in immune functioning that were related to the specific stressful event, either in measures taken at home or at the hospital. They concluded that state dependent anxiety did not impair the patient’s immune function. However, they did find that patients who were assessed to have high *trait anxiety* displayed a decrease in helper/inducer T-cells in response to the stressful event of anticipating the commencement of the chemotherapy while in the hospital, and also when measured at home two days prior to the therapy. Their conclusion was that, “trait anxiety might make individuals exposed to a stressor more vulnerable to immune change, while such changes may not be observed at a basal non stressful period.” Fredrikson et al (1993p87)

This finding is construed here as supportive of the notion that Grossarth-Maticek’s subjects who were high in trait anxiety (i.e high Type 1 and Type 2 scorers) were more vulnerable to immune compromise as a consequence of the stress associated with
perceiving or learning that they were prone to cancer or CHD compared to health prone subjects.

Khalsa (1997) has described the immediate neurological and endocrine responses to stressful events in terms of excessive secretions of a range of stress hormones, including the brain damaging cortisol. With the limbic system becoming ‘overloaded’ and ‘washed’ by excessive secretions of toxic cortisol, the stage is set for the degenerative cascade of neurological damage (if it had not already begun simply due to a lifetime of neurotic stress and trait anxiety), leading to even more excessive secretion of cortisol. It is known that cortisol not only kills brain cells via the production of free-radicals, but it damages the functioning of the immune system, rendering it less able to control the growth of cancer cells.

The anxiety created by learning that one is in an elevated risk group for cancer/CHD may become part of the ‘back drop’ of stable anxiety (although trait anxiety would have already existed in neurotic people), from which many subsequent life experiences and events are perceived as being even more stressful and taxing. Similar to the degenerative cascade effect of the neurological-endocrine damage caused by excessive cortisol, a psychological cascade effect is created whereby moderate events are experienced as very stressful, and excessive amounts of immunosuppressive cortisol continues to be released into the blood supply. In addition, concrete cognitive cues to cancer/CHD worry would have been maintained or increased with age, reactivating the initial cancer worry experienced as a result of the interview.

This type of psychological cascade effect was described by Dykema, Bergbower & Peterson (1995) as an alternative to the Diathesis-Stress theory of illness- the actual perception of a stressor, and the perception of impact of the stressor, is also a function of the predisposing personality characteristics of the individual- pessimistic people can easily ‘make mountains out of molehills, and molehills out of nothing’, thereby ‘cascading’ their distress. This view is consistent with Monroe et al's (1991 p411)
suggestion that the nature of the diathesis will actually have an influence on the nature and course of a stressful life event.

“Most people are, at least in some part, the creators of the circumstances they endure...a diathesis influences the manner in which the person negotiates life’s course, and consequently the nature of the stressors to which he or she is exposed...stress is not a random process, but rather part of a developmental sequence systematically influenced by the diathesis. Whereas the construct of stress may still play an important role in the evolving scheme, it is generated to a considerable degree by the person’s behaviour, which in turn is likely to be influenced by the diathesis.”

As such, it is proposed here that although the stressful event of learning of an elevated cancer/CHD risk status in the course of the interview is not likely to have created an immediate and irreversible immunosuppressive effect (see Kemeny 1993; Fredrikson et al 1993), this risk status is likely to have interacted with the predisposing neuroticism and trait anxiety in a cascading manner to exacerbate an elevated level of ongoing, back ground stress. The situation may have created a ‘sleeper effect’ as described in an earlier section, in which the power of the risk information increased over time rather than decreased. This psychological cascade into heightened anxiety is at least a parallel to the physiological degenerative cascade as described by Khalsa (1997), however is more likely to be merely the psychological aspect of the same degenerative effect on the whole organism. Part of this cascading effect is the ongoing fear of cancer/CHD which is commonly experienced in our society; the sustained amount of internal and environmental cues leading to ongoing concerns about cancer/CHD, and the knowledge of being in an elevated risk group with an enhanced expectation of serious illness- the conditions are ripe for immunosuppression via chronic stress and, if the reports of PNI are correct, subsequent cancer or CHD from associated distress.

Research by Kubzansky, Kawachi & Spiro (1997) lend weight to this possibility. They report results from the 20 year follow up of 1,759 initially healthy men in a prospective
study—these results demonstrated that ‘worry’, as a cognitive component of anxiety bore a strong association with subsequent CHD. Worry was measured with a Worry Scale which segmented worry into five different domains of concern:- social conditions, health, finances, self definition, and aging. Kubzansky et al (1997) demonstrated that there were associations between the health and finances worry subscales and CHD. In particular,

“a one unit increase in the health worry score was associated with a modest increase in risk of angina pectoris (age adjusted RR=1.39; 95% CI:1.05 to 1.84), and with an increase in risk of sudden death (age adjusted RR=2.19; 95% CI:1.10 to 4.76)…. Thus, high levels of worry in specific domains may be a risk factor for CHD in older men, suggesting that non clinical levels of anxiety may be associated with CHD”. (Kubzansky et al 1998 p.51)

This result clearly suggests the health damaging effects of anxiety and worry about health concerns, even at a sub clinical level.

In summary, Grossarth-Maticek’s proband’s perception of risk status is likely to have not changed in the years following the interview experience - they would have received very little if any information to counter the effects of the perception of being cancer or CHD prone. Only Grossarth-Maticek’s experimental subjects who participated in the intervention study received information to counter the sense of proneness. These subjects underwent Grossarth-Maticek’s Creative Novation Behaviour Therapy, which would have been an ideal forum to reverse the perception of risk and bolstering the subject’s sense of control over the risk of cancer/CHD by addressing the relevant personality/behavioural characteristics- notably, these subjects experienced and died from cancer and CHD at significantly lower rates than did the control subjects.

For the control subjects, and for all other subjects in Grossarth-Maticek’s study, it is reasonable to expect that all that would have happened in the intervening years is that the concrete environmental cues would have continued in the form of health warnings for such behaviours as smoking; incidences and deaths by cancer of acquaintances, and
media attention relating to the incidence of cancer and CHD would have remained high, or indeed increased. Bianchi (1971) reports that fear of death, illness and injury reach a peak prevalence at the age of 60 years, well within the average age range for Grossarth-Maticek’s subjects. Subjects in McCaul et al’s (1998) study reported that there were many environmental cues to cancer anxiety, such as: the media (they reported 157 breast cancer thoughts during one week that were prompted by newspaper, magazine, radio and television coverage of cancer), just talking with others or observing others with cancer—this could be expected to be an increasingly common experience in the over 50 age group. According to Gutteling (1987), 94% of women in this age range knew someone who had experienced cancer, while 90% knew someone who had died of cancer. In addition to this, generic somatic cues would have increased in frequency as a result of increases in age; and finally, responding to enquiries from Grossarh-Maticek’s researchers about their current health status, together would all remind the subjects, in both sporadic and ongoing manners of their risk status and act to re- evoke their cancer/CHD worry.

In this context, the ‘sleeper effect’ begins to seem more plausible, as these social experiences increase with the age of the subjects, generating more cancer/CHD anxiety. Add to this set of possibilities the fact that the cancer and CHD prone subjects are defined as being neurotic, and therefore experience greater amounts of generalised anxiety, tension, depression, worry, etc. Chronic cancer and CHD anxiety can be hypothesized as one possible outcome for these subjects.

Synthesis:
The existing research based literature supports the following conclusions:-

- subjects in interviews and experiments are able to deduce the purpose of the study or interview as well as implications which it has for them via non deliberate, subtle non verbal cues, referred to as an interviewer/experimenter bias (Cosby 1981; Rosenthal 1994)- this creates the possibility of Grossarh-Maticek’s subjects inferring their risk status from the interview.
• Grossarth-Maticzek’s interviewers in the full interview condition were compelled by the requirements of the condition to provide as much information as possible to the subjects and to answer any questions asked (Grossarth-Maticzek, Eysenck & Barrett 1993) - this creates the possibility of interviewers actually informing subjects of their IRI scores and the meaning of them as per the rules of the condition. Grossarth-Maticzek did not respond to the current author’s queries as to whether this occurred or not, so this possibility cannot be accepted or excluded.

• Subjects who score high on Neuroticism are vulnerable to excessive anxiety and worry, as well as to pessimistic attributional styles. Introverted Neurotics are also more sensitive to subtle environmental cues and stimuli (Monte 1995).

• Cancer is the most feared condition in western culture (Berman & Wandersman 1990); cancer and CHD account for most deaths in western culture and are highly feared due to the perception of uncontrollability, aversive consequences, perception of high likelihood of death, and lack of knowledge about causes and warning signs (Brown et al 1992).

• Stressful life events, including ill health, have been associated with the onset of various cancers (Butow, Hiller, Thackway and Kricker 1997; Cooper & Faragher 1989).

• Learning of the risk of ill health is a stressful life event leading to increases in worry and anxiety. (Horowitz, Hulley, Alvarez, Billings, Benfari, Blair, Borhani & Simon 1980; Reelick, de Haes & Sherman 1984; Bloom & Monterossa 1982).

• Being in a naturally occurring cancer risk group produces a level of anxiety which often reaches a pathological level, interfering with daily functioning and mood, requiring mental health services (Lerman, Trock, Rimer, Jepson, Brody & Boyce 1991; Holland 1996; Elsesser, van Berkel, Sartory, Biermann-Gocke & Ohl 1994; Josten, Evans, & Love 1985).

• Excessive anxiety associated with a sense of helplessness and hopelessness leads to increases in levels of cortisol in the blood stream (Khalsa 1997).

• Excessive amounts of cortisol are associated with damage to neurons in the hippocampus which is responsible for ‘turning off’ the excessive secretions of cortisol, leading to increasing amounts of cortisol secretion. (McEwan et al 1993).
• Excessive amounts of cortisol damages the functioning of the immune system, making the organism more vulnerable to cancerous cells (Khalsa 1997).

• The power of expectations on health outcomes has been demonstrated by Phillips, Ruth & Wagner (1993) wherein belief in vulnerability to serious health problems according to year of birth was demonstrated to be linked to actual mortality via a self fulfilling prophecy in Chinese-Americans.

• Increases in worry about health issues has been associated with increases in CHD. (Kubzansky et al 1997).

• Having higher levels of background stress make one more sensitive to acute stressors (Gump et al 1999).

It is proposed here that a moderate psychological effect could have become exaggerated due to expectations and associated anxiety generated in Grossarth-Maticek’s full interview condition. As such, a moderate psychological effect may have been transformed into a more powerful effect as a result of the cancer/CHD anxiety created, and maintained by ongoing environmental cues and increasing somatic symptoms. In effect, it is being proposed that the result of finding oneself with an ‘at-risk’ status, and internalizing this status thereby creating a negative expectation, produced a level of subjective stress in the most psychologically vulnerable of Grossarth-Maticek’s probands such that the functioning of their immune systems was compromised over the years of the study resulting in a higher than expected death rates. This possibility will be the research focus of the current study.
THE ROLE OF PSYCHO-SOCIAL EXPERIENCE IN CHRONIC DISEASE

VOLUME II

pp. 225-435
Chapter 12

The Current Study

Testing an alternate explanation of Grossarth-Maticek’s results:
Medical research already supports the role which the traditional medical risk-factors play in the causation of cancers and CHD, and these are largely uncontroversial. The current study is investigating the role which a psycho-social factor may have played in the causation of cancer and CHD in the subjects of Grossarth-Maticek’s Yugoslavian and Heidelberg studies. The psycho-social factor of interest in this study is the perception of vulnerability to cancer or CHD which may have been induced in Grossarth-Maticek’s subjects, leading to a possible increase in the chance of obtaining either of these conditions via the process discussed in prior chapters. Schuller & Fox (1991) suggested the possible role of experimenter bias operating in the administration of the interview, but did not develop this theme beyond one paragraph- these authors appear to not have seen the relevance of this possibility to solving the puzzle of Grossarth-Maticek’s results. The current author has developed this theme further and has re-evaluated Grossarth-Maticek’s findings in light of it, with the conclusion that from the latter’s data alone, there is significant reason to suggest a treatment effect contributing to the results. A positive finding in this study would allude to a potential ‘unknown, favourable and non replicable’ condition which Amelang (1991) suggested as being behind Grossarth-Maticek’s findings.

It is considered here that there are four possible approaches to the ‘jig-saw puzzle’ of explaining Grossarth-Maticek’s results:-

i) a long term replication study of Grossarth-Maticek’s research.
ii) measure the effects on autonomic arousal and threat perception in a replication of Grossarth-Maticek’s full interview condition
iii) test the accuracy of interviewer’s predictions of subjects eventual IRI scores.
iv) examine the effects of being in a naturally occurring at risk group for cancer.

These possible approaches to explaining Grossarth-Maticek’s results will now be examined in more detail.
i) The first possible approach is to undertake a long term prospective replication study of Grossarth-Maticek’s research. Such an approach may directly prove the existence of a treatment effect by demonstrating the ability of the Grossarth-Maticek interview condition to add to cancer/CHD rates over a long period of time. For obvious ethical reasons, it is not possible to directly test this hypothesis, i.e. to create a study in which it is hypothesized that some subjects will die as a result of their participation. It is not suggested here that Grossarth-Maticek ever set out to do this, nor that he was aware of the possible implication of his methodology as per the current hypothesis, nor even that he became aware of this implication at a later time.

ii) With the impossibility of the above approach from an ethical perspective, a feasible strategy is to break this line of query down into its component parts, i.e. pieces of the ‘jig-saw puzzle’. The first aspect of this would be to demonstrate the short term effects on subject’s levels of cancer/CHD risk perception and anxiety and autonomic arousal of participation in a replication of Grossarth-Maticek’s interview condition of Trust and Understanding. Central to this approach is the question of whether some subjects left Grossarth-Maticek’s interviews with a heightened perception of vulnerability to cancer or CHD. Did subjects actually ask the interviewers at the end of the interview what the results meant for them, and did an interviewer bias operate? It is well possible that Grossarth-Maticek’s interviewers provided this information in their attempts to maintain a climate of Trust and Understanding, as answering all questions posed by the proband was one of the conditions of the interview condition. The current study will attempt to replicate Grossarth-Maticek’s interview condition of Trust and Understanding (referred to as the Full Interview condition in the current study), giving them the opportunity to inquire about the meaning of the results towards the end of the interview. Note will be taken of the number of subjects who express an interest in knowing their Short IRI scores and the score implications. This information will be given to all of the subjects who express an interest in knowing of their Short IRI scores, however, unlike Grossarth-Maticek’s original subjects, the current subjects will not be left with a cancer or CHD threat perception as a result of the interview (where as Grossarth-Maticek’s subjects may have been). Subjects who do not want to learn of their Short IRI scores will not be told these. Any introduced perception of risk status will be ‘undone’ by extensive debriefing at the end of the interview so that no subjects will leave the interview with a perception of cancer/CHD risk above the rate with which they entered the interview.
This approach has the ability to demonstrate if a significant amount of subjects in this interview condition inquire about the meaning of their interview answers; and if acquiring a perception of risk in the context of the interview leads subjects to report a heightened level of cancer/CHD anxiety and risk perception as measured by self reports. The current study will also assess for signs of an immediate alarm reaction to news of risk in the form of heart rate response. Both of these sources of data are crucial information to the current hypothesis, as it is based on the as yet untested assumption that the Type 1 & 2 subjects in Grossarth-Maticek’s study left the interview with an elevated risk perception and consequent heightened cancer/CHD anxiety levels. If the current study demonstrates this effect, then it needs to be noted that this is only a *short term* effect and does not directly demonstrate the deleterious long term effects of the interview as is hypothesized here. Again, this cannot be directly tested, however it can be inferred by the results of the second aspect of the current study (to be discussed below).

iii) As it is unknown if Grossarth-Maticek’s interviewers told their subjects of their IRI scores or whether the transmission of this knowledge was delivered via an interviewer bias, it is important to establish if an interviewer using Grossarth-Maticek’s condition of Trust and Understanding is able to develop an informal evaluation of the subject’s typology prior to the formal assessment. For Grossarth-Maticek’s interviewers to be able to unwittingly commit an interviewer bias, they must have first had the opportunity to form their own impression of the subject’s typology. They would have had ample opportunity to do this during the course of discussing the subject’s positive and negative life events and their typical responses to these (as a condition of the Full Interview). Secondly, in order for the current hypothesis to be supported, the interviewers must have been able to form an *accurate* perception of the eventual typology as assessed by the Short IRI. That is, in order for vulnerable people (Type 1 and Type 2) to made more psychologically vulnerable by receiving risk information impressions via an interviewer bias, the interviewer would have had to form an impression of the subject’s typology which was supported by the subject’s actual IRI scores. There would have had to be a high degree of concordance between the interviewer’s subjective impression of the typology and the subject’s actual IRI scores.
In the current study, the interviewer will make predictions about each subject’s typology in the Full Interview condition based on the impression formed during the discussion of positive and negative life events. These predictions will be made and recorded prior to the administration of the Short IRI, and will be compared with the actual Short IRI scores. A prediction for each subject will be made in terms of what their highest scoring type will be, i.e either Type 1, Type 2, Type 3 or Type 4. If it is found that there is a high degree of concordance between the predictions and the actual Short IRI scores, this will be taken as evidence that Grossarth-Maticek’s interviewers were also able to form accurate impressions of how the subjects would score on the IRI, making an interviewer bias more possible. There would, after all, be no self fulfilling prophecy possible if the interviewers were forming inaccurate impressions of typology, e.g if they mistakenly formed the impression that subjects were Type 1 scorers when in fact they were assessed on the IRI as being Type 4 scorers- according to the current hypothesis, they would not be psychologically vulnerable to the risk information by virtue of possessing the ‘healthy autonomous’ personality dimensions. If the interviewer is unable to accurately predict Short IRI status, then it is questionable whether Grossarth-Maticek’s interviewers would have been able to do the same, and thereby inadvertently convey to their subjects their risk status according to Grossarth-Maticek’s theory. If the level of prediction is very poor, then there is little chance of an experimenter effect. If, on the other hand the level of prediction is good, then this remains a possible explanation of Grossarth-Maticek’s findings.

iv) The second aspect of the current study is aimed at testing the hypothesis that finding oneself in a heightened risk group (as may have Grossarth-Maticek’s Type 1 & 2 subjects) is anxiety arousing not only in the short term, but over a protracted period of time- whether the illness anxiety maintains itself over time. The current hypothesis is based on the notion that Grossarth-Maticek’s Type 1 & 2 subjects, being characteristically less emotionally stable, will have maintained their elevated cancer/CHD anxiety levels for the length of the prospective study, so as to produce the reported results at all of the follow up times, extending to 15 years. It is possible that Type 1 & 2 subjects maintained their cancer/CHD anxiety over time, and that high levels of Neuroticism added in this process. As this can also not be directly tested for the ethical reasons given above, an indirect test of the general hypothesis must be developed. The method of choice to demonstrate the interaction effect between personality type and risk information producing chronic cancer/CHD anxiety is to locate a sample of people who are in a naturally occurring heightened risk group. An obvious population for
this sample are those people who are aware of having a first order relative who currently has, or has had one of the cancers which is known to have a strong hereditary factor, e.g. breast cancer, colorectal (bowel) cancer, melanoma, leukaemia, ovarian cancer, prostate cancer; or a CHD with a known hereditary factor, i.e. angina pectoris, heart attack, weak heart. Many researchers have already demonstrated that people who find themselves in groups at risk of cancer or CHD for family history reasons experience a heightened level of cancer risk or CHD risk perception and anxiety over the general population, however there have been no studies which examine whether this anxiety maintains itself over a protracted period. Frost et al (2000) did measure breast cancer anxiety subjects on how long they had known of the family risk factor and anxiety outcomes—subjects were assessed up to one year following the diagnosis, but no studies have examined this effect for a protracted period of time, e.g. 15 years. If cancer/CHD anxiety is found to be resilient over time, and contributed to by high levels of Neuroticism, then this would provide some indirect evidence of the power of a risk perception to maintain itself over time and to generate high levels of anxiety. In a sense, this is a major part of the study, as Study 1 can only test the possibility that people may have an initial emotional reaction to learning of a cancer/CHD risk—on its own, the empirical literature does not support the contention that this news of risk status would have an immediate adverse effect on health status via immunosuppression. If subjects with a family history demonstrate an elevated level of illness risk perception and anxiety which is tenacious _over time_, then the general thesis of this research program is further supported.

v) Finally, if it is demonstrated that subjects in a replication of Grossarth-Maticek’s interview condition experience a temporary heightening of arousal and risk perception as a result of the interview; and it if is demonstrated that subjects who approximate Grossarth-Maticek’s subjects (by virtue of finding themselves in a naturally occurring heightened risk group) display heightened cancer anxiety levels which are maintained over time, then the last piece of the equation is the power of risk perception and consequent protracted anxiety to adversely effect over-all health status. Previous research in psychoneuroimmunology has been cited in earlier sections which testify to the power of extended excessive anxiety and stress to adversely effect general health outcomes. If the hypotheses of the current study are supported, then this may be taken as indirect evidence of the potential for extended and excessive anxiety to play a role in the causation of cancer and CHD.
The current research program intends to utilize approaches ii, iii, and iv in attempting to throw some light onto Grossarth-Maticek’s reports. This will be undertaken via Study 1 (utilizing approaches ii and iii above), and Study 2 (utilizing approach iv above).

**Study 1:**
One of the goals of the current study is to test the power of Grossarth-Maticek’s interview to create a new risk perception and associated anxiety. Subjects who receive a risk assessment, according to the Short IRI, in the context of a replication of Grossarth-Maticek’s interview will be compared on outcome measures with subjects who receive a health prone assessment. For an interviewer bias to be a possible explanation of Grossarth-Maticek’s remarkable results, the current author suggests that his data collection method must have had the power to create a risk perception and alarm reaction in subjects in regards to the prospect of them obtaining cancer/CHD at a later date. The current study will evaluate the possibility that Grossarth-Maticek obtained his outstanding prospective results due in some part to a treatment effect which his experiment induced in vulnerable subjects, ie. the interviews may have made vulnerable subjects even more vulnerable to cancer and CHD in a process which took many years to eventuate, but which began in the assessment interview via an experimentally induced cancer/CHD risk perception and subsequent self fulfilling prophecy. The current study will attempt to replicate Grossarth-Maticek’s full interview condition. The statistical analysis will ascertain if the full interview condition has the power to stimulate the proposed arousal and perception of risk utilizing a series of Univariate ANOVA tests with Changes in Heart Rate as the dependent variable and the receipt of IRI scores as the dependent variable. Study 1 will also examine Changes in Self Report Measures as dependent variables- Univariate ANOVA tests will be conducted on Changes in Self Report Measures from time1 to time2 in both the Self Administered and the Full Interview conditions.

Figure 4 below displays a chronological presentation of the interview timeframe and variables of interest in order to elucidate the process to be undertaken. As can be seen in Figure 4, subjects in the Full Interview condition will be measured for mean heart rate during the first part of the interview (T1 timeframe), and will also be measured for mean heart rate in the second part of the interview (T2 timeframe). Separating the two timeframes will be the stimulus of learning of Short IRI scores- for some subjects this
will entail learning of a cancer or CHD proneness, whereas for other subjects this will entail learning of a health proneness. The statistical analysis, described above, will test for changes in mean heart rates between the two timeframes as a result of the stimulus of Short IRI scores being provided.

**Figure 4. Chronology of Heart Rate measures during Full Interview.**

<table>
<thead>
<tr>
<th>START</th>
<th>(time 1)</th>
<th>END</th>
</tr>
</thead>
<tbody>
<tr>
<td>IRI score given</td>
<td></td>
<td></td>
</tr>
<tr>
<td>(\text{life discussion &amp; questionnaire administration.})</td>
<td></td>
<td>(score discussion period)</td>
</tr>
<tr>
<td>(T1 mean heart rate)</td>
<td></td>
<td>(T2 mean heart rate)</td>
</tr>
</tbody>
</table>

As Study 1 is also assessing Changes in the Self Report Measures as a dependent variable, which are hypothesised to be influenced by the receipt of risk information according to the Short IRI results, a similar statistical analysis will be conducted as for the Changes in Mean Heart Rate variable. Figure 5 shows the chronology of both the Self Administered and Full Interview conditions in regards to the collection of self report measures. As can be seen in Figure 5, the self report measures will be taken at the beginning of each interview condition (Self Report 1). In the Self Administered condition, the self report measures will be taken again when the subject hands in their completed Short IRI (Self Report 2)- no risk or health prone information will be provided to them. In the Full Interview condition, self report measures will be taken after the subject has learnt of their Short IRI score and the discussion of the meaning of the score (Self Report 2).
Figure 5. Chronology of Self Report measures during both interview conditions.

<table>
<thead>
<tr>
<th>Self Administered interview:</th>
</tr>
</thead>
<tbody>
<tr>
<td>START</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Full Interview condition:</th>
</tr>
</thead>
<tbody>
<tr>
<td>IRI score given</td>
</tr>
<tr>
<td>START</td>
</tr>
</tbody>
</table>

In order to replicate Grossarth-Maticek’s full interview condition, the following guidelines are noted:- initially, subjects “were invited to take part in a scientific study of psychosocial factors in health and illness”. (Grossarth-Maticek et al 1993 p.946).

Grossarth-Maticek, Eysenck & Barrett (1993 p.945) detail the following in regards to protocol of their interview condition which entailed creating an atmosphere of Trust and Understanding. Their protocol is provided here as this constitutes the operationalisation of the interview conditions to be replicated.

“The method of administration was always by a trained interviewer, who spent one hour explaining the purpose of the questionnaire administration, assured the subject of confidentiality, answered questions both general and specific, and tried to win the trust and confidence of the subject by showing interest and concern, being friendly and courteous, giving information, and being responsive to questions ... the questions were read aloud by the interviewer, and explanations were given after each question as to the precise meaning of the question following any queries by the subject.”
This is referred to as the *explanation* factor. The next factor, *trust*, is described below as:-

“Participants were invited to talk with the interviewer for 45-60 minutes, discussing in the first part positive and negative events in their lives and their typical reactions to these situations. Following this part of the interview, participants were asked in the second part if they trusted the purpose of the questionnaire administration or if they still had some questions to ask. The interviewer did his best to answer such questions as were raised, and only began the administration of the questionnaire when the subject stated that the interviewer as well as the purpose and also design of the questionnaire administration were trusted. Following the discussion devoted to gaining the trust of the subject, the interviewer would continue with the explanation of all the items in the questionnaire. This group enjoyed both the explanatory and the trust producing paradigms”.

These factors, Trust and Understanding, will be replicated for the Full Interview condition in the current study. The other interview condition is described below.

b) Self Administered condition: an interview in which the subject is given the questionnaire to self administer. This will follow the alternative data collection method described by Grossarth-Maticek et al (1993 p.946) below:-

“Group D.- This group (n=687) constituted the control group, receiving neither the explanatory nor the trust producing interaction with the interviewer. Instead, they were given the questionnaire and asked to fill it in without prior discussion or explanation of the meaning of the questions.” (Grossarth-Maticek et al 1993)

**Potential predictor variables** in the current Study 1 are as follows (the literature suggests that these variables could have an influence on the dependent variables and, as such, will constitute part of the current study).
Neuroticism, measured in the Eysenck Personality Questionnaire (Revised)- EPQ-R. Neuroticism is reported by Schmitz’s (1992) to be factorally associated with Type 1 and Type 2 on the Short IRI. Grossarth-Maticek & Eysenck (1991.p3) refer to the cancer prone personality as the ‘cognitive aspects of the neurotic disorder’ and make repeated references to the Type 1 and Type 2 behaviour patterns as being neurotic.

Extraversion, measured in the EPQ-R. Levels of Introversion/Extraversion are relevant to the current study as Grossarth-Maticek & Eysenck (1991) state that Type 1 scorers are introverted neurotics, while Type 2 scorers are extroverted neurotics.

Monitor, measured in the Miller Behavioural Style Scale (MBSS). Miller (1996) states that people high in Monitoring experience more anxiety on learning of health risk information compared to people who are low in Monitoring.

Blunter, measured in the MBSS. Miller (1996) states that people high in Blunting are less anxious in response to health risk information as they tend to distract themselves from it as a coping strategy. As such, they are less likely to attend to such information than are those low in Blunting.

Gender. Men experience both cancer and CHD at higher rates than women, so it reasonable to expect gender would influence the degrees of cancer and CHD proneness.

Age. As age is the best predictor of both cancer and CHD, it is likely that it will effect subjects’ perceptions of cancer and CHD risk, and thereby effect the change scores in the dependent variables.

Cancer in family. Previous research supports the notion that having close family members with cancer creates a sense of vulnerability to the condition and higher levels of cancer anxiety. This variable can be seen as a possible predictor of heart rate response as well as changes in Perceived Risk of Cancer as one would expect those with family factors to already perceive themselves as being vulnerable- any information which supports this could exacerbate the existing sense of vulnerability. Frost et al (2000) studied a sample of 78 subjects in a Familial Cancer Program, 39 with and 39
without cancer. A wide range of data was gathered from these people, but these researchers did not present an analysis of the study question which is the focus of the current Study 2. Frost et al (2000) reported a mean of 17 years (sd 12) of having the familial risk knowledge for subjects with a cancer diagnosis, and 11 years (sd 10) of having familial risk knowledge for the non cancer group. Unfortunately, they did not present an analysis of how this variable interacted with personality factors to influence perceptions of risk and the consequent anxiety levels.

**CHD in family.** Previous research supports the notion that having close family members with CHD creates a sense of vulnerability to the condition and higher levels of CHD anxiety. This variable can be seen as a possible predictor of heart rate response as well as changes in Perceived Risk of CHD as one would expect those with family factors to already perceive themselves as being vulnerable- any information which supports this could exacerbate the existing sense of vulnerability.

The **dependent variables** in Study 1 are:-

**Changes in mean Heart Rate:** With subjects in the Full Interview condition, heart rate will be monitored throughout the entire interview process. Mean heart rates will be calculated for two different time frames of the interview, time1 and time2. The demarcation between time1 and time2 is the stimulus of presentation of the Short IRI scores. From these means, Changes in mean Heart Rate will be calculated by the following equation:- time2 mean heart rate minus the time1 mean heart rate.

Presentation of the Short IRI scores by the interviewer will be accompanied by an explanation and interpretation in terms of what predictions Grossarth-Maticek would make on the basis of the scores. As such, the stimulus (learning of the score) is not necessarily at the point when the score is presented, but arguably can be at the point when the subject is able to interpret what the score means for them and their future according to Grossarth-Maticek. This point of understanding and meaning could occur at any time during this phase of the interview.
**Changes in Self Report measures.** The second dependent variable in this study is the self report data obtained from subjects concerning perceived cancer/CHD risk and cancer/CHD anxieties. These will be obtained on 10 point Likert scales both before the questionnaire is administered (at the beginning of time1) and again at the completion of the interview process (at the end of time2). For Full Interview subjects, this means they will be asked the Self Report questions again after they have received their Short IRI scores (time2). For the Self Administered subjects, they will be asked the Self Report questions again when they hand back the completed Short IRI. From these two sampling occasions, changes in these self report measures will be calculated as the following variables by subtracting the value for time1 scores from time2 scores.

The **Independent variables** in Study 1 are:-

**Short IRI Typology scores.**

The current author considers that there are several ways in which ‘risk’ can be inferred from the Short IRI scores by the subject.

**Definition 1**- the actual IRI score (airi_c).

The Short IRI Type scores contain different levels of risk.

- Type 1 being the highest score is presumed to pose a risk of cancer proneness;
- Type 2 being the highest score is presumed to pose a risk of CHD proneness;
- Type 3 being the highest score is presumed to pose relative good health proneness;
- Type 4 being the highest score is presumed to pose optimal health proneness.

When subjects have indicated an interest in knowing of their scores, they will be provided with their Short IRI typology results and an explanation as to what Grossarth-Maticek would suggest it means. Grossarth-Maticek suggests that the **highest score indicates the degree of risk.** For example, a subject who scores in the following way,

Type 1 score= 6; Type 2 score= 2; Type 3 score= 4; Type 4 score= 2

will be advised that Grossarth-Maticek would read this as a psychological proneness to cancer.
Whereas a subject who scores in the following way,

Type 1 score= 2; Type 2 score = 3; Type 3 score = 1; **Type 4 score= 8**

will be advised that Grossarth-Matichek would read this as a psychological proneness to positive health.

**Definition 2- relative risk index 1 (rri1).** When a Type 1 or 2 score is within 1 point of the Type 4 score. It is possible for a subject to obtain a high Type 4 score and still perceive risk information in regards to cancer or CHD proneness. Such as,

Type 1 score= 6; Type 2 score = 5; Type 3 score = 4; **Type 4 score= 7**

This subject will still be advised that according to Grossarth-Matichek, they are more prone to positive health than to cancer or CHD, but there is some concern with their scores in that Type 1 & 2 are both high as well. In this example, the Type 1 score is within one point of the Type 4 score.

It is possible that subjects who receive a profile wherein their Type 1 or 2 score is within 1 point of their Type 4 score will still infer a sense of risk. As such, the analyses will include this definition of risk as well as definition 1 for separate statistical analysis.

**Definition 3-relative risk index 2 (rri2).** When a Type 1 or 2 score is more than 5/10. It is possible for these subjects to still infer a risk when their ‘illness prone’ scores are more than half of the total possible scores on that particular scale, regardless of their Type 4 score. For example, a person with the following profile

Type 1 score= 3; **Type 2 score= 6**; Type 3 score = 4; **Type 4 score = 8**

could still be expected to perceive a degree of risk information. As such, the statistical analyses will include this definition of risk as well as definitions 1 and 2.
**Interview conditions**

In this study, subjects will be randomly allocated to either of the two following interview conditions.

a) Full Interview condition- an in-depth interview in which the conditions of understanding and trust (as per Grossarth-Maticzek’s operational definitions) are created by the interviewer before the questionnaire items are presented to them and answers obtained by the interviewer, or

b) Self Administered condition- an interview in which the subject is given the questionnaire to self administer.

As stated earlier, it is expected that Study 1 has the power to determine if the receipt of risk information in the context of Grossarth-Maticzek’s full interview is able to create an alarm reaction, as measured by both autonomic changes and changes in self report measures of risk perception and disease anxiety. A further component of Study 1 is to test whether the interviewer has the ability to predict a subject’s eventual Short IRI score (in terms of disease prone or health prone) on the basis of the subject’s description of their positive and negative life events as discussed in the first part of the interview. Predictions will be compared with the eventual Short IRI scores and a binomial test will be conducted to determine whether the predictions were accurate above the rate of chance.

The research endeavours described in Study 1 reflect the possible approaches to explaining Grossarth-Maticzek’s reports as detailed earlier in this chapter, ie. approaches ii) and iii). Approach iv) will be described below as Study 2 in the current research program.

**Study 2:**

Study 2 is examining cancer/CHD risk perception and anxiety in a group naturally ‘at risk’ of certain cancers/CHD’s according to family history. The goal of Study 2 is to ascertain if perception of risk and anxiety in this ‘at-risk’ sample is resilient to the effects of time, and enhanced by the subject’s level of Neuroticism. The purpose of this second goal is to indirectly test the overall proposal of this study, ie. the most neurotic of
Grossarth-Maticek’s subjects left his interview with a new perception of risk for cancer or CHD- this risk perception and anxiety maintained itself over the course of time (up to 15 years), and was enhanced by the type of rumination associated with neurotic anxiety. In the absence of a 15 year prospective study, this hypothesis cannot be directly tested, so a group of subjects who naturally view themselves as being at risk of cancer or CHD (for family history reasons) will be identified from within the current sample and studied. This group represents an approximation of Grossarth-Maticek’s subjects who were identified as being at risk of cancer or CHD, and who the current author presumes left the interview with a new risk perception. Similarly, subjects with first order family members who have experienced the hereditable cancers and CHD are presumed to view themselves at risk of these conditions. Whether this risk perception and illness anxiety maintains itself over time, and whether it is associated with Neuroticism scores will be ascertained in the current study.

The dependent variables in Study 2 will be taken from the Health Psychology Questionnaire which will be completed by all study participants regardless of whether they attend the follow-up interview or not. As these measures are to be taken before the subject has participated in any discussion about psycho-social factors relating the ill health, these will be responses unadulterated by the interview process.

All of the following dependent variables will be measured by subjects responses on 10 point Likert scales.

- **Cancer Anxiety** (from Health Psychology Questionnaire)
- **CHD Anxiety** (from Health Psychology Questionnaire)
- **Cancer risk perception** (from Health Psychology Questionnaire)
- **CHD risk perception** (from Health Psychology Questionnaire)
The independent variables in Study 2 are:-

**Cancer in Family**-number. Subjects who nominate that they have first order relatives with cancers that are known to have an hereditary component will be asked to state how many family members fall into this category.

**Cancer in Family**- years. Subjects who nominate that they have first order relatives with cancers that are known to have an hereditary component will be asked how many years they have been aware of this fact.

**CHD in Family**- number. Subjects who nominate that they have first order relatives with CHD’s that are known to have an hereditary component will be asked to state how many family members fall into this category.

**CHD in Family**- years. Subjects who nominate that they have first order relatives with CHD’s that are known to have an hereditary component will be asked how many years they have been aware of this fact.

**Neuroticism.** The EPQ-R forms part of the Health Psychology Questionnaire. Subjects will be scored on their levels of neuroticism on this test.

Combining the examinations conducted under Study 1 and Study 2 of the current research program, answers should be provided as to:–

1. whether health risk information provided in Grossarth-Maticek’s full interview procedure has the power to create an immediate alarm reaction, as well as a self reported increase in risk perception and associated anxiety;
2. whether the interviewer is able to accurately predict the eventual Short IRI score on the basis of the discussion of subjects life events and their typical reactions;
3. whether there is a relationship between having first order relatives with particular cancers/CHD’s and risk perception as well as associated anxiety; and whether these associations maintain across the passage of time, and if association is enhanced by Neuroticism.
Positive findings in these examinations will provide a degree of understanding as to how Grossarth-Maticek arrived at his remarkable results via the proposed interviewer bias.

**Heart Rate reactivity to psychological experience:**
The current study will be using changes in heart rate as a measure of induced anxiety as a result of participation in Grossarth-Maticek’s full interview condition- subjects who receive health risk information will be compared on changes in heart rate with subjects who receive health prone information. As such, a review of heart rate research literature is required. In support of using heart rate response as an indicator of psychological stress, Reidbord & Redington (in Port & van Gelder 1995 p.534) state,

> “The dynamics of the cardiovascular system are largely driven by multiple interconnections in the brain through neural and humoral pathways. These pathways are part of the sympathetic and parasympathetic autonomic nervous system, and form a direct connection between brain centres and peripheral organs, Thus, under certain circumstances heart function may provide indirect, yet timely and precise access to autonomic nervous system dynamics, and thereby to core brain states”.

As the Full Interview condition specifies that all information will be provided to subjects in order to assist them in understanding the research, subjects in the current study will be given the opportunity to learn of their Short Interpersonal Risk Inventory score at the end of the interview process. In order to measure the amount of autonomic arousal in subjects on receipt of their scores heart rate responses to this stimuli will be measured as an indicator of anxiety.

What kind of person would respond with heightened stress and associated autonomic arousal to perceiving a high risk status for themselves during Grossarth-Maticek’s interview format? Gump & Mathews (1999) reviewed 19 published studies in their attempt to ascertain the effect of ‘background stressors’, which occur in a chronic manner
(eg. occupational stress, marital and family stress, daily hassles) on responses to acute stressors. They concluded that a majority of studies support the notion that ongoing stressors produce heightened autonomic reactivity to acute stressors, and are also associated with slow recovery following acute stressors. This conclusion is relevant to the current study in that it is assumed that people who were the most psychologically vulnerable in Grossarth-Maticke’s study (ie, Type 1 & 2 subjects) are also those who score higher in measures of neuroticism and experience higher levels of background stress. According to Gump et al’s (1999) conclusions, these would be the people most likely to have a larger autonomic response to perceiving that they were thought to be at risk of cancer or CHD, as they concluded that there is an association between high levels of background stress and high levels of autonomic response to acute stress. The more psychologically vulnerable people were those who one would expect to obtain a higher Type 1 or Type 2 score; they are also those people who one would expect to have higher levels of background stress; and they are the people who Gump et al (1999) suggest would display a larger autonomic response to an acute stressor (eg. learning of a poor health risk status) and a slower recovery time from that stressor. Models of stress adaptation (Gump et al 1999) suggest that repeated coping with background stressors produce fatigue in the organism which could diminish coping reserves, in turn affecting one’s capacity for coping with subsequent or concurrent stressors.

Philippot (1993) makes the point that even though there is no definition of emotion which is universally accepted amongst researchers, there is a general consensus that emotion is a multi faceted phenomenon. Kubzansky, Kawachi, Weiss & Sparrow (1998 p.48) state,

“Emotion theorists have suggested that specific emotions are a product of the interaction between the person and the environment, and they mediate continually changing situations and the individual behaviour. As such, emotions are a process that motivates the organism to respond to its environment and allows an adaptive flexibility of response that is not available to organisms which rely on instinct. Thus, emotions are considered to be functionally appropriate processes which
may have dysfunctional consequences when the system is taxed beyond the limits of its capability.”

As Steiner, Ryst, Berkowitz, Gschwendt, Koopman (2002) suggest, studying the human stress response as an emotional experience is a complicated process in that it incorporates multiple elements. Levenson (1992 p.23) makes the point that “emotions are short lived phenomenon, typically lasting only a few seconds”. The experimental study of emotions generally entails eliciting certain emotion states in the subject, however, individuals differ in their emotional reactions to the same situations. What may deeply affect one individual may fail to move another; a stressful interview situation may elicit anger, fear or depression in different individuals, or any combination of these (Philippot 1993).

There is an assumption in the current study that learning of a risk for cancer/CHD status according to the Short IRI will lead to an anxiety/stress response, however it is possible that a range of responses may occur, e.g satisfaction that one’s long held worst fears are confirmed by the Short IRI score; a perverse form of pleasure resulting from now having a victim status vis-à-vis a life threatening condition; a form of excitation may even result at the prospect of being relieved of standard expectations, ‘wellness’ roles and responsibilities, as well as an anticipation of finally being looked after by others; excitement at facing the greatest challenge of one’s life.

A further question facing the researcher is how to best measure a particular response? Physiological measures of autonomic arousal such as heart rate are popular in the research literature. However Bloom & Trautt (1977) report that Finger Pulse Volume is a sensitive measure of autonomic arousal associated with anxiety while Kantor, Endler, Heslegrave, Kocovski (2001) state that heart rate is a common example of a physiological measure of anxiety. Blumenthal, Lane & Williams (1985) state that increased heart rate is part of the classic fight or flight response pattern, along with accelerated blood pressure and vasodilation in skeletal muscles in preparation for the strenuous activity associated with self defense. Zahn, Nurnberger, Berrenttini & Robinson (1990) state that there is considerable evidence linking anxiety to autonomic responses, such as heart rate in
studies comparing controls to subjects with various types of anxiety states. Amongst researchers of anxiety disorders, autonomic manifestations of anxiety are given virtually equal billing with subjective and behavioural indices (Zahn et al 1990 p100). Within these autonomic manifestations, heart rate is often proposed as an objective index of anxiety. Evans & Steptoe (2001 p.367) cite research evidence demonstrating that “pressure and heart rate are elevated during episodes of perceived stress, independent of physical activity and posture”. The often stated conclusions with normal subjects are in contrast to studies with subjects experiencing anxiety conditions- with the former, the relationship between anxiety and autonomic indices have not been consistently observed whereas they have been with people experiencing an anxiety disorder (Zahn et al 1990).

Fear is an emotion which is generally experienced as being stressful. Levinson et al (1992) categorize it as a negative emotion which has been found to be associated with accelerated heart rates, along with anger and sadness. Sharpley (2002 p56) states, “The speed at which the heart beats is determined by the autonomic nervous system (ANS), which operates via the sympathetic nervous system (SNS) and the parasympathetic (PNS) nervous systems. Activation of the SNS and withdrawal of the PNS are generally associated with “stress” reactions, that is, increases in the physiological and psychological arousal of the body to a threat or demand. Thus SNS dominance over PNS acts to increase HR (heart rate) and BP (blood pressure) as well as other aspects of our physiological functioning, so that blood and oxygen can be carried quickly to the muscles which we use when under stress. Conversely, PNS dominance over SNS automatically decreases HR and BP (and other physiological responses). Between the two of them, the SNS and PNS allow the body to achieve a balance, so that we react to threat appropriately, but are not continually over-aroused.”

It is hypothesized in the current study that when subjects are informed that the Short IRI has designated them as a person with a risk status for cancer or CHD, they will respond with a measurable fear response indicated by an increase in their heart rate. Fear in this
context may also be labeled anxiety, as the emotion is fear of a future possible event. In the current study, heart rate response will be used as a measure of autonomic arousal to the stimuli provided. Levinson et al (1992 p24) state,

“The association of fear with accelerated heart rate may reflect fear’s close association with the motor program of “flight”, which makes significant metabolic demands on the heart.”

In a review of the research literature testing for a specific fear response as indicated by heart rate, Levinson et al (1992) present a list of studies which have demonstrated a clear and consistent set of findings associating heart rate accelerations with the experience of fear. The studies reviewed cover a wide range of methodologies from traditional laboratory stress tests, through to the use of mental imagery of feared situations as compared to happy situations. Levinson et al (1992) conclude that the weight of evidence is overwhelmingly in support of increases in heart rate as a response to fear, anger and sadness, although they note that there have been some studies which have failed to support these associations.

Blumenthal et al (1985) state that increased heart rate, blood pressure, cortisol, serum cholesterol levels, epinephrine and norepinephrine are seen in animals as well as humans when they are subjected to both acute and chronic stress. It is anticipated that learning of a risk status according to the Short IRI will be a stressful event for subjects in the current study, and that it will activate a fight-flight response, indicated by increases in heart rate.

Of interest to this study is the finding which Valdimarsdottir (2002) reported from a study with women who were in a naturally occurring risk group for breast cancer due to family history. He found that in a laboratory stress test, women with familial breast cancer risk displayed higher heart rates than did women at normal level of breast cancer risk, and women in a non stress task. Their anxiety about cancer risk obviously generalized to a higher level of autonomic reactivity to other stressors.
Huang, Ebey, and Wolf (1989) state that researchers seeking to create a measurable emotional stress reaction in study participants have traditionally relied upon a range of stimuli such as mental arithmetic, horror movies, mild electric shocks, time urgency, or various types of distractions while the subject is attempting to complete a task. They view this as being a less than ideal strategy, however as emotional arousal depends more on the significance which the stressor has to the individual rather than on the quantity of the stimuli. As such, their preference is to measure physiological reactions during an interview, discussing issues of known importance to the subject after a control period of neutral conversation. The stressful discussion period is followed by a period of reassurance and diversion- the researchers collected a range of physiological measures indicating levels of arousal, including heart rate. As in the current study, the interviewers, after engaging in neutral conversation, initiated a discussion with the subjects in relation to an earlier life experience which was known to have been stressful to the subject. This section of the interview was followed by a changing of topics in which the subjects were asked to discuss happy, rewarding circumstances in their lives. This procedure was undertaken with 17 subjects, demonstrating an increase in heart rate measures during the stress interview section of the procedure for 12 of the 17 subjects.

In a naturalistic study comparing the heart rates of bereaved individuals with depressed or control subjects O'Connor, Allen & Kaszniak (2002) found that bereaved subjects were characterized by significantly higher heart rates- this would concur with the fact that bereavement is a stressful experience. Davidson, Marshall, Tomarken & Henriques (2000) studied the different levels of arousal between a social phobic group compared to a control group in a situation which most people find stressful, anticipating making a public speech. While both of the groups displayed increases in autonomic arousal as indicated by increases in heart rate, the social phobia group displayed greater heart rate acceleration before and after the speech was given.

Carels, Blumenthal & Sherwood (2000), in a study of 162 men and women whose levels of emotional reactivity was measured during a 24 hour period, found that subjects who reported higher levels of daily stress, trait anxiety and depression demonstrated greater
increases in heart rate associated with negative emotions than did subjects with different profiles. Further, they concluded that high anxiety subjects tended to be more emotionally responsive to cues in their environment which indicated a possible threat. This conclusion has significance in the current study as it is hypothesized that the more vulnerable subjects (ie. those who the Short IRI assesses as being cancer or CHD prone) will be more emotionally responsive, as demonstrated by heart rate acceleration to threat information which may be given to them in the interview, ie. their Short IRI risk status.

Fichera & Andreassi (2000) examined the effects of a stressful situation, a public speaking task, on cardiovascular reactivity of a sample of 86 men and women. All of the subjects displayed a marked cardiovascular response during the task with increased heart rate. In a similar study, Kantor et al (2001) found that their subjects showed an elevated heart rate during a seminar presentation (as opposed to the viewing of a seminar presentation), and that the maximum heart rates were obtained in the first 15 minutes of the task. In a stressful mental arithmetic task Sanz & Villamarin (2001) report that subjects displayed increases in heart rate while undergoing the task, with most subject’s heart rates peaking at the beginning of the task and maximum levels being recorded in the first minute. They refer to this as being a typical pattern of cardiovascular reactivity which also entails an increase in systolic pressure, a slight increase in diastolic pressure, and a decrease in skin temperature (Sanz et al 2001). They also report an inverse relationship between self efficacy and heart rate during the stressful task, with higher self efficacy scorers displaying lower heart rate responses to stress. Huwe, Hennig & Netter (1998) found a positive relationship between the stress associated with undergoing an oral exam and increases in heart rate in a study with 84 subjects. The heart rate scores increased during the exam, peaked immediately after the exam, and were seen to be lower than pre-exam levels four weeks later. Steiner et al (2002), using a sample of 133 non clinical adolescents, also reported an increase in heart rate measures during a stress task, this time delivering a speech.
Possible confounds in heart rate research:
Greenfield & Sternbach (1992) state that there is no general agreement on the shape of heart rate responses. In a similar vein Quigley, Barret & Weinstein (2002) posit that heart rate response, whether it be accelerative or decelerative, will depend upon the cognitive processing of the stressful situation. This is relevant to the current study as it is anticipated that subjects who learn of a risk for cancer or CHD status according to the Short IRI will perceive this news as threatening information. Baldaro, Mazzetti, Codispoti, Tuozzi, Bolzani & Trombini (2001) also reported a heart rate deceleration in response to threatening stimuli, referred to as bradycardia. Simons, Detenber, Roedema & Reiss (2001) studied the different responses in heart rate due to both the ‘medium of the stimuli’ (in the form of moving footage compared to still images) as well as the message of the stimuli (the level of emotional valence which it holds for subjects). From their study, examining the effects of emotion evoking film footage of 35 subjects, Simons et al (2001) demonstrated that there were several phases to heart rate response. Firstly, subjects displayed a short deceleration indicative of orienting to image onset. This was followed by a mid-interval heart rate acceleration, a phase in which the affective properties of the stimuli dominate. Finally, motion in the film footage becomes the dominant factor, with heart rate returning to a bradycardia pattern as moving images appear to capture more attentional resources. While the current study does not entail the use of either moving or still visual images, it does present the subjects with a new stimuli (in the form of IRI scores) which they are required to attend to. For many of the subjects, the valence of this stimuli will be mild in that they will be told of a proneness to positive health according to the IRI scores- this may merely confirm the pre-existing views which they hold of themselves. For other subjects, it can be expected that the valence of the stimuli will be strong in that some will be informed of a cancer or CHD risk status according to the Short IRI.

A complicating factor with measuring heart rate response to psychological stress is the different levels of responsivity according to the levels of fitness in the subjects, as well as an inherent attribute of some people to have low heart rates (Boutcher, Nugent & Weltman 1995). In regards to genetic inheritance for anxiety disorders, Zahn et al (1990)
studied the response patterns to stress of a population of subjects who were considered to be at risk of developing anxiety disorders due to having parents with such disorders. They concluded that only high risk subjects showed a clear generalized ANS reaction to stress. A similar potential confound of heart rate response as an indicator of stress is the prior life experience which subjects have undergone. Buckley & Kaloupek (2001) found a role for PTSD in heart rate responses of Vietnam veterans with PTSD. Also studying Vietnam veterans with PTSD, Beckham, Barefoot, Fairbank, Vrana, Feldman & Moore (2002) concluded that the PTSD group showed higher heart rate responses than the non PTSD group. Lynch, Lynch & Friedman (1992) state that the mere behaviour of talking leads to increases in both blood pressure and heart rate. Gender differences are another possible confound with heart rate research that has been reported in the literature (Valdimarsdottir 2002).

In the current study, levels of anxiety about cancer and CHD proneness are also being assessed via self reports on a Likert scale. It is important to assess if there will be a self reported increase in anxiety as a result of participating in this study and learning of IRI scores, as well as an effect on self reported perceived risk of cancer/CHD. Kantor et al (2001) report that correlations between psychological self reports of anxiety and physiological measures such as heart rate are typically weak. It is, however, of interest in this study to have more than one measure of anxiety (ie. heart rate), and self reports on a Likert scale are a standard way of assessing cancer anxiety (Holland & Rowland 1989).

Despite the above mentioned limitations to heart rate research, the large amount of articles reporting research of stress reactions with heart rate as an index of response testifies to the usefulness of this as an outcome measure.

The use of Self Report measures:
The current study will also utilize several self report measures in order to ascertain the effects of the interview on subjects in regards to their perceptions of risk to cancer and CHD, as well as their cancer and CHD anxiety levels. Subjects will provide answers in regards to their perception of risk and anxiety levels on 10 point Likert Scales. Justification for this type of risk perception assessment
question is provided by Audrain, Schwartz, Lerman, Hughes, Peshkin & Biesecker (1997). In their study of women who presented for genetic counselling in regards to breast and ovarian cancer, Audrain et al (1997 p.372) used a single Likert-type scale item to assess perceptions of breast cancer and ovarian cancer risk:

“In your opinion, compared to other women of your age, what are your chances of getting breast cancer?” 1= much lower to 5= much higher… This item was adapted from an item used and validated in previous studies”.

This research group have published many articles regarding cancer risk perception and anxiety and rely almost exclusively on Likert-type scales such as the one used in the current study. These are the psychological measures which will indicate if subjects come to view themselves as being more vulnerable to cancer/CHD as a consequence of learning of their Short IRI score. It is possible, of course, that these questions may obtain a socially desirable response as they are self reports, whereas the Changes in Mean Heart Rate measures are an objective indicator of autonomic arousal, here being interpreted as an indicator of anxiety. As such, it is considered more thorough to include both self report measures as well as the objective measure of changes in heart rate in order to gauge arousal/stress levels as well as changes in perception of risk.

Grossarth-Maticek’s Yugloslavian probands were elderly, whereas his Heidelberg sample was more in the middle aged group. For the current study, it is considered important to replicate a sample with these age groups, but also to ascertain if the findings were relevant for a wider age range including younger people. As such, subjects will be recruited from a range of groups ensuring an appropriate age spread. It is anticipated that as per Grossarh-Maticek’s reports, most people in a non clinical sample will be assessed as ‘health prone’ on the Short IRI. As such, there is a concern that there may not be enough subjects assessed as ‘cancer prone’ or ‘CHD prone’ according to the Short IRI to adequately test the study hypotheses. The coordinators of the Lismore Anxiety Management Centre will be contacted in order to discuss the research project and to ascertain if they would be willing to invite their patients to participate in the current study.
As stated earlier in this chapter, the goals of the current research project are to ascertain if a replication of Grossarth-Matichek’s Full Interview condition has the power to create an alarm/stress reaction and increased perception of risk in subjects who receive health risk information. In addition, a second goal is to ascertain if subjects who are within a health risk group, by virtue of having first order relatives with hereditable cancers and CHD’s, display a heightened perception of risk and anxiety compared to subjects not in this group; whether this risk perception and anxiety is maintained over time, and if Neuroticism adds to this outcome.

In summary, the current research program entails two studies, designed to ascertain whether negative health risk information has the power to create an immediate alarm reaction and increased sense of risk on self report measures; whether the interviewer can accurately predict subjects eventual Short IRI scores; and whether there is a relationship between being in a heightened risk group for cancer/CHD and disease anxiety/risk perception. In order to achieve these study aims, the following research will be conducted:

**Study 1:** The effects of a negative health prediction

- **Question 1**- when compared to subjects who receive a positive health prediction, do subjects who receive a negative health prediction show an increase in heart rate on presentation of a negative health prediction?
- **Question 2**- when compared to subjects who receive a positive health prediction, do subjects who receive a negative health prediction show an increase in self report measures of disease risk perception and anxiety on presentation of a negative health prediction?
- **Question 3**- is the current experimenter able to predict subjects eventual Short IRI scores (in terms of health risk or positive health prediction) on the basis of the life discussion conducted as part of the Full Interview condition?
Study 2: The relative effects of family history, Neuroticism and time on disease risk perception and anxiety.

- Question 1- will subjects with first order relatives known to have hereditable cancers/CHD’s show higher levels of cancer/CHD risk perception when compared to subjects with no such family history?
- Question 2- will a known family history of hereditable cancer/CHD interact with Neuroticism to influence risk perception and anxiety scores?
- Question 3- will a known family history of hereditable cancer/CHD interact with length of this knowledge (in years) to influence risk perception and anxiety scores?

The steps involved in collecting the data required to answer these questions will be detailed in the next chapter.
Chapter 13

METHODOLOGY

The current research program is designed to ascertain whether negative health risk information has the power to create an immediate alarm reaction and increased sense of risk on self report measures; whether the interviewer can accurately predict subjects eventual Short IRI scores; and whether there is a relationship between being in a heightened risk group for cancer/CHD and disease anxiety/risk perception. This research will contain elements of a single case design which will be replicated for each of the subjects in each of the various conditions. This type of study was developed by early behavioural research on schedules of reinforcement and is often used in applied and clinical settings when behaviour modification techniques are used (Cosby 1981).

The current research program is broken into two main studies.

Study 1: The effects of a negative health prediction

- Question 1- when compared to subjects who receive a positive health prediction, do subjects who receive a negative health prediction show an increase in heart rate on presentation of a negative health prediction?
- Question 2- when compared to subjects who receive a positive health prediction, do subjects who receive a negative health prediction show an increase in self report measures of disease risk perception and anxiety?
- Question 3- is the current experimenter able to predict subject’s eventual Short IRI scores (in terms of health risk or positive health prediction) on the basis of life discussion?

Study 2: The relative effects of family history, Neuroticism and time on disease risk perception and anxiety.

- Question 1- will subjects with first order relatives known to have hereditable cancers/CHD’s show higher levels of cancer/CHD risk perception when compared to subjects with no such family history?
• Question 2- will a known family history of hereditable cancer/CHD interact with Neuroticism to influence risk perception and anxiety scores?
• Question 3- will a known family history of hereditable cancer/CHD interact with length of this knowledge (in years) to influence risk perception and anxiety scores?

**Study 1.**
The goal of Study 1 is to assess if receiving a health risk prediction, in the context of a replication of Grossarth-Maticek’s full interview condition, has the power to create an initial alarm reaction, and to create changes in subjects’ risk perception of cancer/CHD and associated illness anxiety. Study 1 will implement a research design which will empirically test for these possible consequences using changes in heart rate measures and changes in self report measures as the dependent variables. The purpose of this study will be to test whether the threatening information provided to some subjects in these interviews is able to create a treatment effect of heightened arousal, increased perception of risk and associated anxiety. Subjects in the current study will be randomly allocated to either the Full Interview condition, or to the Self Administered interview condition. Subjects in the Full Interview condition will be informed of their Short IRI results and the implications of their scores as it is presumed that Grossarth-Maticek’s subjects learnt of their risk status either as a consequence of being told or as a consequence of an interviewer bias. Subjects in the Self Administered condition will not be informed of their Short IRI scores. Dependent variables (changes in heart rate and changes in self report measures of illness risk perception and anxiety) will be taken, whereby responses of subjects receiving risk information will be compared with responses of subjects receiving health prone information.

**Study 1 Hypotheses:**

i) **Hypothesis 1.** There will be significantly larger heart rate responses of Full Interview subjects informed of a negative health prediction compared to the heart rate responses of subjects informed of a positive health prediction.

ii) **Hypothesis 2.** The Changes in Self Report variables will show increases in risk perception and illness anxiety in subjects in the Full Interview condition who
receive risk information, whereas it will show no increases in these variables for subjects in the Full Interview condition who receive health prone information or for subjects in the Self Administered interview condition.

iii) **Hypothesis 3.** In the Full Interview condition, the experimenter will make predictions about the subject’s typology on the Short IRI (ie. whether their highest score is likely to be Type 1, Type 2, Type 3 or Type 4) which will be accurate (in relation to the actual Short IRI scores to be calculated) at a rate above that of chance.

**Study 2.**
The goal of Study 2 is to assess if finding oneself in a heightened risk group for cancer or CHD has the power to increase subjects levels of cancer/CHD anxiety and risk perception; and whether the relationship is strengthened by Neuroticism; and whether the hypothesized cancer/CHD anxiety and risk perception maintains itself over time. In order to meet these study aims, Study 2 will be assessing the risk perceptions of first order relatives of people who have experienced a cancer or CHD with known heredity etiology for levels of cancer/CHD anxiety, as well as their responses to the Eysenck Personality Questionnaire, and the Miller Behavioural Style Scale.

**Study 2 Hypotheses:**

i) **Hypothesis 1.** Subjects who respond that they have first order relatives with cancers/CHD known to have a strong genetic component will demonstrate higher levels of cancer/CHD risk perception and anxiety than subjects with no such family history.

ii) **Hypothesis 2.** Neuroticism scores will add to the variance of cancer/CHD risk perception and anxiety when comparing subjects with and those without a relevant family history.

iii) **Hypothesis 3.** This effect will be demonstrated over time, with the level of cancer/CHD risk perception and anxiety remaining high regardless of the amount of intervening years from which they learnt of their heightened level of cancer/CHD risk.
Research Program Participants.

In order to obtain sample for Studies 1 & 2 with a wide age range, subjects will be recruited from the following sources:-

- Probus Clubs- these are organizations for senior citizens which meet on a monthly basis for social purposes. This cohort is similar in age to Grossarth-Maticek’s Yugoslavian sample, ie. elderly. Four local Probus Clubs in the North Coast area of NSW (Lismore, Casino, Ballina and Alstonville) will be contacted and members will be invited to participate. Probus clubs are community based social groups for retirees of a professional and/or business background. They tend to meet in a community setting once a month and engage in a range of social activities including outings, listening to guest speakers, and engaging in discussions of particular topics.

- The student body of Southern Cross University. Most of these will be students of the Lismore campus (where the interviews will be conducted), however there will also be students from the Coffs Harbour campus as well as external students from different parts of Australia who will complete the Health Psychology Questionnaire and return via email (this data will be used for Study 2). A recruiting email will initially be sent out to all Southern Cross University students by the current author’s academic supervisor.

- Subjects will be recruited from the staff body of Southern Cross University via an email to the entire staff body to ensure a more ‘middle-aged’ range of subjects.

- Subjects will be recruited from the researcher’s work colleagues. These people offered questionnaire responses, however they will not be invited in for interviews as it is considered a possible complication to have the interviewer being well known to the subject in a work context- their data will join that from other respondents to the Health Psychology Questionnaire who are unable to attend the interview and will be used for Study 2.

- Subjects will be recruited from the patient population of the Northern Rivers Area Health Service Anxiety Management Clinic (AMC).
Questionnaire respondents will be selected for participation in the interview if they fulfill the following conditions:-

1) they nominate their willingness to attend an interview;
2) they have not experienced a cancer or CHD diagnosis.

**Study 1 Protocols:**

- receive the completed Health Psychology questionnaires (Appendix 4) and score.
- invite subjects from all the Probus Club, University and Anxiety Management Clinic sources in for an interview session if they fulfill two conditions stated above.
- subjects will be randomly allocated to either of the interview conditions.
- Full Interview condition: an in-depth interview in which the conditions of Trust and Understanding are created by the interviewer before the questionnaire items are presented and answers obtained by the interviewer—this will follow the protocol as described by Grossarth-Maticek (see chapter 11).
- Self Administered condition, whereby subjects are simply given the Short IRI (Appendix 5) and asked to complete it by themselves.
- In the Full Interview condition, develop the conditions of Trust and Understanding by such actions on behalf of the interviewer as:- spending up to 45-60 minutes explaining the purpose of the questionnaire administration, assuring the subject of confidentiality, answering questions both general and specific; discussing positive and negative life events of the subject, and their typical reactions to these; and trying to win the trust and confidence of the subject by showing interest and concern, being friendly and courteous, giving information, and being responsive to questions.
- on commencing the interview, describe and explain the bio-feedback equipment (Polar Heart Rate Monitor) which will measure subject’s levels of autonomic arousal via heart rate measurements. When subjects are comfortable to do so, demonstrate how they can attach the monitor across their own chest and allow them to do so (in private).
- on commencement of the interview, subjects will be informed that this is a longitudinal health study and their permission will be sought to attempt to contact them in 2 years time and each 2 years subsequent to that— the purpose would be a five minute phone conversation to
ascertain if there have been any major health issues that have arisen in the intervening 2 years. (see appendices for Interview format).

• subjects will be asked to respond to the following Self Report questions pertaining to cancer and CHD risk perception and anxiety (Appendix 4- the same self report questions were used as in the Health Psychology Questionnaire).

• in the Full Interview condition, when the conditions of understanding and trust have been established (checked with the subject), the interviewer will then administer Grossarth-Maticek’s Short Interpersonal Reactions Inventory (Short IRI). Respondent’s answers will be recorded by the interviewer on the answer sheet.

• On completion of the test administration, subjects in the Full Interview condition will be asked if there are any questions they would like to ask about the study, and it will be made clear to them that they can learn of their Short IRI scores if they would like to.

• The time will be noted when subjects are given their scores and this time will be corroborated against the time and heart rate recording of the Polar Heart Rate Monitor. This device allows the heart rate to be measured at regular intervals and records each of these measurements over the interview time frame. It will then be downloaded to a software program titled Advisor. This program will produce a print out chart for each interview showing the heart rate measurements each 5 seconds. It will also produce the values of each of these measurements which can then be transported to an Excel Spreadsheet enabling data analysis, including comparisons of mean heart rates for time1 and time2 timeframes.

• It is anticipated that a descriptive explanation of the Short IRI scores and their meaning will be required- this may take up to 20 minutes in order to ensure that the subject is aware of what the score means for them in terms of Grossarh-Maticek’s predictions.

• The Perceived Cancer Risk question, Cancer Anxiety question; Perceived CHD Risk question and CHD Anxiety questions will again be asked.

• After learning of their Short IRI scores and the implications according to Grossarth-Maticek, the subjects will be de-briefed as to the contrary findings of validity of the Short IRI, and re-assured that the result is not a predictor of cancer or CHD. This phase will continue until the subject has understood the equivocal value of the IRI in terms of predicting cancer or CHD.

An emphasis will be made on the research findings which run contra to Grossarth-Maticek’s claims, eg. Amelang’s research. It will be emphasized that all other researchers in the health
psychology field have found either no consistent and convincing proof of psycho-social factors in ill-health, or at best only moderate effects. Young subjects will be informed that age is the largest predictor of these conditions and that even if Grossarth-Matichek is correct, they have many years in which to effect the behavioural changes required to minimize their psychological risk factors, as well as other life style risks. Reference will be made to Grossarth-Matichek’s intervention studies in which he claims that where at-risk people were able to modify their behavioural excesses, they were able to reduce the incidence of cancer and CHD. Subjects will be given references to self help books (eg. Seligman’s ‘Learned Optimism’) and information on free counseling services which are available to them if they feel to need for help in effecting change in their lives. Each subject will be given a handout (see Appendix 9) in which the above assertions are made; it will also contain a list of early warning signs of cancer and they will be encouraged to seek early medical advice if any of these signs are present. In short, great effort will be made to re-assure subjects that a high risk score is not a prognosis of cancer or CHD, and that there are many things they can do to prevent these conditions.

- subjects in the Self Administered condition will not be told of their scores on the Short IRI. They will also be asked the Perceived Cancer Risk question, Cancer Anxiety question; Perceived CHD Risk question and CHD Anxiety question on completion of the Short IRI. These subjects will then be informed of the nature of the research as this was not done for them before the administration of the questionnaire- any debriefing required will occur at this point.

- Subjects in both interview conditions will be thanked for their participation in the study and the interview will be terminated when they have no more questions to ask.

- Statistical analyses will be conducted.

**Study 1.**

Subjects will be assessed on a range of dependent variable measures (mean Heart Rate and Self Report measures of perceived risk of cancer and CHD, and cancer and CHD anxiety) at two stages of the data collection process- time1 & time2. For subjects in the Full Interview condition, time1 entails the timeframe in the interview when they are introduced to the general topic area (psycho-social factors in the causation of cancer and CHD); have any questions or queries about the topic area
answered; are given a thorough description of the current study aims, purposes and methodology; are asked if they have any queries or concerns about these; are asked to discuss positive and negative life events and their typical reactions to these; and finally, when they state they have sufficient Trust and Understanding in the current study, are asked to respond to the Short IRI questions, with each item being read out and explained by the experimenter. The time1 timeframe concludes for the Full Interview subjects when they have completed answering all of the Short IRI questions. Subjects will then be asked if there is anything they would like to know about the study, and it will be made clear to them that they can know their Short IRI scores if they wish to. If they respond that they would like to know of their Short IRI scores, note will be made of this, and the scores will be calculated while the subject waits (taking less than 5 minutes to score). The time2 timeframe commences with the subjects being told what their Short IRI scores are and having these explained to them- this discussion may take from as little as 1-2 minutes, and as long as 20 minutes for the interpretation to be understood. The Self Report measures will be taken at the commencement of time1 and at the completion of time2. Heart Rate will be measured throughout the entire interview so that a mean Heart Rate score for time1 can be calculated, and a mean Heart Rate score for time2 can be calculated.

For subjects in the Self Administered interview condition, only the Self Report measures will be taken at time1 and time2. They will be asked the Self Report questions prior to commencing the Short IRI, and again on concluding the Short IRI. As they will not be told of their Short IRI scores, there is no need to obtain their heart rates, or to measure changes in heart rates in response to learning of their health prediction.

**Study 1 interview sequences.**

The sequence of the Full Interview condition is as follows:-

1) Brief description of study purpose→
2) invitation to participate in a long term prospective study→
3) cancer/CHD likelihood and anxiety questions→
4) discussion of significant life events and typical responses→
5) ensure trust and understanding in the study purpose→
6) Full interviewer administration of the Short IRI→
7) questioning subject’s desire to know of their score
8) working out of Short IRI score
9) presentation and interpretation of Short IRI score
10) post questionnaire asking of cancer/CHD likelihood and anxiety questions
11) debrief
12) finish.

The sequence of the **Self Administered** condition is as follows:-

1) invitation to participate in a long term prospective study
2) cancer/CHD likelihood and anxiety questions
3) giving the subject the Short IRI to self administer- standardized instructions from the tool were read out to the subject
4) receive the completed questionnaire
5) re-administer the Self Report questions
6) provide explanation and conduct any debrief as required
7) finish.

Figure 6 below shows the steps involved in data collection in flowchart form.
Figure 6. Steps involved in data collection in Study 1.

1. Recruit subjects
2. Health Psych Questionnaire: has Subject had Cancer or CHD?
   - Yes: Health Psych Questionnaire data used for Study 1& 2
   - No: Random allocation to either:
     - Full Interview condition
     - Self Administered interview condition
3. Full Interview condition:
   - Attach and explain Polar Heart Rate Monitor
   - Discuss subject’s positive/negative life events and responses
   - Note prediction of eventual IRI results
   - Subject reports sufficient Trust and Understanding of the research?
     - Yes: Interviewer administer Short IRI
     - No: Continue explaining purpose of the study until sufficient Trust & Understanding is reported
4. Self Administered interview condition:
   - Administer Self Report Questions
   - Hand subject Short IRI for self admin.
   - On completion, ask Self Report Questions.
   - End interview
The Variables in Study 1:

**Neuroticism** - measured in the EPQ.

**Extraversion** - measured in the EPQ.

**Monitor** - measured in the MBBS.

**Blunter** - measured in the MBSS.

**Gender**

**Age**

**Cancer in family**

**CHD in family**
In order to obtain these measures, subjects in this study will be asked to provide answers to the following: inventories-
1) Eysenck Personality Questionnaire (EPQ)- (Appendix 4).
2) The Short Interpersonal Reactions Inventory- (Appendix 5).
The Inventory asks 10 questions corresponding to each of the four scales used and one extra scale for Type 4 (Health prone) as a reversed item scale (Type 4b).
3) the cancer/CHD risk perception questions and anxiety questions (Appendix 4).
4) The Health Psychology Questionnaire- (Appendix 4). This has been devised to contain the EPQ-R, the Miller Behavioural Style Scale, and additional items relating to respondent’s gender, demographic characteristics, and previous diagnoses of cancer/CHD. The Questionnaire asks respondents if they are willing to participate in the next stage of data collection, and to provide their name and phone number if willing.
5) And finally, physiological measures of arousal will be obtained by the Polar Heart Rate Monitor.

The dependent variables in Study 1:
The dependent variables in Study 1 are:-

i) Changes in mean Heart Rate:
With subjects in the Full Interview condition, heart rate will be monitored throughout the entire interview process. Mean heart rates will be calculated for time1 and for time2 timeframes of the interview. From these means, Changes in mean Heart Rate will be calculated by the following equation:- time2 mean heart rate minus the time1 mean heart rate.

A decrease in heart rate from time1 to time2 will be reflected in a negative value on this score; and increase in heart rate from time1 to time2 (indicating an alarm response) will be reflected in a positive value on this score.

ii) Changes in Self Report measures
The second dependent variable in this study is the self report data obtained from subjects concerning perceived cancer/CHD risk and cancer/CHD anxieties. These will be obtained on 10 point Likert
scales both before the questionnaire is administered (at time1) and again at the completion of the interview process. For Full Interview subjects, this means they will be asked the Self Report questions again after they have received their Short IRI scores (time2). For the Self Administered subjects, they will be asked the Self Report questions again when they hand back the completed Short IRI. From these two sampling occasions, changes in these self report measures will be calculated as the following variables by subtracting the value for time1 scores from time2 scores.

The following variables will be generated:

- a) Change in Perceived-Risk- Cancer
- b) Change in Cancer Anxiety
- c) Change in Perceived- Risk- CHD
- d) Change in CHD Anxiety.

A negative score will indicate a decrease in the above risk/anxiety variables, while a positive score will indicate an increase in the risk/anxiety variables, i.e. a negative score, for example, means that the subject’s cancer anxiety had gone down from the pre-interview sampling to the post interview sampling; whereas a positive score on these change variables means that, for example, the subject’s cancer anxiety has increased from pre-interview to post interview.

**The Independent variables in Study 1:**

The independent variables in this study are:-

i) **Short IRI Typology scores.**

Results will be calculated according to each of the three definitions of ‘risk information’ as detailed in Chapter 11.

ii) **Interview conditions**

Subjects will be randomly allocated to either of the two following interview conditions.

a) Full Interview condition
b) Self Administered condition

**Reliability and Validity data for Study 1 measures:**
The research instrument is made up of measures which have been well utilised in published Health Psychology research.

1) The Short IRI. Eysenck (1990) reports the validity of the IRI (in terms of accuracy of predictions in a prospective study) as being expressed in a correlation =0.81.

   The Short Interpersonal Reactions Inventory- for items, see Appendix 5. This scale was devised by Grossarth-Matick. The Inventory asks respondents to answer 50 questions, with 10 questions corresponding to each of the four scales used with an extra reverse item scale for Type 4 Health Prone. These scales are:-
   Scale 1 (Cancer Prone). Scale 2 (Coronary Heart Disease prone)
   Scale 3 (Relatively Healthy). Scale 4a (Autonomous- Healthy). Scale 4b (reversed items Autonomous Healthy).

   Eysenck and Grossarth-Matick (1991) state that the Inventory was able to predict the mortality by cancer and CHD rates in 81% of cases.

   The averaged test re-test reliability of the two scales of most interest (Scale 1: Cancer prone & Scale 2: CHD prone) are reported as showing a correlation =0.95 (Eysenck & Grossarth-Matick 1991).

   Grossarth-Matick & Eysenck (1990) report that the test-retest correlations of all of their scales, including those to be used in the current study are in excess of 0.80.

2) EPQ-R: Eysenck & Eysenck (1994) report that the EPQ-R has reliability (alpha co-efficients) of 0.79 for males and 0.76 for females. The test-retest reliabilities with one month between testing, using a sample of 160 mixed sex subjects are:- Psychoticism 0.71; Extroversion 0.92; Neuroticism 0.89; Lie Scale 0.83.

3) Risk perception and anxiety questions.
Lipkus, Biradavolu, Fenn, Keller & Rimer (2001) conducted a study assessing risk perceptions and anxieties of women to breast cancer using a Likert scale as in the current study. Test-retest correlations were from .71 (p<.001) to .62 (p<.002) in two different groups. Lipkus et al (2001) also report moderate (.2-.4) associations between breast cancer perceived risk and breast cancer worry, suggesting that the measures are non-redundant.
The variables in Study 2:
Data from items in the Health Psychology Questionnaire will be utilized in Study 2, including Family History questions (Appendix 4). Study 2 entails obtaining data from subjects who have first order relatives with cancers and CHD’s which are known to have a substantial genetic component.

The dependent variables in Study 2:
The following measures will be taken from the Health Psychology Questionnaire which will be completed by all study participants regardless of whether they attend the follow-up interview or not. As such, the sample for Study 2 will be larger than for Study 1 as the latter contains data only from subjects who attended the interview.

All of the following variables will be measured by subjects responses on 10 point Likert scales.

i) Cancer Anxiety  
ii) CHD Anxiety  
iii) Cancer risk perception  
iv) CHD risk perception

The independent variables in Study 2:

i) Cancer in Family-number  
Subjects who nominate that they have first order relatives with cancers that are known to have an hereditary component will be asked to state how many family members fall into this category.

ii) Cancer in Family- years  
Subjects who nominate that they have first order relatives with cancers that are known to have an hereditary component will be asked how many years they have been aware of this fact.

iii) CHD in Family- number  
Subjects who nominate that they have first order relatives with CHD’s that are known to have an hereditary component will be asked to state how many family members fall into this category.
iv) **CHD in Family- years**

Subjects who nominate that they have first order relatives with CHD’s that are known to have an hereditary component will be asked how many years they have been aware of this fact.

v) **Neuroticism**

This will be measured by the EPQ-R and forms part of the Health Psychology Questionnaire.

**Statistical Analyses to be used in the current research project:**

The current study will utilize the following software package in conducting the required statistical analyses- SPSS 11.5 for Windows.

A descriptive analysis will be undertaken to determine if the subjects in the Full Interview condition represent the same sample as subjects in the Self Administered condition on a range of demographic variables. T tests will be conducted comparing males and females on the family history of cancer and CHD variables; the same analysis will be conducted on interview condition. Males and females will be compared with a t test on the dependent variable of Change in Heart Rate to determine whether gender plays a role in this variable. T tests will also be used to compare the responses of males and females on all of the dependent variables in Study 1, as well as the predictor variables and potential predictor variables, again, to determine if gender will effect the outcomes.

The same t test analysis will be conducted, comparing the responses of subjects in the Full Interview condition with those in the Self Administered condition to determine if subjects in either condition represent the same sample on the range of variables.

Following this descriptive analysis, an analysis of Grossarth-Maticke’s taxonomy will be conducted in order to determine if the data collected in this sample conforms with the patterns described in his literature (Grossarth-Maticke 1980). According to his reports (Grossarth-Maticke 1980), types on the Short IRI should show correlations with particular variables in this study. A pattern of correlations between these variables and types from the Short IRI will allow the current researcher to have a degree of confidence in the results found. A lack of expected correlations will lead to more caution in interpreting the results. These correlational analyses will be undertaken for the entire
sample, and for each of the interview conditions respectively to determine if there is a consistency of correlations across the interview conditions.

The main study hypotheses of the current program will then be addressed. Before proceeding to this however, the effects of both gender and age will be evaluated in terms of the receipt of risk information. As detailed earlier, there are three definitions of risk information used in this study. The effects of gender and age on the receipt of risk information will be evaluated according to each of these definitions of risk information using Chi-Squared analyses. Age will be broken down into three categories, i.e. under 30; 30-49; 50 and over; gender is broken into two categories of male and female. Separate Chi-Squared analyses will be conducted with each these variables (age and gender) for each of the three definitions of receipt of risk information. A correlational analysis will then be conducted to determine the nature of the relationships between each of the study variables and the dependent variable of Changes in Mean Heart rate. In testing the main hypothesis of Study 1, a series of Univariate ANOVA tests will be undertaken with the Changes in Heart rate variable and the receipt of risk information (as per the three definitions) or health prone information. These will be followed by an analysis of the contribution of both gender and age, using a Type I hierarchical decomposition sum of squares method in which each term is adjusted for only the term that precedes it in the model, i.e., age and gender before the risk variables. Each of the three definitions of receipt of risk information will be used in the analysis.

As Study 1 is also examining self report measures as dependent variables, Univariate ANOVA tests will be conducted on Changes in Self Report measures from time1 to time2 in the Self Administered condition and for the Full Interview condition (using each of the three definitions of health risk information according to the Short IRI scores). In order to test the current experimenter’s ability to accurately predict subjects eventual Short IRI scores above the rate of chance, a binomial test will be conducted.

In testing the hypotheses of Study 2, a correlational analysis examining the relationships between the self report measures of cancer/CHD anxiety and risk perception and the
relevant variables will be conducted. This will be followed by a series of Multiple Regression Analyses, examining the effects on cancer/CHD anxiety and risk perception with the between subjects factor being ‘yes’ or ‘no’ family history of cancer or CHD. Additional factors such as Neuroticism scores and years of having this knowledge will be examined for their effects.

Table 16 displays the proposed statistical analyses for both Study 1 and 2, as well the expected outcomes and hypotheses which these are related to.

Table 16. Proposed statistical analyses.

<table>
<thead>
<tr>
<th>Analysis</th>
<th>Expected Outcome</th>
<th>Related to Hypotheses</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Study 1:</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Univariate ANOVA tests on Changes in Mean Heart Rates.</td>
<td>The Changes in Mean Heart Rates for subjects who receive risk information will show an increase in heart rate from time1 to time2.</td>
<td>There will be significantly larger heart rate responses of Full Interview subjects informed of a negative health prediction compared to the heart rate responses of subjects informed of a positive health prediction.</td>
</tr>
<tr>
<td>Binomial test</td>
<td>Observed cases of accurate predictions of eventual IRI risk scores will be greater</td>
<td>The current experimenter will be able to accurately predict an IRI risk.</td>
</tr>
</tbody>
</table>

270
than that of chance. assessment based on subject’s descriptions of their live events.

<table>
<thead>
<tr>
<th>Study 2. Analysis</th>
<th>Expected Outcome</th>
<th>Related to Hypothesis</th>
</tr>
</thead>
<tbody>
<tr>
<td>Correlational Analysis</td>
<td>Significant positive correlations will be found between family history of cancer/CHD and cancer/CHD risk perception and anxiety.</td>
<td>Subjects with first order relatives known to have hereditable cancers/CHD’s will show higher levels of cancer/CHD risk perception and anxiety compared to subjects with no such history.</td>
</tr>
<tr>
<td>Multiple Regression Analysis</td>
<td>The additional factor of Neuroticism will explain variance in risk perception and anxiety.</td>
<td>Known family history of hereditable cancers/CHD’s will interact with Neuroticism to increase risk perception and anxiety.</td>
</tr>
<tr>
<td>Multiple Regression Analysis</td>
<td>The additional factor of years of knowledge of family history will explain variance in risk perception and anxiety.</td>
<td>Any observed relationships between family history and self report measures will not diminish with the introduction of years.</td>
</tr>
</tbody>
</table>
Chapter 14

RESULTS

This chapter will describe the results of the current study in the following order:-

- **A) Descriptive analysis** - the purpose of this section is to compare the trends of findings from the current sample in relation to the findings from Grossarth-Matick’s samples. If the same trends are observed as in Grossarth-Matickeks samples (eg. Type 1 scores reflecting high Neuroticism and Introversion; Type 2 scores reflecting high Neuroticism and Extroversion; Type 4 scores reflecting low Neuroticism), then one can have more confidence in the overall results. If such correlations are not found, then either Grossarth-Maticek’s reports or the current findings must be called into question.

- **B) Study 1 Results** - the outcomes of the statistical analyses described in the previous chapter will be presented. The purpose of this will be to establish if the receipt of a negative health prediction is seen to produce an alarm reaction (as seen in increases in heart rate) and an increased sense of risk and disease anxiety in subjects who received such information when compared with subjects who received a health prone prediction.

- **C) Study 2 Results** - the outcomes of the statistical analyses described in the previous chapter will be presented. The purpose of this will be to establish if subjects with a first order relative known to have hereditable cancers display a higher sense of disease risk and anxiety when compared with subjects without such a family history. Further, the role of Neuroticism and the years of this knowledge will be assessed for their contribution to the observed variance.

**Section A) Descriptive analysis:**

Below are two sets of tables that describe the sample through variables relating to family history of cancer and CHD (Tables 17 & 18) and through the variables for statistical analysis (Tables 19, 20 & 21). Each set of data are represented by Gender (Tables 17, 19 & 20) and
by Interview Condition (Tables 18 & 21). A Chi-Square analysis shows no “Gender by Interview Condition” bias (Chi-Square=0.00, p=.960, df=1).

Demographic characteristics by Gender and Interview Condition:

Table 17 shows distributions on the family history of cancer and CHD data by Gender for the entire sample. T tests for these variables show no differences within the sample based on gender. Males and Females represent the same population on these data.

Table 17. Family history of cancer & CHD data by Gender for the entire sample.

<table>
<thead>
<tr>
<th></th>
<th>Male</th>
<th>Female</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>n= 26</td>
<td>n= 79</td>
<td>N= 105</td>
</tr>
<tr>
<td>Mean</td>
<td>Age</td>
<td>Age</td>
<td>Age</td>
</tr>
<tr>
<td>SD</td>
<td>43.04</td>
<td>39.01</td>
<td>40.01</td>
</tr>
<tr>
<td>min</td>
<td>18.27</td>
<td>15.64</td>
<td>16.33</td>
</tr>
<tr>
<td>max</td>
<td>18</td>
<td>18</td>
<td>18</td>
</tr>
<tr>
<td>mean</td>
<td>76</td>
<td>77</td>
<td>77</td>
</tr>
<tr>
<td>t</td>
<td>.1091</td>
<td>.362</td>
<td>.379</td>
</tr>
<tr>
<td>p</td>
<td>.278</td>
<td>.718</td>
<td>.705</td>
</tr>
<tr>
<td>No. of family</td>
<td>.46</td>
<td>.39</td>
<td>.41</td>
</tr>
<tr>
<td>with cancer.</td>
<td>.86</td>
<td>.83</td>
<td>.84</td>
</tr>
<tr>
<td>Years of family</td>
<td>3.65</td>
<td>3.00</td>
<td>3.16</td>
</tr>
<tr>
<td>with cancer.</td>
<td>8.98</td>
<td>7.15</td>
<td>7.60</td>
</tr>
<tr>
<td>No. of family</td>
<td>.65</td>
<td>.42</td>
<td>.48</td>
</tr>
<tr>
<td>with CHD.</td>
<td>.89</td>
<td>.68</td>
<td>.74</td>
</tr>
<tr>
<td>Years of family</td>
<td>8.96</td>
<td>5.80</td>
<td>6.58</td>
</tr>
<tr>
<td>with CHD.</td>
<td>14.53</td>
<td>12.26</td>
<td>12.86</td>
</tr>
</tbody>
</table>

Table 18 shows distributions on the family history of cancer and CHD data by Interview Condition for the entire sample. T tests for these variables show no bias by Interview Condition. Full Interview and Self-Administered groups represent the same population on these data.
In summary, Tables 17 & 18 show that in regards to the demographic characteristics of family history of cancer and CHD, there are no statistically significant differences in the sample based on gender or interview type.

**The study variables by Gender and Interview Condition:**

Tables 19, 20 and 21 are presented in order to ascertain the uniformity of the population sampled. These tables compare means and standard-deviations for each of the dependent, independent and potential predictor variables by Gender and Interview Condition.

As can be seen in Table 19, there are no statistically significant differences in the dependent variable of Change in Mean Heart Rate due to Gender in this sample.

<table>
<thead>
<tr>
<th>Dependent variable</th>
<th>Male</th>
<th>Female</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>Change in Heart Rate</td>
<td>-3.42 2.87</td>
<td>-3.06 2.75</td>
<td>-3.14 2.76</td>
</tr>
</tbody>
</table>

As the dependent variable ‘Change in Mean Heart Rates’ only relates to the analysis of the Full Interview condition subjects, whereas the other Self Report dependent variables relate to the entire sample, the latter will be presented separately in Tables 20 & 21. Potential predictor variables, Neuroticism, Extroversion, Monitor & Blunter are included in Tables
20& 21 as the health psychology literature suggests relationships between them and ‘signature responses’ (eg. alarm) to news of risk, including altered perceptions of risk and physiological responsiveness. Neuroticism, Extroversion/Introversion, and Monitor/Blunter scores all have implications for anxiety responses, as discussed in earlier sections. These personality dimensions proffered by Eysenck (1965) and Miller (1996) have the potential to influence the dependent variable measures.

Other potential predictors included in Tables 20 & 21 are the perceived Likelihood of cancer and CHD measures. These have been included as they may have an effect on the dependent variables. Subjects who already view themselves as being at risk may not be alarmed to learn that the Short IRI confirms this perception. As such, the anticipated alarm reaction may not occur in an ‘at-risk’ subject merely because there was a pre-existing perception of risk.

The measures of cancer likelihood and CHD likelihood in Table 20 and 21 were obtained in the first phase of data collection within the Health Psychology Questionnaire which was completed by the respondents at least one month prior to the interview. As with all the other potential predictors in this study, these Likelihood measures were therefore not effected by the interview conditions. Again, it is important to assess if the gender and interview condition cohorts significantly differ on these dimensions, as differences would effect the dependent variable measures with significant differences implying that the results may be more related to these factors than to the experimental conditions. Valdimarsdottir (2002) states that research has demonstrated greater increases in heart rate responses for women in response to stress. However, other researchers (Steiner, Berkowitz, Geschwendt & Koopman 2002) point out that the salience of the stressor to the different genders is a major factor in determining if men or women display larger heart rate responses.

Table 20 shows the mean scores for dependent variables (changes in Self Report measures), hypothesized predictors and potential predictors by Gender for the entire sample. As can be seen in Table 20, the $t$ and $p$ values indicate that there are no significant differences between males and females on these variables. Note that the mean Type 1 scores for males are higher than they are for females (3.69 cf 2.73 respectively) with a $p$ value of .051. This is extremely
close to being statistically significant. Grossarth-Matick & Eysenck (1990) found in the Heidelberg sample, that men scored higher on Type 4 than women, with the male mean for Type 4 being 4.53, and the female mean for Type 4 being 3.94. As Grossarth-Matick & Eysenck (1990) do not provide standard deviations for these means, it is not possible to generate $t$ and $p$ values and thus determine where the difference was by chance. At a cursory glance it seems unlikely that the gender differences found in Grossarth-Matick & Eysenck (1990) would reach statistical significance. Within the set of Hypothesised Predictors, the Type 4 measures are the highest scores. This would suggest that health proneness, according to Grossarth-Matick’s claims, is the prediction for most subjects in this sample. Most subjects received news from the Short IRI that they were prone to positive health. This would be expected in that around 2 in every 5 of Grossarth-Matick’s subjects were assessed as being prone to cancer/CHD, with 3 out of 5 assessed as being prone to positive health. The data here is consistent with what we would expect to find in the general population based on Grossarth-Matick & Eysenck’s (1990) reports.
Table 20. Descriptive statistics by Gender for the hypothesised Predictors and Potential Predictors in the full sample.

<table>
<thead>
<tr>
<th></th>
<th>Male</th>
<th>Female</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>n= 26</td>
<td>n= 79</td>
<td>N= 105</td>
</tr>
<tr>
<td></td>
<td>Mean</td>
<td>SD</td>
<td>Mean</td>
</tr>
<tr>
<td>Dependent variables</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Change in Cancer Risk Perc.</td>
<td>-.81 1.52</td>
<td>-.43 .93</td>
<td>-.52 1.11</td>
</tr>
<tr>
<td>Change in Cancer anxiety</td>
<td>-.35 .69</td>
<td>-.34 1.12</td>
<td>-.34 1.03</td>
</tr>
<tr>
<td>Change in CHD Risk Perc.</td>
<td>-.42 1.03</td>
<td>-.41 1.18</td>
<td>-.41 1.14</td>
</tr>
<tr>
<td>Change in CHD anxiety</td>
<td>-.35 1.77</td>
<td>-.90 1.74</td>
<td>-.76 1.75</td>
</tr>
<tr>
<td>Hypothesised Predictors</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Type 1</td>
<td>3.69 2.36</td>
<td>2.73 2.07</td>
<td>2.97 2.18</td>
</tr>
<tr>
<td>Type 2</td>
<td>2.21 2.49</td>
<td>1.63 2.00</td>
<td>1.75 2.13</td>
</tr>
<tr>
<td>Type 3</td>
<td>3.38 2.23</td>
<td>2.63 1.63</td>
<td>2.82 1.81</td>
</tr>
<tr>
<td>Type 4</td>
<td>7.85 1.54</td>
<td>7.29 1.86</td>
<td>7.43 1.79</td>
</tr>
<tr>
<td>Potential Predictors</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Cancer likelihood</td>
<td>4.52 2.16</td>
<td>4.23 2.34</td>
<td>4.30 2.29</td>
</tr>
<tr>
<td>CHD likelihood</td>
<td>4.06 2.41</td>
<td>4.48 2.24</td>
<td>4.38 2.28</td>
</tr>
<tr>
<td>Extroversion</td>
<td>8.15 3.45</td>
<td>7.58 3.22</td>
<td>7.72 3.27</td>
</tr>
<tr>
<td>Neuroticism</td>
<td>4.96 3.84</td>
<td>5.51 3.52</td>
<td>5.37 3.59</td>
</tr>
<tr>
<td>Monitor score</td>
<td>10.11 2.52</td>
<td>10.38 2.41</td>
<td>10.31 2.43</td>
</tr>
<tr>
<td>Blunter score</td>
<td>4.68 2.35</td>
<td>4.39 2.47</td>
<td>4.46 2.43</td>
</tr>
</tbody>
</table>

Table 21 compares the mean scores for each of the dependent, independent and potential predictor variables by Interview Condition. Table 21 also includes the additional potential predictor variables that were included in Table 20- the reasons for their inclusion in Table 21 are the same as detailed above for Table 20. Table 21 shows the Change in Mean Heart Rate variable for the Full Interview subjects only, as it was only these subjects with whom this variable was relevant due to the Self Administered interview condition subjects not receiving their Short IRI scores- as such, no t test was conducted on this variable by Interview Condition.

As can be seen in Table 21, the only significant difference between the Full Interview and the Self Administered interview groups are Changes in Perceived Risk for Cancer, with subjects in the Full Interview condition showing a greater drop in perceived risk than subjects in the Self Administered condition (p<.05).
Table 21 shows that Full Interview and Self-Administered groups represent the same population on all variables involved in hypothesis testing as there are no significant differences between subjects in the Interview Conditions apart from the one reported above. As such, it can be concluded that the subjects in each interview condition represent the same population with no bias operating to effect the results.

<table>
<thead>
<tr>
<th>Table 21. Descriptive statistics by Interview Condition for Dependent variables, Independent variables and potential predictor variables.</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
</tr>
<tr>
<td></td>
</tr>
<tr>
<td></td>
</tr>
<tr>
<td>Dependent variables</td>
</tr>
<tr>
<td>Change in Heart Rate</td>
</tr>
<tr>
<td>Change in Perc-Risk for Cancer</td>
</tr>
<tr>
<td>Change in Cancer anxiety</td>
</tr>
<tr>
<td>Change in Perc-Risk for CHD</td>
</tr>
<tr>
<td>Change in CHD anxiety</td>
</tr>
<tr>
<td>Hypothesised Predictors</td>
</tr>
<tr>
<td>Type 1</td>
</tr>
<tr>
<td>Type 2</td>
</tr>
<tr>
<td>Type 3</td>
</tr>
<tr>
<td>Type 4</td>
</tr>
<tr>
<td>Potential Predictors</td>
</tr>
<tr>
<td>Cancer likelihood</td>
</tr>
<tr>
<td>CHD likelihood</td>
</tr>
<tr>
<td>Extroversion</td>
</tr>
<tr>
<td>Neuroticism</td>
</tr>
<tr>
<td>Monitor score</td>
</tr>
<tr>
<td>Blunter score</td>
</tr>
</tbody>
</table>

* significant at \( p \leq .05 \)

NB: Full Inter = Full Interview Condition, Self-Ad = Self-Administered Condition

In summary, as there are no differences in the study variables according to both Gender and Interview Condition (apart from that shown in Table 21 above), the data for the following analysis is considered to be free of bias for the Interview Condition and Gender- males and females, and subjects in either interview condition appear for the most part to present with the same values on each of the study variables.
Analysis of Grossarth-Maticke’s taxonomy:

It is important to establish if Grossarth-Maticke’s health and illness prone constructs (Grossarth-Maticke & Eysenck 1990) used in this study actually display patterns which are consistent with their reports. Without such a concordance, doubt would have to be cast on either their or the current results.

Correlations for the full sample:

Grossarth-Maticke and Eysenck (1990) report that there is a correlation between Type 1 and Type 2 scores (as they are both predictive of ill-health); and between Type 1/Type 2 scores and Type 3 scores as the latter is a mixture of the behavioral tendencies of the former; and a negative correlation between Type 1/Type 2/Type 3 scores and Type 4 scores, as the latter entails a behavioural style which is oppositional to the other three styles.

Table 22 shows the correlations that were calculated for each of the Short IRI scales in the current sample, ie. Type 1 (Cancer prone); Type 2 (CHD prone); Type 3 (relative health); Type 4 (Positive health). These figures relate to all subjects who were in either the Self Administered condition or the Full Interview Condition. As can be seen, Type 1 scores across the sample are strongly positively correlated with Type 2 scores, and less strongly positively correlated with Type 3 scores (all of these correlations reach levels statistical significance at the .001 level). Type 1 scores also negatively correlate with Type 4 scores at a significance level of .05. Type 2 scores are significantly positively correlated with Type 1 scores; significantly positively correlated with Type 3 scores; and significantly negatively correlated with Type 4 scores (all of these correlations reach levels statistical significance at the .001 level). Type 3 scores positively correlate with Type 1 and Type 2 scores (both at the .001 level) and negatively correlate with Type 4 scores (also at the .001 level). Type 4 scores are significantly negatively correlated with Type 1 scores at the .05 level), show a stronger significantly negative correlation with Type 2 scores, and are significantly negatively correlated with Type 3 scores (both at the .001 level). As can be seen, there is confirmation in the current sample of Grossarth-Maticke’s proposition that Type 1, Type 2 and Type 3 are oppositional to Type 4 scores- they appear to be measuring differing tendencies. This table
shows a pattern of responses that are consistent with Grossarth-Maticzek & Eysenck’s reports (1990).

Table 22. Short IRI scale correlations for the entire study sample (N=105).

<table>
<thead>
<tr>
<th></th>
<th>IRI T1</th>
<th>IRI T2</th>
<th>IRI T3</th>
<th>IRI T4</th>
</tr>
</thead>
<tbody>
<tr>
<td>IRI T1</td>
<td>Correlation</td>
<td>1</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Sig. (2-tailed)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>IRI T2</td>
<td>Correlation</td>
<td>.444(**)</td>
<td>1</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Sig. (2-tailed)</td>
<td>.000</td>
<td></td>
<td></td>
</tr>
<tr>
<td>IRI T3</td>
<td>Correlation</td>
<td>.403(**)</td>
<td>.524(**)</td>
<td>1</td>
</tr>
<tr>
<td></td>
<td>Sig. (2-tailed)</td>
<td>.000</td>
<td>.000</td>
<td></td>
</tr>
<tr>
<td>IRI T4</td>
<td>Correlation</td>
<td>-.236(*)</td>
<td>-.390(**)</td>
<td>-.355(**)</td>
</tr>
<tr>
<td></td>
<td>Sig. (2-tailed)</td>
<td>.015</td>
<td>.000</td>
<td>.000</td>
</tr>
</tbody>
</table>

** Correlation is significant at the 0.01 level (2-tailed). * Correlation is significant at the 0.05 level (2-tailed).

Table 23 shows the correlations between Short IRI type scores and personality variables of Extroversion, Neuroticism, Monitor and Blunter scores. It can be seen in Table 23 that there are strong positive correlations between Type 1 scores and Type 2, Type 3 and Neuroticism scores (all p<.01). Type 1 and Extroversion scores show a significant negative correlation (p<.01). There are non significant positive correlations between Type 1 and Monitor and Blunter scores. Type 2 scores are positively correlated with Type 1, Type 3 and Neuroticism scores (all p<.01), and with Monitor scores (p<.05) whereas the positive correlation with Blunter scores is non significant. Type 2 scores are strongly negatively correlated with Type 4 scores (p<.01) and non significantly negatively correlated with Extroversion. Type 3 scores are strongly positively correlated with Type 1, Type 2 and Neuroticism scores (all p<.01) and non significantly correlated in a positive direction with Monitor and Blunter scores. Type 4 scores are negatively correlated with Type 1 scores (p<.05), Type 2, Type 3 and Neuroticism scores (all p<.01). There is a positive but non-significant correlation between Type 4 scores and Extroversion, and negative but non significant correlations with Monitor and Blunter scores.
Extraversion is the inverse measure to Introversion on the EPQ. Introversion is reported by Grossarth-Maticek & Eysenck (1990) to be factorially related to the Type 1 profile in that the Type 1 profile reflects a combination of high Neuroticism and Introversion; while the Type 2 profile is said to reflect a combination of high Neuroticism and Extroversion.

Extroversion scores show negative correlations with Type 1 scores (p<.01), as well as with Type 2 and Type 3 scores (non significant)- this means that Introversion shows a significant correlation with Type 1 scores (p<.01), and non significant correlations with Type 2 and Type 3 scores. There is a significant negative correlation between Extroversion and Neuroticism scores (p<.05) and non significant positive correlations with Type 4, Monitor and Blunter scores.

Neuroticism positively correlates with Type 1, Type 2 and Type 3 scores (all p<.01), and negatively correlates with Type 4 scores (p<.01). Neuroticism also negatively correlates with Extroversion (p<.01). There are non-significant positive correlations between Neuroticism and Monitor and Blunter scores.

In regards to Miller’s (1996) construct of Monitor/Blunter, it can be seen that there are no significant correlations between either of these variables and other personality variables, other than a significant positive correlation between Monitor scores and Type 2 scores (p<.05). This is consistent with Miller’s (1996) claim that the Monitor construct measures a different facet of personality/coping style than do other constructs such as Neuroticism.

In summary, this table shows a pattern of measures obtained which are consistent to those reported by Grossarth-Maticek & Eysenck (1990) in regards to the relationships between the Short IRI scores and personality measures of Neuroticism and Introversion/Extroversion.
Table 23. Correlations between independent and personality variables for full sample (N=105)

<table>
<thead>
<tr>
<th>Type</th>
<th>Extroversion</th>
<th>Neuroticism</th>
<th>Monitor score</th>
<th>Blunter score</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Correlation</td>
<td>.350(**)</td>
<td>.336(**)</td>
<td>.007</td>
</tr>
<tr>
<td></td>
<td>Sig. (2 tailed)</td>
<td>.002</td>
<td>.000</td>
<td>.942</td>
</tr>
<tr>
<td>Type 2</td>
<td>Correlation</td>
<td>-.146</td>
<td>.520(**)</td>
<td>.246(*)</td>
</tr>
<tr>
<td></td>
<td>Sig. (2 tailed)</td>
<td>.136</td>
<td>.000</td>
<td>.011</td>
</tr>
<tr>
<td>Type 3</td>
<td>Correlation</td>
<td>-.090</td>
<td>.471(**)</td>
<td>.100</td>
</tr>
<tr>
<td></td>
<td>Sig. (2 tailed)</td>
<td>.364</td>
<td>.000</td>
<td>.308</td>
</tr>
<tr>
<td>Type 4</td>
<td>Correlation</td>
<td>.189</td>
<td>-.401(**)</td>
<td>-.186</td>
</tr>
<tr>
<td></td>
<td>Sig. (2 tailed)</td>
<td>.053</td>
<td>.000</td>
<td>.057</td>
</tr>
</tbody>
</table>

** Correlation is significant at the 0.01 level (2-tailed).
* Correlation is significant at the 0.05 level (2-tailed).

Table 24 shows the correlation matrix for family history of cancer and CHD and the independent variables of Short IRI scores for the entire sample- each of these variables are also considered to be characteristics which make the subjects vulnerable to displaying an alarm reaction on learning of their Short IRI scores. As can be seen, the only correlation additional to those presented above is a positive one between CHD history and Type 4 scores (p<.05).

Table 24. Correlations between Short IRI Types and family history of cancer & CHD for full sample (N=105)

<table>
<thead>
<tr>
<th>Type</th>
<th>Cancer history</th>
<th>CHD history</th>
</tr>
</thead>
<tbody>
<tr>
<td>Type 1</td>
<td>Correlation</td>
<td>-.072</td>
</tr>
<tr>
<td></td>
<td>Sig. (2 tailed)</td>
<td>.468</td>
</tr>
<tr>
<td>Type 2</td>
<td>Correlation</td>
<td>.040</td>
</tr>
<tr>
<td></td>
<td>Sig. (2 tailed)</td>
<td>.686</td>
</tr>
<tr>
<td>Type 3</td>
<td>Correlation</td>
<td>-.083</td>
</tr>
<tr>
<td></td>
<td>Sig. (2 tailed)</td>
<td>.401</td>
</tr>
<tr>
<td>Type 4</td>
<td>Correlation</td>
<td>.169</td>
</tr>
<tr>
<td></td>
<td>Sig. (2 tailed)</td>
<td>.085</td>
</tr>
</tbody>
</table>

** Correlation is significant at the 0.01 level (2-tailed).
* Correlation is significant at the 0.05 level (2-tailed).
Table 25 shows the correlation matrix for variables of cancer and CHD risk perception and anxiety prior to the interview, and the independent variables of Short IRI scores for the entire sample- each of these self report variables are also considered to be characteristics which make the subjects vulnerable to displaying an alarm reaction on learning of their Short IRI scores. As can be seen, there are no additional significant correlations apart from a positive one between Type 3 scores and the risk perception of cancer (p<.05)

<table>
<thead>
<tr>
<th>Type</th>
<th>Correlation</th>
<th>Cancer risk perception</th>
<th>Anxious about cancer</th>
<th>CHD risk perception</th>
<th>Anxious about CHD</th>
</tr>
</thead>
<tbody>
<tr>
<td>Type 1</td>
<td>.133</td>
<td>-.004</td>
<td>-.005</td>
<td>.073</td>
<td></td>
</tr>
<tr>
<td></td>
<td>.177</td>
<td>.970</td>
<td>.959</td>
<td>.464</td>
<td></td>
</tr>
<tr>
<td>Type 2</td>
<td>.174</td>
<td>-.187</td>
<td>-.069</td>
<td>-.036</td>
<td></td>
</tr>
<tr>
<td></td>
<td>.076</td>
<td>.058</td>
<td>.487</td>
<td>.717</td>
<td></td>
</tr>
<tr>
<td>Type 3</td>
<td>.244(*)</td>
<td>-.119</td>
<td>-.082</td>
<td>-.119</td>
<td></td>
</tr>
<tr>
<td></td>
<td>.012</td>
<td>.228</td>
<td>.405</td>
<td>.229</td>
<td></td>
</tr>
<tr>
<td>Type 4</td>
<td>-.159</td>
<td>.099</td>
<td>.053</td>
<td>.061</td>
<td></td>
</tr>
<tr>
<td></td>
<td>.106</td>
<td>.318</td>
<td>.595</td>
<td>.539</td>
<td></td>
</tr>
</tbody>
</table>

** Correlation is significant at the 0.01 level (2-tailed).
* Correlation is significant at the 0.05 level (2-tailed).

Table 26 shows the correlations between personality variables used in this study and self report measures of cancer/CHD risk perception and anxiety for the full sample taken prior to the interview in the Health Psychology Questionnaire. The entire table is too large to display here- as such, Table 26 shows only the correlations which demonstrated a level of significance. As can be seen, there are positive correlations between Anxious about cancer and Anxious about CHD (p<.01); and Anxious about CHD and Likely to get CHD (p<.01); and a negative correlation between Neuroticism and Extroversion (p<.01).
### Table 26. Correlations between the different potential predictor variables for the full sample (N=105)

<table>
<thead>
<tr>
<th></th>
<th>Neuroticism</th>
<th>Anxious about cancer</th>
<th>Anxious about CHD</th>
<th>Likely to get CHD</th>
<th>Extroversion</th>
</tr>
</thead>
<tbody>
<tr>
<td>Neuroticism</td>
<td>Correlation</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Sig. (2 tailed)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Anxious about Cancer</td>
<td>Correlation</td>
<td>.127</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Sig. (2 tailed)</td>
<td>.192</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Anxious about CHD</td>
<td>Correlation</td>
<td>.101</td>
<td>.542**</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Sig. (2 tailed)</td>
<td>.299</td>
<td>.000</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Likely to get CHD</td>
<td>Correlation</td>
<td>-.005</td>
<td>.229</td>
<td>.653**</td>
<td>1</td>
</tr>
<tr>
<td>Sig. (2 tailed)</td>
<td>.959</td>
<td>.018</td>
<td>.000</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Extroversion</td>
<td>Correlation</td>
<td>-.251**</td>
<td>-.046</td>
<td>-.073</td>
<td>-.170</td>
</tr>
<tr>
<td>Sig. (2 tailed)</td>
<td>.009</td>
<td>.638</td>
<td>.456</td>
<td>.085</td>
<td></td>
</tr>
</tbody>
</table>

** Correlation is significant at the 0.01 level (2-tailed).
* Correlation is significant at the 0.05 level (2-tailed).

**Summary of correlations in the entire sample:**

In summary, Tables 21-26 show a pattern of correlations for the entire sample between the study variables that is consistent with Grossarth-Maticzek & Eysenck’s (1990) reports. There are significant positive correlations between Types 1, 2 and 3; and each of these significantly negatively correlate with Type 4 scores. In regards to the relationships between the Short IRI scores and personality measures of Neuroticism and Introversion/Extroversion, the current sample shows the same pattern as that reported by Grossarth-Maticzek & Eysenck (1990), i.e significant positive correlations between Types 1, 2 & 3 and Neuroticism scores; a significant negative correlation between Type 4 and Neuroticism scores; and a significant negative correlation between Type 1 and Extroversion scores, suggesting a positive correlation between Introversion and Type 1 scores. Apart from a positive correlation between Type 2 scores and Monitor scores, there are no significant correlations between Monitor and Blunter scores and the Short IRI scores. In regards to the Short IRI scores and family history of cancer or CHD, there are no significant correlations apart from a positive one between Type 2 scores and the risk of getting CHD. Apart from a positive correlation between Type 2 scores and Monitor scores, there are no significant correlations between Monitor and Blunter scores and the Short IRI scores. In regards to the Short IRI scores and family history of cancer or CHD, there are no significant correlations apart from a positive one between Type 2 scores and the risk of getting CHD.
3 scores and the risk perception of cancer. In regards to the Self Report measures of risk perception and illness anxiety, there are no correlations found between these variables and Short IRI Types, apart from a positive one between Type 3 scores and the risk perception of cancer.

The same relationships will now be ascertained for each of the interview conditions taken separately.

**Correlations for Full Interview condition subjects:**

A correlational analysis was conducted between Grossarth-Maticek’s Type scores for the Full Interview condition subjects. The same set of correlations are evident between these variables in the Full Interview condition as were found in the total sample. Types 1, 2, and 3 are positively correlated (all at the p<.01 level); and each of these are negatively correlated with Type 4 scores (each at the p<1 level, apart from Type 1 and Type 4 scores which are correlated at the p<.05 level). (The table can be viewed as Table 59 in Appendix 6).

A correlational analysis was conducted between personality variables (Neuroticism, Extroversion, Monitor, Blunter scores) and the Short IRI scores for subjects in the Full Interview condition only- each of these personality variables are also considered to be characteristics which make the subjects vulnerable to displaying an alarm reaction on learning of their Short IRI scores. A similar pattern of correlations emerge from the Full Interview condition subjects as does for the entire sample, the only difference being a non significant positive correlation between Monitor scores and IRI Type 2 scores in the Full Interview condition, whereas this was a significant correlation in the entire sample. (The table can be viewed as Table 60 in Appendix 6).

A correlational analysis was conducted between the Short IRI Types and the Short IRI scores and cancer history and CHD history in the family for the Full Interview sample. There are no additional significant correlations between the variables reported.(The table can be viewed as Table 61 in Appendix 6).
A correlational analysis was conducted between Short IRI scores and the self report variables of Cancer risk perception, CHD risk perception, Cancer anxiety, CHD anxiety taken prior to the interview for the Full Interview condition subjects. The pattern of correlations is the same as it is for the entire sample, with the only difference being a significant positive correlation between Likely to experience cancer and IRI Type 2 in the Full Interview condition, whereas this was a non significant correlation in the entire sample. (The table can be viewed as Table 62 in Appendix 6).

A correlational analysis was conducted between self report variables and personality variables for the Full Interview condition subjects. The only difference in the pattern of correlations observed between the same variables for the entire sample are a significant negative correlation between Likely to get CHD and Monitor scores (r= -.249*) in the Full Interview condition, whereas this was not evident in the entire sample. In addition, the entire sample showed a significant negative correlation between Extroversion and Neuroticism (r= -.236*), whereas this was not evident in the Full Interview condition. (The table can be viewed as Table 63 in Appendix 6).

Summary of correlations in the Full Interview Condition:
With few variations, the same pattern of responses can be seen in the Full Interview Condition as was evident in the entire sample, i.e. the responses were consistent with those reported by Grossarth-Maticek & Eysenck (1990); and the additional potential predictor variables demonstrated few significant correlations with the Short IRI scores.

Correlations for Self Administered interview condition subjects:
A correlational analysis was conducted between Grossarh-Maticek’s Type 1, 2, 3, 4 scores for the Self Administered condition subjects (n=40). As can be seen in Table 27, there are some minor differences in correlations between these variables from the entire sample to the Self Administered sample. Type 1 scores are positively correlated to Type 2 and Type 3 scores (p<.01 and p<.05 respectively), and negatively with Type 4 scores, although unlike the entire sample or the Full Interview condition, this fails to reach significance. Type 2 scores are positively correlated with Type 1 and Type 3 scores (both p<.01), and negatively correlated with Type 4 scores (p<.05). Type 3 scores are positively correlated with Type 1
and Type 2 scores (both p<.01), and negatively but not significantly correlated with Type 4 scores. Type 4 scores only show a negative correlation with Type 2 scores (p<.05). These correlations are in the same direction as those reported by Grossarth-Maticek & Eysenck (1990), differing only in levels of significance- in this regard, the Self Administered subjects differ from the entire sample and from the Full Interview subjects.

Table 27. Short IRI scale correlations for the Self Admin. condition subjects (n=40).

<table>
<thead>
<tr>
<th>Short IRI Type</th>
<th>IRI T1</th>
<th>IRI T2</th>
<th>IRI T3</th>
<th>IRI T4</th>
</tr>
</thead>
<tbody>
<tr>
<td>IRI T1</td>
<td>Correlation</td>
<td>1</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Sig. (2-tailed)</td>
<td>.</td>
<td></td>
<td></td>
</tr>
<tr>
<td>IRI T2</td>
<td>Correlation</td>
<td>.444(**)</td>
<td>1</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Sig. (2-tailed)</td>
<td>.004</td>
<td></td>
<td></td>
</tr>
<tr>
<td>IRI T3</td>
<td>Correlation</td>
<td>.399(*)</td>
<td>.454(**)</td>
<td>1</td>
</tr>
<tr>
<td></td>
<td>Sig. (2-tailed)</td>
<td>.011</td>
<td>.003</td>
<td></td>
</tr>
<tr>
<td>IRI T4</td>
<td>Correlation</td>
<td>-.261</td>
<td>-.399(*)</td>
<td>-.246</td>
</tr>
<tr>
<td></td>
<td>Sig. (2-tailed)</td>
<td>.104</td>
<td>.011</td>
<td>.127</td>
</tr>
</tbody>
</table>

** Correlation is significant at the 0.01 level (2-tailed).
* Correlation is significant at the 0.05 level (2-tailed).

Table 28 shows the correlations between personality variables for subjects in the Self Administered condition only. As there are differences between the Self Administered sample and the entire sample on these correlations, the correlations will be shown in Table 27 below. In the Self Administered condition, there is no significant correlation between IRI Type 1 and Neuroticism as there is in the entire sample. In the Self Administered sample, Type 1 scores significantly negatively correlate with Extroversion (p<.05), meaning that Type 1 scores correlate significantly with Introversion in a positive direction (as Extroversion and Introversion are polar opposite measures on the EPQ). And there is a positive but non significant correlation between Type 1 scores and Neuroticism. Type 2 scores positively correlate with Neuroticism scores (p<.01), and show a negative but non significant correlation with Extroversion scores. Type 3 scores show a negative but non-significant correlations with Type 4 and Extroversion scores, as well as positive but non significant
correlations with Neuroticism scores. Extroversion scores show a significant negative
correlation with Type 1 scores (p< .05), meaning that Introversion is positively correlated
with Type 1 scores. Extroversion also shows negative but non-significant correlations with
Type 2, Type 3 scores Neuroticism scores; and a positive but non significant correlation with
Type 4 scores. Neuroticism scores show a positive but non-significant correlation with Type
1 and Type 3 scores; a significant positive correlation with Type 2 scores (p< .01), and a
significant negative correlation with Type 4 scores (p< .05). It shows a negative but non
significant correlation with Extroversion scores. Finally, Monitor scores show a significant
positive correlation with Type 3 scores (p<.05). Again, the same pattern of correlations are
evident in the Self Administered subjects as are seen in the entire sample and in Grossarth-
Maticek & Eysenck’s (1990) reports, however unlike the entire sample or the Full Interview
condition, several of these correlations fail to reach levels of significance.

Table 28. Correlations of personality variables and Short IRI scores for Self Admin. Interview condition subjects (n=40).

<table>
<thead>
<tr>
<th></th>
<th>Extroversion</th>
<th>Neuroticism</th>
<th>Monitor score</th>
<th>Blunter score</th>
</tr>
</thead>
<tbody>
<tr>
<td>Type 1 Correlation</td>
<td>-.344(*)</td>
<td>.244</td>
<td>.127</td>
<td>.233</td>
</tr>
<tr>
<td>Sig. (2 tailed)</td>
<td>.030</td>
<td>.130</td>
<td>.436</td>
<td>.167</td>
</tr>
<tr>
<td>Type 2 Correlation</td>
<td>-.077</td>
<td>.429(**)</td>
<td>.313(*)</td>
<td>.051</td>
</tr>
<tr>
<td>Sig. (2 tailed)</td>
<td>.637</td>
<td>.006</td>
<td>.049</td>
<td>.755</td>
</tr>
<tr>
<td>Type 3 Correlation</td>
<td>-.052</td>
<td>.242</td>
<td>.006</td>
<td>.061</td>
</tr>
<tr>
<td>Sig. (2 tailed)</td>
<td>.749</td>
<td>.133</td>
<td>.971</td>
<td>.709</td>
</tr>
<tr>
<td>Type 4 Correlation</td>
<td>.148</td>
<td>-.366(*)</td>
<td>-.206</td>
<td>.006</td>
</tr>
<tr>
<td>Sig. (2 tailed)</td>
<td>.361</td>
<td>.020</td>
<td>.202</td>
<td>.969</td>
</tr>
</tbody>
</table>

** Correlation is significant at the 0.01 level (2-tailed).
* Correlation is significant at the 0.05 level (2-tailed).

A correlational analysis between Short IRI scores and cancer in family and CHD in family
was conducted for the Self Administered sample. Unlike the same analysis for the entire
sample, there are no significant correlations to report. (The table can be viewed as Table 64
in Appendix 6).
A correlational analysis between Short IRI Types and self report variables of cancer risk perception, CHD risk perception, cancer anxiety and CHD anxiety for the Self Administered interview condition subjects, taken in the Health Psychology Questionnaire, was conducted. As can be seen in Table 29, Likely to get cancer negatively correlates with Type 3 scores (p<.01); and Anxious about CHD negatively correlates with Type 3 scores (p<.05). This differs from the same analysis of the entire sample in that neither of these correlations were present in the latter, however there was a positive correlation between Likely to experience cancer and IRI Type 3 scores.

Table 29. Correlations between Short IRI Types and potential predictor variables for Self Admin condition subjects (cont) (n=40)

<table>
<thead>
<tr>
<th>Type 1</th>
<th>Correlation</th>
<th>Cancer risk perception</th>
<th>Anxious about cancer</th>
<th>CHD risk perception</th>
<th>Anxious about CHD</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Correlation</td>
<td>.049</td>
<td>.170</td>
<td>-.159</td>
<td>.108</td>
</tr>
<tr>
<td></td>
<td>Sig. (2 tailed)</td>
<td>.763</td>
<td>.301</td>
<td>.335</td>
<td>.514</td>
</tr>
<tr>
<td>Type 2</td>
<td>Correlation</td>
<td>.060</td>
<td>-.246</td>
<td>-.247</td>
<td>-.130</td>
</tr>
<tr>
<td></td>
<td>Sig. (2 tailed)</td>
<td>.714</td>
<td>.131</td>
<td>.130</td>
<td>.432</td>
</tr>
<tr>
<td>Type 3</td>
<td>Correlation</td>
<td>.008</td>
<td>-.019</td>
<td>-.440(**)</td>
<td>-.347(*)</td>
</tr>
<tr>
<td></td>
<td>Sig. (2 tailed)</td>
<td>.960</td>
<td>.909</td>
<td>.005</td>
<td>.030</td>
</tr>
<tr>
<td>Type 4</td>
<td>Correlation</td>
<td>-.147</td>
<td>.002</td>
<td>-.030</td>
<td>.035</td>
</tr>
<tr>
<td></td>
<td>Sig. (2 tailed)</td>
<td>.364</td>
<td>.992</td>
<td>.856</td>
<td>.830</td>
</tr>
</tbody>
</table>

** Correlation is significant at the 0.01 level (2-tailed).
* Correlation is significant at the 0.05 level (2-tailed).

Correlations were calculated between all of the following variables:- Neuroticism, extroversion, Monitor, Blunter, cancer history, CHD history, Likely to get Cancer, Likely to get CHD, Anxious about Cancer, Anxious about CHD) for the Self Administered interview condition subjects. Only one significant correlation was evident, between Anxious about CHD and Likely to get CHD (r=.517**). This differs from the same analysis of the entire sample in that positive correlations were also observed between Anxious about cancer and Anxious about CHD; and Extroversion and Neuroticism in the entire sample- these were not evident in the Self Administered sample.
Summary of correlations in the Self Administered Condition:

There were some differences in the pattern of correlations from the Self Administered interview condition and the entire sample, however most of these were in terms of levels of significance. The Self Administered subjects showed responses that were less significant than, although for the most part in the same direction as Grossarth-Maticek & Eysenck’s (1990) reports. In this tendency, it differed slightly from both the entire sample and the Full Interview condition subjects.

Section B) Study 1: The effect of risk prediction and interview condition:

The questions posed in the current study are:-

Study 1: The effects of a negative health prediction

- Question 1- when compared to subjects who receive a positive health prediction, do subjects who receive a negative health prediction show an increase in heart rate on presentation of a negative health prediction?
- Question 2- when compared to subjects who receive a positive health prediction, do subjects who receive a negative health prediction show an increase in self report measures of disease risk perception and anxiety on presentation of a negative health prediction?
- Question 3- is the current experimenter able to predict subject’s eventual Short IRI scores (in terms of health risk or positive health prediction) on the basis of life discussion?

Study 1: Question 1- Responses of Heart Rate to risk information:

For subjects in the Full Interview condition, does being informed of a health risk prediction create an alarm reaction (measured by Changes in Mean Heart Rate) when compared to the reaction created for subjects who are informed of a positive health prediction?

Crosstabs for Gender:

In order to establish if gender is a factor influencing the receipt of risk information, subjects were divided into their gender groupings in relation to the three definitions of receipt of risk
information from Short IRI scores (see Methodology for description of the three definitions used).

Table 30 shows the amount of subjects in the Full Interview condition who received risk information in the course of the interview (according to definition i of risk information (airi_c), i.e. their risk profile was determined by their highest type score from amongst Type 1, Type 2, Type 3 & Type 4) - these figures are shown by Gender. As can be seen in Table 30, there were considerably more females in the sample than males, but the proportions of males and females receiving risk information was similar - 12.0% of females obtained risk information from their Short IRI score, and 13.3% of males also received risk information.

Table 30 also shows the amount of subjects in the Full Interview condition who received risk information (according to definition ii) of risk information, i.e. their Type 1 or Type 2 scores were within 1 point of their Type 4 score) in the course of the interview. The proportions of males and females receiving risk information again similar, as seen with definition i) of risk information - 17.2% of women obtained risk information according to this definition, while only 18.2% of men received risk information.

Table 30 also shows the amount of subjects in the Full Interview condition who received risk information (according to definition iii) of risk information, i.e. their Type 1 or Type 2 scores were greater than or equal to 5/10) in the course of the interview - these figures are shown by Gender. The proportions of males and females receiving risk information was not as similar as seen with both of the other definitions of risk information - 26.0% of women obtained risk information according to this definition, while 40% of men received risk information.

As can be seen in Table 30, none of the Chi-Squared statistics reached a level of significant difference, suggesting that there was no difference within the sample based on gender on any of the definitions of receipt of risk information.
Table 30. Crosstabulations for Full Interview subjects of receipt of risk information by Gender showing three methods for defining receipt of risk information (n=65).

<table>
<thead>
<tr>
<th>Definition of risk</th>
<th>Gender</th>
<th>No N</th>
<th>Yes N</th>
<th>Chi-Squared statistics</th>
</tr>
</thead>
<tbody>
<tr>
<td>i) airi_c</td>
<td>Male</td>
<td>13</td>
<td>2</td>
<td>Chi Sq=.019a</td>
</tr>
<tr>
<td></td>
<td>Female</td>
<td>44</td>
<td>6</td>
<td>df= 1</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Sig (2 sided)= .890</td>
</tr>
<tr>
<td>(highest Type score)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>ii) rri1</td>
<td>Male</td>
<td>9</td>
<td>2</td>
<td>Chi Sq=.005a</td>
</tr>
<tr>
<td></td>
<td>Female</td>
<td>24</td>
<td>5</td>
<td>df= 1</td>
</tr>
<tr>
<td>(T1/T2 scores within 1 of T4 score)</td>
<td></td>
<td></td>
<td></td>
<td>Sig (2 sided)= .944</td>
</tr>
<tr>
<td>iii) rri2</td>
<td>Male</td>
<td>9</td>
<td>6</td>
<td>Chi Sq= 1.093a</td>
</tr>
<tr>
<td></td>
<td>Female</td>
<td>37</td>
<td>13</td>
<td>df= 1</td>
</tr>
<tr>
<td>(risk score =&gt;5/10)</td>
<td></td>
<td></td>
<td></td>
<td>Sig (2 sided)= .296</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>a. 1 cells (25.0%) have expected count less than 5.</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Table 31 shows the amount of subjects in the Self Administered condition who were assessed to be in a health risk category according to definition i of risk information (airi_c), ie. their risk profile was determined by their highest type score from amongst Type 1, Type 2, Type 3 & Type 4)- these figures are shown by Gender . As can be seen in Table 31, there were considerably more females in the sample than males, but the proportions of males and females being assessed as at risk of poor health according to their Short IRI scores were similar- 17.2% of females obtained highest Type 1 or Type 2 scores of their Short IRI scores, and 18.2% of males also received risk information. Subjects in the Self Administered interview condition were not given their Short IRI scores, so these figures do not represent the receipt of risk information- they merely show the scores which Self Administered subjects did obtain and their assessed risk status according to the Short IRI. As they did not receive their Short IRI scores, the alternate definitions of receipt of risk information are not relevant to the Self Administered subjects and as such have not been calculated.
Table 31. Crosstabulations for Self Administered subjects of receipt of risk information by Gender showing airi_c definition of receipt of risk information (n=40).

<table>
<thead>
<tr>
<th>Definition of risk</th>
<th>Gender</th>
<th>No</th>
<th>Yes</th>
<th>Chi-Squared statistics</th>
</tr>
</thead>
<tbody>
<tr>
<td>i) airi_c</td>
<td>Male</td>
<td>9</td>
<td>2</td>
<td>Chi Sq= .005a</td>
</tr>
<tr>
<td>(highest Type score)</td>
<td>Female</td>
<td>24</td>
<td>5</td>
<td>df= 1</td>
</tr>
</tbody>
</table>

a. 1 cells (25.0%) have expected count less than 5. The minimum expected count is 1.93

Summary of Crosstabs for Gender:
Tables 30 and 31 demonstrate that there are no significant differences in the rates of males and females who received risk information according to the three definitions used for the Full Interview subjects; and the assessment of risk status according to definition of risk used with the Self Administered subjects.

Crosstabulations for Age Categories:
Table 32 shows the amount of subjects in the Full Interview condition who received risk information (according to definition i of risk information (airi_c), i.e. their risk profile was determined by their highest type score from amongst Type 1, Type 2, Type 3 & Type 4)- these figures are shown by Age. As can be seen in Table 32, 19.0 % of the younger age group (under 30) obtained risk information according to their Short IRI scores; while 14.3% of the middle aged group (30-49) received risk information; and no subjects in the 50+ age group received risk information according to this definition of risk.

Table 32 also shows the amount of subjects in the Full Interview condition who received risk information (according to definition ii) of risk information, i.e their Type 1 or Type 2 scores were within 1 point of their Type 4 score) in the course of the interview- these figures are shown by Age category. In the under 30 age group, 23.8 % obtained risk information according to this definition; 14.3% of the 30-49 age group received risk information; and 6.3% of the 50+ age group received risk information. According to the definition of health risk information used in this table, one subject in the 50+ age group received risk information in that their Type 1 or Type 2 score was within one point of their Type 4 score. The subject was given this information as they nominated an interest in learning of it; and s/he was not excluded for ethical reasons as s/he did not present as being unable to psychologically cope
with the news. This definition of risk information is still subject to the selection bias as detailed in the previous section as elderly people were prevented from learning of this information if they impressed the interviewer as being psychologically too vulnerable to receive it. The data of 4 subjects (assessed by the Short IRI as fulfilling this criteria of being at health risk) was not available for this analysis as they were not given their Short IRI scores for this ethical reason.

Table 32 also shows the amount of subjects in the Full Interview condition who received risk information (according to definition iii) of risk information, i.e their Type 1 or Type 2 scores were greater than or equal to 5/10) in the course of the interview- these figures are shown by Age category. In the under 30 age group, 42.9 % of subjects received risk information according to this definition; 28.6% in the 30-49 age group received risk information; and 12.5% of the 50+ age group received risk information. According to the definition of health risk information used here, 2 subjects in the 50+ age group received risk information in that their Type 1 or Type 2 score was greater than or equal to 5/10. The subjects were given this information as they nominated an interest in learning of it; and they were not excluded for ethical reasons as they did not present as being unable to psychologically cope with the news. This definition of risk information is still subject to the selection bias as detailed in the previous section as elderly people were prevented from learning of this information if they impressed the interviewer as being psychologically too vulnerable to receive it. The data of 6 subjects (assessed by the Short IRI as fulfilling this criteria of being at health risk) was not available for this analysis as they were not given their Short IRI scores for ethical reasons.

From viewing Table 32, it can be seen that age category does not influence the receipt of risk information in the Full Interview sample.
Table 32. Crosstabulations for Full Interview subjects of receipt of risk information by Age Category showing three methods for defining receipt of risk information (n=65).

<table>
<thead>
<tr>
<th>Definition of risk</th>
<th>Age Category</th>
<th>No</th>
<th>Yes</th>
<th>Chi-Squared statistics</th>
</tr>
</thead>
<tbody>
<tr>
<td>i) airi_c</td>
<td>Under 30</td>
<td>17</td>
<td>19</td>
<td>Chi Sq= 3.231a</td>
</tr>
<tr>
<td>(highest Type score)</td>
<td>30-49</td>
<td>24</td>
<td>14</td>
<td>Df= 2</td>
</tr>
<tr>
<td></td>
<td>50 and over</td>
<td>16</td>
<td>0</td>
<td>Sig (2 sided)= .199</td>
</tr>
<tr>
<td>ii) rrl1</td>
<td>Under 30</td>
<td>16</td>
<td>23</td>
<td>Chi Sq= 2.197b</td>
</tr>
<tr>
<td>(T1/T2 scores within 1 of T4 score)</td>
<td>30-49</td>
<td>24</td>
<td>14</td>
<td>Df= 2</td>
</tr>
<tr>
<td></td>
<td>50 and over</td>
<td>15</td>
<td>6</td>
<td>Sig (2 sided)= .333</td>
</tr>
<tr>
<td>iii) rrl2</td>
<td>Under 30</td>
<td>12</td>
<td>42</td>
<td>Chi Sq= 4.056a</td>
</tr>
<tr>
<td>(risk score =&gt;5/10)</td>
<td>30-49</td>
<td>20</td>
<td>28</td>
<td>Df= 2</td>
</tr>
<tr>
<td></td>
<td>50 and over</td>
<td>14</td>
<td>12</td>
<td>Sig (2 sided)= .132</td>
</tr>
</tbody>
</table>

a. 1 cells (25.0%) have expected count less than 5.
b. 3 cells (50%) have expected count less than 5.

Table 33 shows the amount of subjects in the Self Administered condition who were assessed to be in a health risk category according to definition i of risk information (airi_c), ie. their risk profile was determined by their highest type score from amongst Type 1, Type 2, Type 3 & Type 4)- these figures are shown by Age. As can be seen in Table 33, the youngest and the oldest age groups were assessed as having the most risk scores according the Short IRI (airi_c definition): 28.6% of subjects under the ages of 30 obtained highest Type 1 or Type 2 scores of their Short IRI scores, as did 21.4% of people in the 50+ age group; while 0% of subjects in the 30-49 age group were assessed as being in the risk group. Subjects in the Self Administered interview condition were not given their Short IRI scores, so these figures do not represent the receipt of risk information- they merely show the scores which Self Administered subjects did obtain and their assessed risk status according to the Short IRI.
<table>
<thead>
<tr>
<th>Definition of risk</th>
<th>Gender</th>
<th>No</th>
<th>Yes</th>
<th>Chi-Squared statistics</th>
</tr>
</thead>
<tbody>
<tr>
<td>i) airi_c</td>
<td>Under 30</td>
<td>10</td>
<td>4</td>
<td>Chi Sq= 3.884</td>
</tr>
<tr>
<td></td>
<td>30-49</td>
<td>12</td>
<td>0</td>
<td>df= 2</td>
</tr>
<tr>
<td></td>
<td>50 and over</td>
<td>11</td>
<td>3</td>
<td>Sig (2 sided)= .143</td>
</tr>
</tbody>
</table>

Summary of Crosstabs for Age:
Tables 32 and 33 demonstrate that there are no significant differences in regards to age categories and the receipt of risk information according to the three definitions used for the Full Interview subjects; and no significant differences in regards to age categories and the assessment of risk status according to definition of risk used with the Self Administered subjects.

Correlations of study variables and Changes in Mean Heart Rates:
The tables in the following section show the correlations between the Changes in Mean Heart Rates variables for subjects in the Full Interview condition and variables which are hypothesized to have a potential effect on the Changes in Heart Rate measures.

The mean heart rates for each subject from two discrete time frames within the Full Interview condition were used in order to calculate Changes in Mean Heart Rates in response to the receipt of Short IRI scores (entailing either health risk information, or positive health information). The time frames were:-

- **time1**: pre-score given- mean heart rate for each subject during the initial phase of the Full Interview, taking in the description of the study topic area and the discussion of subjects’ positive and negative life experiences and their typical responses to these.
- **time2**: post-score given- mean heart rate for each subject during the Full Interview condition, after the receipt of Short IRI scores, until the point where subjects were again asked the self report questions and the debrief was commenced.
As such, Changes in Mean Heart Rates as a function of receipt of Short IRI scores and their interpretation can be calculated simply by subtracting the mean heart rates of time1 from time2. A negative time2-time1 score means that the subject’s heart rate went down in the interview segment when they were given their Short IRI scores in comparison to the first segment of the interview. Conversely, a positive time2-time1 score means that the subject’s heart rate went up in the interview segment when they were given their Short IRI scores in comparison to the first segment of the interview.

Table 33 presents correlations between Changes in Mean Heart Rate measures and the self report measures taken prior to the interview in the Health Psychology Questionnaire which were mailed back to the experimenter by the subjects. Subjects’ answers to these questions are not affected at all by the interview context.

These self report measures of risk perception and anxiety are:-

- Subjects’ perception of their likelihood to experience cancer (q10a_i)
- Subjects’ levels of anxiety about the prospects of obtaining cancer (q10a_ii)
- Subjects’ perception of their likelihood to experience CHD (q11a_i)
- Subjects’ levels of anxiety about the prospects of obtaining CHD (q11a_ii)

Table 33 shows that there are no significant correlations between Changes in Mean Heart Rate (time2-time1) for Full Interview subjects and the self report variables listed. There can be seen a positive correlation between the Anxious about Cancer and Anxious about CHD variables (p<.01). There is also a positive correlation between the Likely to get CHD variable and the Anxious about CHD variable (p<.01).
Table 33. Correlations between changes in mean heart rate scores and variables of risk perception and anxiety for Full Interview subjects taken in the Health Psychology Questionnaire (n=65)

<table>
<thead>
<tr>
<th></th>
<th>Change in mean Heart Rate time2-time1</th>
<th>Likely to get cancer</th>
<th>Anxious about cancer</th>
<th>Likely to get CHD</th>
<th>Anxious about CHD</th>
</tr>
</thead>
<tbody>
<tr>
<td>Likely to get cancer</td>
<td>Correlation</td>
<td>-.050</td>
<td>1</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Sig. (2-tailed)</td>
<td>.695</td>
<td>.</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Anxious about cancer</td>
<td>Correlation</td>
<td>.031</td>
<td>-.024</td>
<td>1</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Sig. (2-tailed)</td>
<td>.805</td>
<td>.806</td>
<td>.</td>
<td></td>
</tr>
<tr>
<td>Likely to get CHD</td>
<td>Correlation</td>
<td>-.115</td>
<td>-.098</td>
<td>.163</td>
<td>1</td>
</tr>
<tr>
<td></td>
<td>Sig. (2-tailed)</td>
<td>.362</td>
<td>.317</td>
<td>.095</td>
<td>.</td>
</tr>
<tr>
<td>Anxious about CHD</td>
<td>Correlation</td>
<td>-.024</td>
<td>-.114</td>
<td>.533(**)</td>
<td>.516(**)</td>
</tr>
<tr>
<td></td>
<td>Sig. (2-tailed)</td>
<td>.850</td>
<td>.244</td>
<td>.000</td>
<td>.000</td>
</tr>
</tbody>
</table>

** Correlation is significant at the 0.01 level (2-tailed).

Table 34 shows the correlations between Changes in Mean Heart Rate scores for Full Interview subjects and family history variables. These family history variables measure the number of family members with an incident of the known hereditary cancers and the known hereditary CHD's. As can be seen in Table 34, there are no significant correlations between these variables, although the correlation between Number of family members with CHD and the Number of family with Cancer is approaching a level of significance (with a p value of -.059).
Table 34. Correlations between changes in mean heart rate scores and demographic variables for Full Interview subjects (n=65).

<table>
<thead>
<tr>
<th></th>
<th>Change in Mean Heart Rate</th>
<th>Number of family with cancer</th>
<th>Number of family with CHD</th>
</tr>
</thead>
<tbody>
<tr>
<td>Number of family with cancer</td>
<td>Correlation</td>
<td>-.183</td>
<td>1</td>
</tr>
<tr>
<td></td>
<td>Sig. (2-tailed)</td>
<td>.145</td>
<td>.</td>
</tr>
<tr>
<td>Number of family with CHD</td>
<td>Pearson Correlation</td>
<td>-.235</td>
<td>-.059</td>
</tr>
<tr>
<td></td>
<td>Sig. (2-tailed)</td>
<td>.059</td>
<td>.637</td>
</tr>
</tbody>
</table>

Table 35 shows the correlations between changes in Mean Heart Rate scores for Full Interview subjects and other personality variables. These are:- Neuroticism scores; Extroversion scores; Monitor scores; Blunter scores. As can be seen, there are no significant correlations to report.

Table 35 Correlations between changes in mean heart rate scores and personality variables which are potential predictors (n=65).

<table>
<thead>
<tr>
<th></th>
<th>Neuroticism</th>
<th>Extroversion</th>
<th>Monitor Score</th>
<th>Blunter Score</th>
</tr>
</thead>
<tbody>
<tr>
<td>mean heart rate change t2-t1</td>
<td>Correlation</td>
<td>-.151</td>
<td>.025</td>
<td>-.148</td>
</tr>
<tr>
<td></td>
<td>Sig. (2-tailed)</td>
<td>.229</td>
<td>.844</td>
<td>.240</td>
</tr>
</tbody>
</table>

Summary of correlations of study variables and Changes in Mean Heart Rates:
Changes in Mean Heart Rate scores did not significantly correlate with any of the other study variables, including the other dependent variables (Self Report measures).

Univariate ANOVA’s for Heart Rate Responses:
In order to answer the question posed above, Univariate ANOVA’s were conducted for Changes in Mean Heart Rate, comparing those subjects in the Full Interview condition who
received health risk information with those subjects who received a positive health prediction. If there are found to be significant differences in the Changes in Heart Rate measures of subjects, based on whether they receive health risk or health prone information, this will be taken as support for the hypothesis that receiving risk information creates an alarm response in subjects who receive it.

On conducting this analysis, it was detected that an outlier exists in the time2-time1 score, as can be seen in the scatterplot (displayed as figure 7 in Appendix 6). This subject obtained a Change in Mean Heart Rate score of -17.71, compared to the sample mean for this variable of -3.21, with a sd of 3.04.

Controlling for the outlier:
This outlier obviously skews the statistics for the rest of the sample. The time2-time1 value was -17.71, whereas the nearest value from another subject was -9. From an analysis of the heart rate chart of Subject 90, it could clearly be seen that this large decrease in average heart rate scores was the result not of a large drop in heart rate in the post-score given (time2) timeframe of the interview, but resulted from an extremely elevated heart rate score in the pre-score given (time1) timeframe of the interview. Further analysis revealed that this high mean score was derived primarily in the segment of time1 in which the subject was required to discuss positive and negative life events. While no individual records were kept detailing the actual issues discussed by subjects in this segment, it is noted that several subjects discussed catastrophic life events such as fatal car accidents; loss of children; loss of siblings, partners and parents; marital break-ups; financial ruination, etc. It is likely that Subject 90 was one such subject and that the high peaks in heart rate (which forced up his mean heart rate for time1) was a reflection of him discussing such traumatic life events. This is further suggested by the observation that in the segment of time1 timeframe when the Short IRI questions were being administered, his heart rate showed a steady decline with few dramatic peaks; and in the post-score given (time2 timeframe), his heart rate still demonstrated a steady decline- as such, it can be concluded that the extremely high mean heart rate score for the time1 timeframe is directly a result of the unique feature of that segment of the interview, ie. discussing traumatic life events. As this study is not directly interested in the heart rate
effects of discussing traumatic life events per se, the high heart rate peaks in time1 are purely peripheral. As such, a decision has been made to control for the effects on heart rate of the discussion of these life events, so that the time2-time1 score may be a more accurate reflection of the effects of answering the Short IRI questions and then learning of his IRI score. It is also noteworthy that Subject 90 is one of the few subjects in this study who did receive risk information according to his Short IRI scores- considering the low amount of ‘cases’, it was decided to leave Subject 90’s data in the sample for analysis.

As a method of eliminating the extreme effects of this outlier, the following strategy has been employed. The highest peaks of Subject 90’s heart rate measures from the time1 timeframe have been modified, with any heart rate score over 116 being eliminated from the calculation of mean heart rate for this segment of the interview. One hundred and sixteen was chosen as this is the next highest heart rate peak in the time1 timeframe, displayed by Subject 88. Recalculating Subject 90’s mean heart rate score for the time1 timeframe without any peaks over 116 produced a mean of 103.83. Even utilizing this method, Subject 90’s mean heart rate score for the time1 time frame is substantially higher than any other subject’s (with Subject 75 showing the next highest mean heart rate score at 90.35). With this new time1 mean heart rate score for Subject 90, the time2-time1 score comes to -13.54. As such, this is still a very high score but will not skew the means involved.

*The ANOVA analysis.*

Univariate ANOVA tests were run on the dependent variable (Changes in Mean Heart Rate measures) from time1 to time2 in the Full Interview condition using each of the three definitions of health risk information according to the Short IRI scores. The following tables compare the Changes in Mean Heart Rate scores for subjects who were given positive health predictions according to the Short IRI (ie. Type 4 scores) compared to those subjects who were given Short IRI scores which entailed some health risk information (according to the particular definition used, as detailed below).

Table 36 shows the comparison of Changes in Mean Heart Rate scores for Full Interview subjects who were given their Short IRI scores. This definition of ‘risk information’ (airi_c)
entailed the subjects being told that their highest Type score on the Short IRI (ie. Types 1-4) depicted their proneness according to Grossarth-Maticek’s (1980) claims. As can be seen in Table 36, when this definition of risk information is used to compare subjects who received risk information and subjects who received health prone information, the differences in the mean heart rate changes from pre-news to post–news shows no difference of statistical significance. Both groups of subject a show negative mean score for the time2– time1 value. This means that their heart rates went down in the interview segment when they were given their Short IRI scores (time2) from their time1 heart rates. Subjects who received risk information demonstrated a larger decrease in mean changes in heart rate in the time2 segment of the interview (post receipt of health predictive information) compared to those subjects who received a health prone prediction, however this difference failed to achieve a level of statistical significance, with a p value of only .182.

Table 36. Summary of Univariate ANOVA statistics for changes in heart rate measures for Full Interview subjects (airi c) (n=65)

<table>
<thead>
<tr>
<th>Information received</th>
<th>S given health prone info</th>
<th>S given health risk information (air)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mean changes in heart rate</td>
<td>-2.97</td>
<td>-4.37</td>
</tr>
<tr>
<td>Standard Deviations</td>
<td>2.31</td>
<td>5.00</td>
</tr>
<tr>
<td>N</td>
<td>57</td>
<td>8</td>
</tr>
</tbody>
</table>

F= 1.82, df = 1,63; P= .182

Table 37 shows the comparison of Changes in Mean Heart Rate scores for Full Interview subjects who were given their Short IRI scores and consequently received health prone or health risk information. This definition of ‘risk information’ (rrli) entailed subjects processing the information that their Short IRI profile depicts one of their risk prone scores (Type 1 or Type 2) as being within only one point of their health prone (Type 4) score. Both groups of subjects show a negative changes in mean score for the time2 – time1 value- this means that their heart rates went down in the interview segment when they were given their Short IRI scores (time2) from their time1 heart rates. Subjects who received risk information demonstrated a larger decrease in heart rate in the Time 2 segment of the interview (post receipt of health predictive information) compared to those subjects who received a health prone prediction, however this failed to achieve a level of statistical significance.
Table 37. Summary of Univariate ANOVA statistics for changes in heart rate measures for Full Interview subjects (rri1) (n=65)

<table>
<thead>
<tr>
<th>Information received</th>
<th>S given health prone info</th>
<th>S given health risk information (rri1)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mean of changes in heart rate</td>
<td>-3.08</td>
<td>-3.50</td>
</tr>
<tr>
<td>Standard Deviations</td>
<td>2.28</td>
<td>4.78</td>
</tr>
<tr>
<td>N</td>
<td>55</td>
<td>10</td>
</tr>
<tr>
<td>F= .199, df = 1,63; P= .66</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Table 38 shows the comparison of Changes in Mean Heart Rate scores for Full Interview subjects who were given their Short IRI scores and consequently received health prone or health risk information. This definition of ‘risk information’ (rri2) entailed subjects processing the information that their Short IRI profile depicts one of their risk prone scores (Type 1 or Type 2) as being equal to or higher than 5/10. As can be seen in Table 38, when this definition of risk information is used to compare subjects who received risk information and subjects who received health prone information, the differences in the mean heart rate changes from pre-news to post–news shows a difference which has a significance level of only p= 0.721. Both groups of subjects show negative mean scores for the time2 – time1 value- this means that their heart rates went down in the interview segment when they were given their Short IRI scores (time2) from their time1 heart rates. Subjects who received risk information demonstrated a larger decrease in heart rate in the time2 segment of the interview (post receipt of health predictive information) compared to those subjects who received a health prone prediction, however this failed to achieve a level of statistical significance.

Table 38. Summary of Univariate ANOVA statistics for changes in heart rate measures for Full Interview subjects (rri2) (n=65)

<table>
<thead>
<tr>
<th>Information received</th>
<th>S given health prone info</th>
<th>S given health risk information (rri2)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mean changes in heart rate</td>
<td>-3.06</td>
<td>-3.33</td>
</tr>
<tr>
<td>Standard Deviations</td>
<td>2.34</td>
<td>3.64</td>
</tr>
<tr>
<td>N</td>
<td>46</td>
<td>19</td>
</tr>
<tr>
<td>F= .13, df = 1,63; P= .721</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
Summary of ANOVA analyses comparing heart rate responses of subjects.

As can be seen from Tables 37-38, the differences in Changes in Mean Heart Rates between subjects who received risk information and subjects who obtained health prone information did not significantly differ—this finding held for each of the three definitions of risk information used. Each of the groups of subjects showed decreases in their Changes in Mean Heart Rate scores, but their values on these variables did not significantly differ.

Univariate ANOVA adjusting for age and gender:

A univariate between-groups analysis of variance was conducted to explore the relative impacts of age, gender and the receipt of risk information on Changes in Mean Heart Rate measures for Full Interview subjects. This analysis used a Type I hierarchical decomposition sum of squares method in which each term is adjusted for only the term that precedes it in the model, i.e., age and gender before the risk variables. Each of the three definitions of receipt of risk information was used in the analysis. Subjects were divided into three groups according to their age (Group 1: under 30 years old; Group 2: 30-49 years old; Group 3: over 50 years old), and they were grouped according to gender (male or female). In calculating the significance levels for differences in Changes in Mean Heart Rates according to these groupings, it was found that there were no statistically significant differences in the results. Table 39 shows the summary of Univariate ANOVA statistics for comparison in changes in heart rate measures (risk information received V’s health prone information received, with ‘risk information’ being defined by the airi_c, rri1 and rri2 definitions of receipt of risk information) for Full Interview subjects after adjusting for age and gender. It can be seen that the addition of age and gender to the model do not produce p values that reach significance.
Summary of Univariate ANOVA statistics for changes in heart rate measures for Full Interview subjects after adjusting for age and gender.

<table>
<thead>
<tr>
<th></th>
<th>F</th>
<th>Df</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>airi_c</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Age</td>
<td>.494</td>
<td>1,61</td>
<td>.485</td>
</tr>
<tr>
<td>Gender</td>
<td>.187</td>
<td>1,61</td>
<td>.667</td>
</tr>
<tr>
<td>Risk</td>
<td>2.336</td>
<td>1,61</td>
<td>.132</td>
</tr>
<tr>
<td><strong>rri1</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Age</td>
<td>.479</td>
<td>1,61</td>
<td>.492</td>
</tr>
<tr>
<td>Gender</td>
<td>.181</td>
<td>1,61</td>
<td>.672</td>
</tr>
<tr>
<td>Risk</td>
<td>.364</td>
<td>1,61</td>
<td>.549</td>
</tr>
<tr>
<td><strong>rri2</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Age</td>
<td>.478</td>
<td>1,61</td>
<td>.492</td>
</tr>
<tr>
<td>Gender</td>
<td>.181</td>
<td>1,61</td>
<td>.672</td>
</tr>
<tr>
<td>Risk</td>
<td>.263</td>
<td>1,61</td>
<td>.610</td>
</tr>
</tbody>
</table>

Summary of ANOVA analysis adjusting for age and gender.
The Changes in Mean Heart Rates shown by those subjects who received risk information did not significantly differ when compared to subjects who received health prone information when taking into account age and gender.

Study 1: Question 2- Responses of Self Report measures to risk information:
For subjects in both Self Administered and Full Interview conditions, are Changes in Self Report measures of illness risk perception and anxiety likely to differ according to the interview type? Are those subjects in the Full Interview condition more likely to complete the interview with a heightened sense of risk and anxiety about cancer and/or CHD when compared to subjects in the Self Administered condition? (the former perhaps receiving risk information, while the latter will not as no such information was given).

The self report measures of interest are:-

i) Perceived Risk for Cancer
ii) Cancer anxiety
iii) Perceived Risk for CHD
iv) CHD anxiety

305
Table 40 shows the means and standard deviation scores for subject’s Self Report measures of Perceived-Risk for Cancer and Cancer-Anxiety, as well as Perceived-Risk for CHD and CHD-Anxiety for the entire sample. These measures were taken on two occasions, time1 (pre-receipt of scores) and time2 (post-receipt of scores). Each of the time1 and time2 measures have been calibrated into a change measure- a negative change measure indicates that subjects ratings of, say cancer anxiety, decreased from time1 to time2. Conversely, a positive change measure would indicate that the subject’s cancer anxiety increased from time1 to time2. Clearly there is a significant main-effect for time. As can be seen, all of the variables in Table 40 show a statistically significant decrease from time1 to time2. As can be seen from the means in Table 40, the sample as a whole significantly decreased in Perceived-Risk for Cancer, Cancer-Anxiety, Perceived-Risk for CHD, and CHD-Anxiety. Note that the mean change in Self Report measures are negative to reflect the decreases from time1 to time2.

Table 40. Descriptive statistics for Perceived-Risk and Anxiety for time1 and time2 (N=105)

<table>
<thead>
<tr>
<th></th>
<th>Time1</th>
<th>Time2</th>
<th>Difference</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Mean</td>
<td>SD</td>
<td>Mean</td>
</tr>
<tr>
<td>Perceived-Risk for Cancer</td>
<td>4.23</td>
<td>2.15</td>
<td>3.70</td>
</tr>
<tr>
<td>Cancer anxiety</td>
<td>2.95</td>
<td>2.10</td>
<td>2.61</td>
</tr>
<tr>
<td>Perceived-Risk for CHD</td>
<td>3.92</td>
<td>2.37</td>
<td>3.51</td>
</tr>
<tr>
<td>CHD anxiety</td>
<td>2.53</td>
<td>2.10</td>
<td>2.19</td>
</tr>
</tbody>
</table>

Table 41 shows the results of paired t tests conducted to establish if there are differences in the perceived risk of cancer and the perceived risk of CHD at time 1, as well as the cancer anxiety and the CHD anxiety scores at time 1 for the entire sample.

Table 41. Comparison of mean cancer and CHD Perceived Risk scores and Anxiety levels at Time 1 (N=105)

<table>
<thead>
<tr>
<th></th>
<th>Cancer</th>
<th>CHD</th>
<th>Difference</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Mean</td>
<td>SD</td>
<td>Mean</td>
</tr>
<tr>
<td>Time 1 Anxiety level</td>
<td>2.95</td>
<td>2.10</td>
<td>2.53</td>
</tr>
<tr>
<td>Time 1 Perceived Risk</td>
<td>4.23</td>
<td>2.15</td>
<td>3.92</td>
</tr>
</tbody>
</table>

306
From these results, there appears to be greater salience for cancer than for CHD in the sample. Subjects at the commencement of the interviews perceived themselves to be at a greater risk of cancer (4.23) compared to CHD (3.92), however this difference failed to reach a level of significance. Cancer does appear to be the more feared condition, with mean Cancer Anxiety scores being 2.95, and mean CHD Anxiety scores being 2.53 at the commencement of the interviews- this difference did obtain a level of significance at the .05 level. It is also interesting to note that the two Perceived Risk measures showed the largest differences between time1 and time2, with the largest decrease being for Perceived-Risk for Cancer.

Univariate ANOVA for Changes in Self Report Measures:
Univariate ANOVA tests were run on Changes in Self Report measures from time1 to time2 in the Self Administered condition and for the Full Interview condition using each of the three definitions of health risk information according to the Short IRI scores (airi_c; rri1; rri2).

Of interest to this study are the changes in these self report measures which occurred in the subjects from time1 (beginning of the interview) to time 2 (on completion of the interview). For Self Administered subjects, the interview was concluded when the completed questionnaire was returned by the subject to the interviewer. This differed from the Full Interview subjects in that their interview concluded at corresponding point where the current author proposes Grossarth-Maticzek’s actual full interviews concluded, i.e with subjects learning of their Short IRI scores. The conclusion point for each of the interview conditions is different, as they both entailed different processes (the effects of these differences being the subject of this study).

The following tables compare the Changes from time1 to time2 in Self Report measures of cancer and CHD risk and anxiety for Full Interview subjects who were given positive health predictions according to the Short IRI (ie. Type 4 scores) compared to those subjects who were given Short IRI scores which entailed some health risk information (according to the particular definition used). As the Self Administered subjects were not informed of their
Short IRI risk status during the course of the interview, the different definitions of risk information hold no relevance- as such, there is no need for a table showing the differences in mean scores for each of the different definitions of risk information for the Self Administered subjects as no such calculations were made.

Changes in Self Reported Cancer Risk Perception.
Table 42 shows the comparison of changes in perceived life time risk for cancer for Full Interview subjects who were given their Short IRI scores according to each of the definitions of risk information. As can be seen in Table 42 there is no significant difference in subject’s changes in perceived life time risk of cancer with each of the definitions of risk information. Subjects who received risk information and those who received health prone information showed a decrease in perception of cancer risk. It is interesting to note that those subjects who were given risk information showed less of a decrease in cancer risk perception (they retained more of their initial cancer risk perception than those who received health prone information), however these differences failed to reach a level of significance.

Table 42. Summary of Univariate ANOVA statistics for changes in self report measures of perceived life time cancer risk for Full Interview subjects with three definitions of risk information. (n=65)

<table>
<thead>
<tr>
<th>Definition of risk information used</th>
<th>cancer risk perception</th>
<th>Given health prone info</th>
<th>Given health risk info</th>
<th>Anova results</th>
</tr>
</thead>
<tbody>
<tr>
<td>i (airi_c)</td>
<td>Mean changes</td>
<td>-.7368</td>
<td>-.6250</td>
<td>F=0.058</td>
</tr>
<tr>
<td></td>
<td>SD</td>
<td>1.25</td>
<td>1.06</td>
<td>Df= 1,63</td>
</tr>
<tr>
<td></td>
<td>N</td>
<td>57</td>
<td>8</td>
<td>P=0.81</td>
</tr>
<tr>
<td>ii (rri1)</td>
<td>Mean changes</td>
<td>-.7455</td>
<td>-.6000</td>
<td>F=0.12</td>
</tr>
<tr>
<td></td>
<td>SD</td>
<td>1.27</td>
<td>.97</td>
<td>df=1,63</td>
</tr>
<tr>
<td></td>
<td>N</td>
<td>55</td>
<td>10</td>
<td>P=0.731</td>
</tr>
<tr>
<td>iii (rri2)</td>
<td>Mean changes</td>
<td>-.85</td>
<td>-.42</td>
<td>F=1.67</td>
</tr>
<tr>
<td></td>
<td>SD</td>
<td>1.26</td>
<td>1.07</td>
<td>df=1,63</td>
</tr>
<tr>
<td></td>
<td>N</td>
<td>46</td>
<td>19</td>
<td>P=0.20</td>
</tr>
</tbody>
</table>

Table 43 shows the comparison of changes in perceived life time risk for cancer for Self Administered subjects which occurred purely as a result of their participation in the study-they were not given their Short IRI scores. As can be seen in Table 43, when subjects who
were assessed as being in a health prone group (Type 4) were compared to those assessed as
being in a cancer/CHD prone group (Types 1 or 2), there is no significant difference in their
changes in perceived life time risk of cancer. Both groups showed a decrease in perception of
cancer risk as a result of their participation in the data collection, however there was a
smaller decrease in perceived risk of cancer in the ‘illness prone’ group- this difference failed
to reach a level of statistical significance.

Table 43. Summary of Univariate ANOVA statistics for changes in self report measures of perceived life time cancer risk
for Self Administered subjects. (n=40)

<table>
<thead>
<tr>
<th>Definition of risk assessment used</th>
<th>Cancer risk perception</th>
<th>Health proneness assessed</th>
<th>Illness proneness assessed</th>
<th>Anova results</th>
</tr>
</thead>
<tbody>
<tr>
<td>(airi_c)</td>
<td>Mean changes</td>
<td>-.2121</td>
<td>-.1429</td>
<td>F=0.04</td>
</tr>
<tr>
<td></td>
<td>SD</td>
<td>.74</td>
<td>1.21</td>
<td>df= 1.38</td>
</tr>
<tr>
<td></td>
<td>N</td>
<td>33</td>
<td>7</td>
<td>p=0.843</td>
</tr>
</tbody>
</table>

Summary of changes to cancer risk perception.

All subjects in the study, regardless of their interview condition or whether they received
health prone or health risk information, showed a decrease in self reported cancer risk
 perception from time1 to time2 of the interview. The differences between those who did and those who did not receive risk information failed to reach a level of significance.

Changes in Self Reported Cancer Anxiety.

Table 44 shows the comparisons of changes in cancer anxiety for Full Interview subjects who
were given their Short IRI scores according to three definitions of receipt of risk information
already detailed above. As can be seen, with each of the definitions of receipt of risk
 information used to compare subjects who received risk information and subjects who
received health prone information, there is no significant difference in their changes in cancer
anxiety. Both groups of subjects, using each of the receipt of risk information definitions,
showed a decrease in cancer anxiety and those subjects who were given risk information
according to airi_c and rri1 definitions showed a greater decrease in cancer anxiety, however these differences failed to reach a level of significance. When the rri2 definition of receipt of
risk information was used, subjects who received risk information showed a less decrease in cancer anxiety, however this also failed to reach a level of significance.

Table 44. Summary of Univariate ANOVA statistics for changes in self report measures of cancer anxiety for Full Interview subjects with three definitions of risk information. (n=65)

<table>
<thead>
<tr>
<th>Definition of risk information used</th>
<th>cancer anxiety</th>
<th>Given health prone info</th>
<th>Given health risk info</th>
<th>Anova results</th>
</tr>
</thead>
<tbody>
<tr>
<td>i (airi_c)</td>
<td>Mean changes</td>
<td>-.33</td>
<td>-.63</td>
<td>F=0.491</td>
</tr>
<tr>
<td></td>
<td>SD</td>
<td>1.02</td>
<td>1.60</td>
<td>Df= 1.63</td>
</tr>
<tr>
<td></td>
<td>N</td>
<td>57</td>
<td>8</td>
<td>p=0.486</td>
</tr>
<tr>
<td>ii (rr1)</td>
<td>Mean changes</td>
<td>-.35</td>
<td>-.50</td>
<td>F=0.17</td>
</tr>
<tr>
<td></td>
<td>SD</td>
<td>.97</td>
<td>1.72</td>
<td>Df= 1.63</td>
</tr>
<tr>
<td></td>
<td>N</td>
<td>55</td>
<td>10</td>
<td>p=0.686</td>
</tr>
<tr>
<td>iii (rr2)</td>
<td>Mean changes</td>
<td>-.43</td>
<td>-.21</td>
<td>F=.56</td>
</tr>
<tr>
<td></td>
<td>SD</td>
<td>.91</td>
<td>1.47</td>
<td>Df= 1.63</td>
</tr>
<tr>
<td></td>
<td>N</td>
<td>46</td>
<td>19</td>
<td>p=0.46</td>
</tr>
</tbody>
</table>

Table 45 shows the comparison of changes cancer anxiety for Self Administered subjects which occurred purely as a result of their participation in the study- they were not given their Short IRI scores. As can be seen in Table 45, when subjects who were assessed as being in a health prone group (Type 4) were compared to those assessed as being in a cancer/CHD prone group (Types 1 or 2), there is no significant difference in their levels of cancer anxiety. Both groups showed a decrease in cancer anxiety as a result of their participation in the data collection, however those assessed as being in an ‘illness prone’ group did show less of a decrease compared to the other subjects- this difference failed to obtain significance.

Table 45. Summary of Univariate ANOVA statistics for changes in self report measures of cancer anxiety for Self Administered subjects. (n=40)

<table>
<thead>
<tr>
<th>Definition of risk assessment used</th>
<th>cancer anxiety</th>
<th>Health proneness assessed</th>
<th>Illness proneness assessed</th>
<th>Anova results</th>
</tr>
</thead>
<tbody>
<tr>
<td>(airi_c)</td>
<td>Mean changes</td>
<td>-.303</td>
<td>-.2857</td>
<td>F=0.002</td>
</tr>
<tr>
<td></td>
<td>SD</td>
<td>.984</td>
<td>.488</td>
<td>Df= 1.38</td>
</tr>
<tr>
<td></td>
<td>N</td>
<td>33</td>
<td>7</td>
<td>p=0.964</td>
</tr>
</tbody>
</table>
Summary of changes to cancer anxiety.

All subjects in the study, regardless of their interview condition or whether they received health prone or health risk information, showed a decrease in self reported cancer anxiety from time1 to time2 of the interview. The differences between those who did and those who did not receive risk information failed to reach a level of significance.

Changes in Self Reported CHD risk perception.

Table 46 shows the comparisons of changes in perceived life time risk for CHD for Full Interview subjects who were given their Short IRI scores according to three definitions of receipt of risk information already detailed above.

As can be seen in Table 46, with each of the definitions of receipt of risk information used to compare subjects who received risk information and subjects who received health prone information, there is no significant difference in their changes in perceived life time risk of CHD. Both groups of subjects, using both airi_c and rri1 definitions, showed a decrease in perception of CHD risk and those subjects who were given risk information showed less of a decrease in CHD risk perception, however these differences failed to reach a level of significance. When the rri2 definition of receipt of risk information is used to compare subjects who received risk information and subjects who received health prone information, there is no significant difference in their changes in perceived life time risk of CHD. Both groups of subjects showed a decrease in perception of CHD risk, and those subjects who were given risk information showed a greater decrease in CHD risk perception, however this difference failed to reach a level of significance.
Table 46 Summary of Univariate ANOVA statistics for changes in self report measures of perceived life time CHD risk for Full Interview subjects with three definitions of risk information. (n=65)

<table>
<thead>
<tr>
<th>Definition of risk information used</th>
<th>CHD risk perception</th>
<th>Given health prone info</th>
<th>Given health risk info</th>
<th>Anova results</th>
</tr>
</thead>
<tbody>
<tr>
<td>i (airi_c)</td>
<td>Mean changes</td>
<td>-.579</td>
<td>-.250</td>
<td>F=0.501</td>
</tr>
<tr>
<td></td>
<td>SD</td>
<td>1.05</td>
<td>2.188</td>
<td>Df= 1,63</td>
</tr>
<tr>
<td></td>
<td>N</td>
<td>57</td>
<td>8</td>
<td>p=0.482</td>
</tr>
<tr>
<td>ii (rr1)</td>
<td>Mean changes</td>
<td>-.56</td>
<td>-.40</td>
<td>F=0.149</td>
</tr>
<tr>
<td></td>
<td>SD</td>
<td>1.05</td>
<td>2.01</td>
<td>Df=1,63</td>
</tr>
<tr>
<td></td>
<td>N</td>
<td>55</td>
<td>10</td>
<td>p=0.70</td>
</tr>
<tr>
<td>iii (rr2)</td>
<td>Mean changes</td>
<td>-.52</td>
<td>-.58</td>
<td>F=.029</td>
</tr>
<tr>
<td></td>
<td>SD</td>
<td>1.09</td>
<td>1.54</td>
<td>Df=1,63</td>
</tr>
<tr>
<td></td>
<td>N</td>
<td>46</td>
<td>19</td>
<td>p=0.87</td>
</tr>
</tbody>
</table>

Table 47 shows the comparison of changes in perceived life time risk of CHD for Self Administered subjects which occurred purely as a result of their participation in the study. As can be seen in Table 47, when subjects who were assessed as being in a health prone group were compared to those assessed as being in a cancer/CHD prone group, there is no significant difference in their changes in perceived life time risk of CHD. Both groups showed a decrease in perception of CHD risk as a result of their participation in the data collection, however there was a smaller decrease in perceived risk of CHD in the ‘illness prone’ group- this difference failed to reach a level of statistical significance.

Table 47. Summary of Univariate ANOVA statistics for changes in self report measures of perceived life time CHD risk for Self Administered subjects. (n=40)

<table>
<thead>
<tr>
<th>Definition of risk assessment used</th>
<th>CHD risk perception</th>
<th>Health proneness assessed</th>
<th>Illness proneness assessed</th>
<th>Anova results</th>
</tr>
</thead>
<tbody>
<tr>
<td>(air1_c)</td>
<td>Mean changes</td>
<td>-.272</td>
<td>-.143</td>
<td>F=1.07</td>
</tr>
<tr>
<td></td>
<td>SD</td>
<td>.977</td>
<td>.899</td>
<td>df= 1,38</td>
</tr>
<tr>
<td></td>
<td>N</td>
<td>33</td>
<td>7</td>
<td>p=0.307</td>
</tr>
</tbody>
</table>

Summary of changes to CHD risk perception.

All subjects in the study, regardless of their interview condition, or whether they received health prone or health risk information, showed a decrease in self reported CHD risk
perception from time1 to time2 of the interview. The differences between those who did and those who did not receive risk information failed to reach a level of significance.

**Changes in Self Reported CHD anxiety.**

Table 48 shows the comparisons of changes in CHD anxiety for Full Interview subjects who were given their Short IRI scores according to three definitions of receipt of risk information already detailed above. As can be seen, with the definitions of receipt of risk information airi_c and rri1, there is no significant difference in their changes in CHD anxiety. Both groups of subjects, in each of the receipt of risk information definitions, showed a decrease in CHD anxiety and those subjects who were given risk information according to airi_c and rri1 definitions showed a greater decrease in CHD anxiety, however these differences failed to reach a level of significance. When the rri2 definition of receipt of risk information was used, subjects who received risk information showed a less decrease in CHD anxiety, and this did reach a level of significance at the .05 level, indicating that there is a statistically significant smaller decrease in CHD anxiety in the ‘Illness-prone’ group of subjects when compared with the ‘health-prone’ group of subjects.

Table 48. Summary of Univariate ANOVA statistics for changes in self report measures of CHD anxiety for Full Interview subjects with three definitions of risk information. (n=65)

<table>
<thead>
<tr>
<th>Definition of risk information used</th>
<th>CHD anxiety</th>
<th>Given health prone info</th>
<th>Given health risk info</th>
<th>Anova results</th>
</tr>
</thead>
<tbody>
<tr>
<td>i (airi_c)</td>
<td>Mean changes</td>
<td>-.895</td>
<td>-.50</td>
<td>F=0.455</td>
</tr>
<tr>
<td></td>
<td>SD</td>
<td>1.484</td>
<td>2.0</td>
<td>df= 1.63</td>
</tr>
<tr>
<td></td>
<td>N</td>
<td>57</td>
<td>8</td>
<td>p=0.502</td>
</tr>
<tr>
<td>ii (rri1)</td>
<td>Mean changes</td>
<td>-.89</td>
<td>-.60</td>
<td>F=0.30</td>
</tr>
<tr>
<td></td>
<td>SD</td>
<td>1.51</td>
<td>1.78</td>
<td>df=1.63</td>
</tr>
<tr>
<td></td>
<td>N</td>
<td>55</td>
<td>10</td>
<td>p=0.59</td>
</tr>
<tr>
<td>iii (rri2)</td>
<td>Mean changes</td>
<td>-1.09</td>
<td>-.26</td>
<td>F=4.01</td>
</tr>
<tr>
<td></td>
<td>SD</td>
<td>1.44</td>
<td>1.67</td>
<td>df=1.63</td>
</tr>
<tr>
<td></td>
<td>N</td>
<td>46</td>
<td>19</td>
<td>p=0.50</td>
</tr>
</tbody>
</table>

Table 49 shows the comparison of changes in CHD anxiety for Self Administered subjects which occurred purely as a result of their participation in the study. As can be seen in Table
49, when subjects who were assessed as being in a health prone group were compared to those assessed as being in a cancer/CHD prone group, there is no significant difference in their changes in CHD anxiety. Both groups showed a decrease in perception of CHD anxiety as a result of their participation in the data collection, however the ‘illness prone’ subjects displayed a greater decrease in CHD anxiety than did the ‘health prone’ subjects but this failed to reach a level of significance.

<table>
<thead>
<tr>
<th>Definition of risk assessment used</th>
<th>CHD anxiety</th>
<th>Health proneness assessed</th>
<th>Illness proneness assessed</th>
<th>Anova results</th>
</tr>
</thead>
<tbody>
<tr>
<td>(air1_c)</td>
<td>Mean changes</td>
<td>-515</td>
<td>-1.143</td>
<td>F=0.5302</td>
</tr>
<tr>
<td></td>
<td>SD</td>
<td>2.167</td>
<td>1.464</td>
<td>df=1,38</td>
</tr>
<tr>
<td></td>
<td>N</td>
<td>33</td>
<td>7</td>
<td>p=0.471</td>
</tr>
</tbody>
</table>

Summary of changes to CHD anxiety.

With the exception of Full Interview subjects who received risk information according to the rri2 definition of risk, all subjects in the study, regardless of their interview condition or whether they received health prone or health risk information, showed a decrease in self reported CHD risk perception from time1 to time2 of the interview. The differences between those who did and those who did not receive risk information failed to reach a level of significance. The exception to this pattern is with Full Interview subjects who received risk information according to the rri2 definition of risk- these subjects showed a significantly lesser decrease in CHD anxiety when compared with subjects who received health prone information.

In summary of the overall Univariate ANOVA analyses that were conducted on Changes in Self Report measures, the overwhelming trend was towards a decrease in the cancer and CHD anxiety and risk perception measures, regardless of the interview type or the information received. The exception to this trend was with Full Interview subjects who received risk information according to the rri2 definition of risk, showing a lesser decrease in CHD anxiety when compared to subjects who received health prone information.
Study 1: Question 3- Predictions of Subjects’ Short IRI scores:
To test the current experimenter’s ability to accurately predict subject’s eventual Short IRI scores, a binomial test was conducted using SPSS. As can be seen from Table 50, it is clear that the accuracy of predictions of Short IRI score occurred at a rate above that of chance, with p<.05. The current experimenter was able to accurately predict the eventual Short IRI scores on the basis of the information which subjects revealed about themselves in the early stages of the full interview process.

Table 50. Frequency and Binomial Test output showing accuracy of health risk predictions (N= 52)

<table>
<thead>
<tr>
<th>Prediction of IRI</th>
<th>Category</th>
<th>N</th>
<th>Observed Prop.</th>
<th>Test Prop.</th>
<th>Asymp. Sig. (2-tailed)</th>
<th>Exact Sig. (2-tailed)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Group 1</td>
<td>Incorrect</td>
<td>16</td>
<td>31%</td>
<td>.50</td>
<td>.008(a)</td>
<td>.008</td>
</tr>
<tr>
<td>Group 2</td>
<td>Correct</td>
<td>36</td>
<td>69%</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Total</td>
<td></td>
<td>52</td>
<td>1.00</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

a Based on Z Approximation.

Section C) Study 2- Effects of familial history, Neuroticism and time on risk perception/anxiety:
Study Two is examining the effects of having a first order relative with a hereditary cancer or CHD on a subject’s perception of risk and illness anxiety- the power of this knowledge to effect risk perception and anxiety levels over time will be evaluated.

In Study 2, three questions are posed requiring an answer through the analysis.

- Question 1- will subjects with first order relatives known to have hereditary cancers/CHD’s show higher levels of cancer/CHD risk perception when compared to subjects with no such family history?
- Question 2- will a known family history of hereditary cancer/CHD interact with Neuroticism to influence risk perception and anxiety scores?
- Question 3- will a known family history of hereditary cancer/CHD interact with length of this knowledge (in years) to influence risk perception and anxiety scores?

To test the Study 2 hypotheses, a series of Multiple Regression Analyses were conducted using SPSS, examining the effects on cancer anxiety and risk perception and CHD anxiety.
and risk perception with the between subjects factor being ‘yes’ or ‘no’ family history of cancer or CHD. Additional factors such as Neuroticism scores and years of having this knowledge were examined for their effects. As such, all three of Study 2 questions are addressed in the following section as the additional variables were added to the Multiple Regression Analysis.

_Cancer Risk Perception:_

The effects of having a family history with hereditable cancers on cancer risk perception was examined in a Multiple Regression Analysis with the contributions of years of this knowledge and Neuroticism being added into the analysis.

Table 51 shows the correlations between the variables studied in this analysis. As can be seen, Cancer in Family shows a significant positive correlation with Years of family Cancer (p<.01).

**Table 51. Correlations between regression analysis variables (N=107)**

<table>
<thead>
<tr>
<th></th>
<th>Likely to get Cancer</th>
<th>Cancer in Family</th>
<th>Years of family Cancer</th>
<th>Neuroticism</th>
</tr>
</thead>
<tbody>
<tr>
<td>Likely to get Cancer</td>
<td>Pearson Correlation</td>
<td>1</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Cancer in Family</td>
<td>Pearson Correlation</td>
<td>.137</td>
<td>1</td>
<td></td>
</tr>
<tr>
<td>Years of family Cancer</td>
<td>Pearson Correlation</td>
<td>.147</td>
<td>.695(**)</td>
<td>1</td>
</tr>
<tr>
<td>Neuroticism</td>
<td>Pearson Correlation</td>
<td>.023</td>
<td>-.087</td>
<td>-.007</td>
</tr>
</tbody>
</table>

** Correlation is significant at the 0.01 level (2-tailed).

The Multiple Regression Analysis involved a hierarchical model building approach, adding blocks into the analysis to establish their contribution in accounting for the variance in the dependent variable, Likely to experience cancer. It can be seen from Table 52 that the Years of family cancer variable did account for the variance to a significant degree (p<.05). This demonstrates a contribution to increase in Cancer risk perception over time from the amount
of years which a person has known of the family cancer history. Neuroticism makes no contribution to explaining the variance seen in the dependent variable.

Table 52. Model Summary of Multiple Regression Analysis for Likely to experience cancer (N=107)

<table>
<thead>
<tr>
<th></th>
<th>B coefficient</th>
<th>SE</th>
<th>Significance</th>
</tr>
</thead>
<tbody>
<tr>
<td>Model 1: Years of family Cancer</td>
<td>.062</td>
<td>.029</td>
<td>.035</td>
</tr>
<tr>
<td>Model 2: Neuroticism</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Years of family Cancer</td>
<td>.031</td>
<td>.062</td>
<td>.619</td>
</tr>
<tr>
<td></td>
<td>.063</td>
<td>.029</td>
<td>.033</td>
</tr>
</tbody>
</table>

_Cancer Anxiety:_

The effects of having a family history with hereditable cancers on Cancer Anxiety was examined in a Multiple Regression Analysis with the contributions of years of this knowledge and Neuroticism being added into the analysis. A bivariate correlational analysis revealed that there were no additional correlations to those reported in Table 51- Cancer Anxiety did not correlate with any other variables, and as such the table will not be presented.

The same Multiple Regression Analysis approach was used for the dependent variable Cancer Anxiety as was reported in the analysis displayed above. As can be seen from Table 53, Model 2 does demonstrate a significance at the p<.01 level, with Years of Family Cancer accounting for the variance to a significant degree, demonstrating that there is a decrease in cancer anxiety over the course of time as a function of knowing of a family cancer history. Again, Neuroticism plays no role in explaining the variance.

Table 53. Model Summary of Multiple Regression Analysis for Cancer Anxiety (N=107)

<table>
<thead>
<tr>
<th></th>
<th>B coefficient</th>
<th>SE</th>
<th>Significance</th>
</tr>
</thead>
<tbody>
<tr>
<td>Model 1: Years of family Cancer</td>
<td>-.063</td>
<td>.034</td>
<td>.066</td>
</tr>
<tr>
<td>Model 2: Neuroticism</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Years of family Cancer</td>
<td>-.080</td>
<td>.072</td>
<td>.273</td>
</tr>
<tr>
<td></td>
<td>-.065</td>
<td>.034</td>
<td>.058</td>
</tr>
</tbody>
</table>
**CHD Risk Perception:**

The effects of having a family history with hereditable heart diseases on CHD risk perception was examined in a Multiple Regression Analysis with the contributions of years of this knowledge and Neuroticism being added into the analysis.

Table 54 shows the correlations between the variables studied in this analysis. As can be seen, there are significant positive correlations between Likely to get CHD and CHD in Family, and Years of family CHD (p<.05). CHD in Family and Years of family CHD also highly correlate at the p<.05 level.

<table>
<thead>
<tr>
<th></th>
<th>Likely to get CHD</th>
<th>CHD in Family</th>
<th>Years of family CHD</th>
<th>Neuroticism</th>
</tr>
</thead>
<tbody>
<tr>
<td>Likely to get CHD</td>
<td>Pearson Correlation</td>
<td>1</td>
<td></td>
<td></td>
</tr>
<tr>
<td>CHD in Family</td>
<td>Pearson Correlation</td>
<td>460(**)</td>
<td>1</td>
<td></td>
</tr>
<tr>
<td>Years of family CHD</td>
<td>Pearson Correlation</td>
<td>.384(**)</td>
<td>.697(**)</td>
<td>1</td>
</tr>
<tr>
<td>Neuroticism</td>
<td>Pearson Correlation</td>
<td>-.005</td>
<td>-.070</td>
<td>-.128</td>
</tr>
</tbody>
</table>

** Correlation is significant at the 0.01 level (2-tailed).

The same Multiple Regression Analysis approach was used for the dependent variable (Likely to get CHD) as was reported in the analyses reported above. As can be seen from Table 55, both Years of family CHD and Neuroticism do not explain any of the variance in the dependent variable, Likely to get CHD.

<table>
<thead>
<tr>
<th></th>
<th>B coefficient</th>
<th>SE</th>
<th>Significance</th>
</tr>
</thead>
<tbody>
<tr>
<td>Model 1: Years of family CHD</td>
<td>-.023</td>
<td>.017</td>
<td>.185</td>
</tr>
<tr>
<td>Model 2: Neuroticism  Years of family CHD</td>
<td>.093</td>
<td>.063</td>
<td>.139</td>
</tr>
<tr>
<td></td>
<td>-.019</td>
<td>.017</td>
<td>.287</td>
</tr>
</tbody>
</table>
**CHD Anxiety:**

The effects of having a family history with hereditable heart diseases on CHD Anxiety was examined in a Multiple Regression Analysis with the contributions of years of this knowledge and Neuroticism being added into the analysis.

Table 56 shows the correlations between the variables studied in this analysis. As can be seen in Table 56, Anxious about CHD shows a significant positive correlation with CHD in Family; and CHD in Family shows a significant positive correlation with Years of family CHD (both p<.05).

![Table 56](image)

**Table 56. Correlations between multiple regression analysis variables (N=107)**

<table>
<thead>
<tr>
<th></th>
<th>Anxious about CHD</th>
<th>CHD in Family</th>
<th>Years of family CHD</th>
<th>Neuroticism</th>
</tr>
</thead>
<tbody>
<tr>
<td>Anxious about CHD</td>
<td>Pearson Correlation</td>
<td>1</td>
<td></td>
<td></td>
</tr>
<tr>
<td>CHD in Family</td>
<td>Pearson Correlation</td>
<td>.259(**)</td>
<td>1</td>
<td></td>
</tr>
<tr>
<td>Years of family CHD</td>
<td>Pearson Correlation</td>
<td>.160</td>
<td>.697(**)</td>
<td>1</td>
</tr>
<tr>
<td>Neuroticism</td>
<td>Pearson Correlation</td>
<td>.101</td>
<td>-.070</td>
<td>-.128</td>
</tr>
</tbody>
</table>

**Correlation is significant at the 0.01 level (2-tailed).**

The same Multiple Regression Analysis approach was used for the dependent variable (CHD Anxiety) as was reported in the analyses reported above. As can be seen from Table 57, the study variables Years of family CHD and Neuroticism do not predict the dependent variable Anxious about CHD at all.

![Table 57](image)

**Table 57. Model Summary of Regression Analysis for Anxious about CHD (N=107)**

<table>
<thead>
<tr>
<th></th>
<th>B coefficient</th>
<th>SE</th>
<th>Significance</th>
</tr>
</thead>
<tbody>
<tr>
<td>Model 1: Years of family CHD</td>
<td>-.008</td>
<td>.015</td>
<td>.573</td>
</tr>
<tr>
<td>Model 2: Neuroticism</td>
<td>.015</td>
<td>.054</td>
<td>.778</td>
</tr>
<tr>
<td>Years of family CHD</td>
<td>-.008</td>
<td>.015</td>
<td>.613</td>
</tr>
</tbody>
</table>
Table 58 shows the correlations between Age and the Self Report measures which were taken in the Health Psychology Questionnaire, prior to the interview. As can be seen, there are correlations between Age and Likely to get CHD, Years of family cancer and Years of family CHD (all \( p<.01 \)). Likely to experience cancer correlates positively with Anxious about cancer, Likely to get CHD, and Anxious about CHD (all \( p<.01 \)). Anxious about Cancer correlates with Likely to get CHD and Anxious about CHD (both \( p<.01 \)). Likely to get CHD correlates with Anxious about CHD and Years of family CHD (both \( p<.01 \)) and to a lesser extent with Years of family cancer (\( p<.05 \)).

Table 58. Correlations between Age and Self Report Measures (N=107)

<table>
<thead>
<tr>
<th></th>
<th>Age</th>
<th>Likely to experience cancer</th>
<th>Anxious about cancer</th>
<th>Likely to get CHD</th>
<th>Anxious about CHD</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age</td>
<td>Pearson Correlation</td>
<td>1</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Sig. (2-tailed)</td>
<td>.</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Likely to experience cancer</td>
<td>Pearson Correlation</td>
<td>.007</td>
<td>1</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Sig. (2-tailed)</td>
<td>.946</td>
<td>.</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Anxious about cancer</td>
<td>Pearson Correlation</td>
<td>-.038</td>
<td>.594(**)</td>
<td>1</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Sig. (2-tailed)</td>
<td>.695</td>
<td>.000</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Likely to get CHD</td>
<td>Pearson Correlation</td>
<td>.385(**)</td>
<td>.322(**)</td>
<td>.229(*)</td>
<td>1</td>
</tr>
<tr>
<td></td>
<td>Sig. (2-tailed)</td>
<td>.000</td>
<td>.001</td>
<td>.018</td>
<td>.</td>
</tr>
<tr>
<td>Anxious about CHD</td>
<td>Pearson Correlation</td>
<td>.153</td>
<td>.281(**)</td>
<td>.542(**)</td>
<td>.653(**)</td>
</tr>
<tr>
<td></td>
<td>Sig. (2-tailed)</td>
<td>.116</td>
<td>.003</td>
<td>.000</td>
<td>.000</td>
</tr>
<tr>
<td>Years of family cancer</td>
<td>Pearson Correlation</td>
<td>.364(**)</td>
<td>.147</td>
<td>.066</td>
<td>.201(*)</td>
</tr>
<tr>
<td></td>
<td>Sig. (2-tailed)</td>
<td>.000</td>
<td>.130</td>
<td>.499</td>
<td>.038</td>
</tr>
<tr>
<td>Years of family CHD</td>
<td>Pearson Correlation</td>
<td>.551(**)</td>
<td>-.102</td>
<td>-.103</td>
<td>.384(**)</td>
</tr>
<tr>
<td></td>
<td>Sig. (2-tailed)</td>
<td>.000</td>
<td>.294</td>
<td>.291</td>
<td>.000</td>
</tr>
</tbody>
</table>

**Correlation is significant at the 0.01 level (2-tailed).
* Correlation is significant at the 0.05 level (2-tailed).
Chapter 15
DISCUSSION

The Sample:
T tests reported in the Results section (tables 17, 19, 20) indicate that the sample used in this study was not biased by gender in the demographic variables of number of family members with hereditable cancers and heart diseases; in the amount of years they have known of this family history; in the dependent variable of Change in Heart Rate; in the potential predictor variables of Cancer Likelihood, CHD Likelihood, Extroversion, Neuroticism, Monitor Score or Blunter Score; and changes in Self Report measures. In dividing the subjects into the two treatment groups (Self Administered and Full Interview), there was again no difference between the groups in regards to the family history of illness variables (Table 18). There was also no difference in the response patterns of the two study groups in regards to responses on the Short IRI; the potential predictor variables and the study’s dependent variables (table 21). The only exception to this is the larger decrease in Change in Risk Perception for Cancer in the Full Interview subjects when compared with the Self Administered subjects. As such, the sample can be viewed as constituting subjects in the two treatment conditions which are the same on demographic variables.

Comparison with Grossarth-Maticek’s reports:
Consistent with Grossarth-Maticek & Eysenck’s (1990) reports, it is seen in this sample that men score higher on each of the Short IRI types (table 20), however in the current sample none of these differences reach a level of statistical significance. In addition, the scores on each of the Short IRI types show no significant difference when comparing the Self Administered with the Full Interview groups (table 21). It was important to establish if Grossarth-Maticek’s health and illness prone constructs (in Grossarth-Maticek & Eysenck 1990) used in this study actually display patterns which are consistent with his reports. For example, if there was a strong positive correlation between Type 1 and Type 4 scores, then the internal consistency of the Short IRI would have to be called into question, as would any of the subsequent results. As Grossarth-Maticek & Eysenck
(1990) report relationships between the Short IRI constructs and other constructs well established in psychology, e.g. Neuroticism, it was important also to assess the relationships between such variables in the current study. Again, if there was found to be, for example, a negative correlation between Neuroticism and Type 1 scores in the current study, then either Grossarth-Maticzek & Eysenck’s (1990) results or those in the current study would have to be considered more questionable. Beyond this need to establish the internal consistency of the reported results with Grossarth-Maticzek’s reports, it was also important to address the issue of vulnerability, as the proposed treatment effect was only assumed to have occurred with the most psychologically vulnerable of Grossarth-Maticzek’s subjects, i.e. Type 1 & 2 subjects, each of which should correlate with Neuroticism.

Other variables in the study were presumed to be potential predictor variables, perhaps with the power to alter the dependent variables, e.g. pre-existing levels of cancer/CHD risk perception and associated levels of anxiety; cancer and CHD in family history; Miller’s (1996) Monitor/Blunter construct. All of these factors may have had the power to alter the dependent variables, either on their own or in conjunction with the independent variables, so it was important to review the nature of the relationships amongst them.

It was proposed in the current study that only Grossarth-Maticzek’s most psychologically vulnerable subjects could have been adversely affected by an interview effect or by being directly told of their IRI risk status. In fact, it is only the most psychologically vulnerable of subjects who would have been assessed as being risk prone and thereby able to suffer the deleterious effects of learning of their negative prediction. As such, it was a presumption running through the current study that the most psychologically vulnerable of the current subjects would display larger alarm reactions to learning of a negative health prediction. Or put another way, the factors presented here were all hypothesized to be measures of psychological vulnerability to an alarm reaction when learning of a negative health prediction— as such, they were considered to be potential predictor variables of the dependent variables. These factors included:-
Short IRI Type 1 score (cancer prone); IRI Type 2 score (CHD prone); Neuroticism; Introversion/Extroversion; history of hereditable cancers in the subject’s family; history of hereditable CHD’s in the subjects family; risk perception of cancer prior to the interview; cancer anxiety prior to the interview; risk perception of CHD prior to the interview; CHD anxiety prior to the interview; Monitor scores.

It was hypothesized that there would be correlations between these factors, and ultimately a relationship between these factors and subject’s changes in heart rate measures on learning of a negative health prediction. A series of correlational studies were conducted to establish if the measures taken from the current sample correspond to the pattern of measures as reported by Grossarth-Maticzek and Eysenck (1990).

Grossarth-Maticzek and Eysenck (1990) claim that there is a correlation between Type 1 and Type 2 scores (as they are both predictive of ill-health); and between Type 1/Type 2 scores and Type 3 scores as the latter is a mixture of the behavioral tendencies of the former; and a negative correlation between Type 1/Type 2/Type 3 scores and Type 4 scores, as the latter entails a behavioural style which is oppositional to the other three styles. In relation to the current study, Types 1 and 2 are viewed as behavioural and psychological characteristics which would make subjects vulnerable to showing an alarm response in the Full Interview condition.

The current study showed that there were positive correlations (for the entire sample) between Type 1, Type 2 and Type 3 scores; while each of these types showed a negative correlation with Type 4 scores (Table 22). This result was expected and is consistent with Grossarh-Maricek & Eysenck’s (1990) reports. If the current study did not show this pattern of correlations, then any results reported would either have to call into question Grossarh-Maticzek’s or the current study’s results.
The current study investigated the relationships between each of the Short IRI types and personality variables for subjects in the full sample, considered to be potential predictor variables. Each of these personality variables were considered to be characteristics which make the subjects vulnerable to displaying an alarm reaction on learning of their Short IRI scores. Potential predictor/personality variables (Neuroticism, Extroversion, Monitor scores) were included in the study as the health psychology literature suggests relationships between these and ‘signature responses’ (eg. alarm) to a range of stimuli, including news of health risk. The responses can include altered perceptions of risk and increases in physiological arousal, suggesting alarm. Neuroticism, Extroversion/Introversion, and Monitor scores all have implications for anxiety responses, as discussed in earlier sections. These personality variables proffered by Eysenck (1965) and Miller (1996) have the potential to influence the dependent variable measures.

Grossarth-Maticek & Eysenck (1990) report a high level of correlation between Type 1 and Type 2 scores and Neuroticism; a high positive correlation between Introversion and Type 1 scores, and a positive correlation between Type 2 and Extroversion scores. Consistent with these reports, the current study also supported the contention that Type 1 scores are reflected in both high Neuroticism and high Introversion scores- these appear to be the core features of a high Type 1 score. Grossarth-Maticek & Eysenck (1990) report that Type 2 scores are reflected in high Neuroticism and high Extroversion scores. The current study provided some support for this in the form of a strong positive correlation between Type 2 and Neuroticism scores, but there was no correlation with Extroversion found (Table 23). This finding may be consistent with Amelang’s (1997) finding that the Short IRI was unable to distinguish between cancer patients and CHD patients, although it was able to predict which subjects were experiencing ill health and which were currently healthy. The current finding might add weight to Amelang’s (1997) contention that the Short IRI is not sensitive enough to discriminate between types of ill-health profiles.
As per Grossarth-Maticek & Eysenck’s (1990) report, Type 3 subjects in this sample demonstrated a high correlation with Neuroticism, as it is suggested to be a combination of the personality profiles of both Types 1 & 2, representing a ‘hysterical’ profile. And finally, Type 4 scores in the current study show a strong negative correlation with Neuroticism, suggesting that as per Grossarth-Maticek & Eysenck’s (1990) reports, Type 4 subjects are the most psychologically stable respondents. Monitor and Blunter scores showed no correlations with any of the other study variables reported in the entire sample, other than with Type 2 scores. This perhaps makes sense as Miller (1986) reports that Monitor characteristics appear to be independent from other personality characteristics.

In regards to Short IRI scores and other study variables, the current study found a small but significant positive correlation between Type 4 scores and CHD history in family (Table 24). There is no known implication of this as far as personality goes, other than to say that it appears Type 4 scorers had more CHD in their family background than did other subjects.

It was expected that the most Neurotic subject’s (reflected in high Type 1, 2 & 3) scores would display the most cancer and CHD risk perception and anxiety. With the exception of a positive correlation between Type 3 scores and Cancer risk perception, this expectation was not borne out in the current study of the entire sample (Table 25). This result suggests that having a high Neuroticism score per se is not enough to make one anxious about the prospect of contracting cancer or CHD, or to view oneself as being more vulnerable to these conditions. This finding has implications for the notion of vulnerability as discussed earlier in this paper. The contention was that being a high Type 1, 2 or 3 scorer (with higher levels of Neuroticism, as established in this study) would make a subject more vulnerable to cancer or CHD anxiety and risk perception. This contention has not been demonstrated by the current study.

In summary, it can be seen from Tables 22-26 of the Results section that the pattern of data for the entire sample of the current study are similar to those reported by Grossarth-
Maticek & Eysenck (1990), and that the other potential predictor variables demonstrate very few correlations with the Short IRI scores.

The same correlations for the Full Interview condition subjects showed a very similar pattern to both the current entire sample and to Grossarth-Maticek & Eysenck’s (1990) reports (tables 59-63 in Appendix 6). The main differences between the Full Interview subjects and the data from the entire sample were with Type 2 scores. In the entire sample, Type 2 scores showed a positive correlation with Monitor scores, whereas this was not apparent in the Full Interview subjects; and Type 2 scores showed a positive correlation with Likely to experience cancer in the Full Interview condition, but not in the entire sample- the Full Interview condition correlation is the type of outcome that was expected, with more Neurotic subjects showing a higher sense of vulnerability to ill-health.

The other difference between the Full Interview subjects and the entire sample is the correlation between Monitor scores and Likely to get CHD (Table 63). Again, this outcome is more consistent with the expectations stated at the outset of the study, however in hindsight it needs to be noted that the characteristics of Monitoring or Blunting are only engaged on the presentation of threatening information- when subjects completed the first of the series of self report measures pertaining to sense of risk, no risk information was presented to them.

When the same correlational analyses were conducted for the Self Administered subjects, again a similar pattern of responses emerged as for the entire sample and as with Grossarth-Maticek & Eysenck’s reports (1990) (Tables 27-29). Most of the differences found were in terms of degrees of significance, not in the direction of correlations. Other differences were with Type 3 scores showing a negative correlation with CHD risk perception and Anxious about CHD. This finding is contrary to the expectations, as it suggests that the higher one’s Type 3 scores, the lower is the subject’s sense of risk to CHD and anxiety about CHD- the opposite finding was expected, and this does not support the overall notion of psychological vulnerability as proposed.
In summary, the current findings do provide some level of support for Grossarth-Matickek & Eysenck’s (1990) reports of general patterns of responses, i.e it appears that Type 1, 2 & 3 scores are positively correlated with Neuroticism scores; whereas Type 4 scores are negatively correlated with Neuroticism. Beyond this basic finding for the entire sample, Full Interview condition and the Self Administered condition, the notions of vulnerability to sense of risk and anxiety about cancer and CHD by virtue of either family history or personality characteristics is for the most part not supported by the current findings. This suggests that other factors are more important in the production of cancer/CHD anxiety and risk perception. Factors such as internal/bodily cues, and amount of cohorts experiencing cancer/CHD (both a function of age) may have more of a role to play in anxiety and risk perception, however the current study did not measure these variables.

Study 1-Question 1:
In order to test Study-Question 1, subjects in the current study who were allocated to the Full Interview condition were given their Short IRI scores after completion of the test administration when this was requested. The exception to this was when the current author considered there to be an ethical concern with giving an elderly person a test result which suggested a vulnerability to cancer or CHD. As Grossarth-Matickek’s (1980) research was conducted with a particular age group (elderly people), it was important to establish if age was a factor in the results obtained in the current study. In order to establish if age was a factor influencing the receipt of risk information, the ages of the subjects were divided into three categories for analysis: i) under 30 years old; ii) 30-49 years old; iii) 50 year old and over in relation to the three definitions of receipt of risk information from Short IRI scores.

Full Interview subjects in the sample of 66 used in this analysis were those who actually did receive their Short IRI scores. Subjects who underwent the interview but who either nominated to not learn of their Short IRI score (1 subject in the oldest age group); and subjects for whom there was an ethical concern in revealing to them their Short IRI score (5 elderly subjects were excluded from learning of their Short IRI score for this reason)
were not given their Short IRI scores and therefore their data did not enter this analysis. One of the ethical considerations in embarking on this study was a confidence in the current researcher’s ability to adequately debrief subjects at the end of the interview in order to ‘un-do’ any sense of risk or anxiety which was not present at the commencement of the interview. When, during the interview administration, the current researcher was not confident in his ability to successfully debrief a subject, he made the decision on ethical grounds to not provide the subject with the Short IRI score- it was simply stated to the subject that the results are not available.

Table 32 shows that there were no subjects in the 50 and over age group who received risk information according to definition airi_c. Of the elderly people who did nominate to learn of their Short IRI score, and for whom there were no ethical concerns, all 16 of them obtained no health risk information according to the definitions used. For this reason, it may be considered that the sample was biased in the sense that the elderly subjects who most impressed the interviewer as being psychologically vulnerable were excluded from learning of their health risk scores due to the ethical concerns detailed above. As such, their data did not enter the analysis, but the data of only the most psychologically robust elderly subjects did enter the analysis- these people were unlikely to be given risk information (borne out by the results), and were therefore not likely to show an alarm response to the Short IRI results. The impression of psychological vulnerability or robustness was developed by the interviewer in the course of the discussion of positive and negative life events and their typical reaction to these, preceding the administration of the Short IRI questions. Those for whom there was such an ethical concern were typically elderly subjects whose presentation suggested the least emotional stability (note: emotional instability is correlated to high Type 1 (cancer prone) and Type 2 (CHD prone scores). Several of the elderly subjects reported WWII combat experience, with statements such as “my nerves have been shot ever since”. Others reported the recent losses of their life long spouses- the prospect of further distressing such subjects was a major ethical consideration and restriction that was not anticipated in this study. In addition, there was a possible selection bias operating in the older sample as elderly subjects were recruited from Probus clubs. It is possible that such social clubs are
only appealing to elderly people who are the least neurotic in the geriatric population, i.e only the most psychologically robust elderly people seek out social contact with peers in a formal group setting. The most psychologically unstable elderly people may have been found in other locations, such as psycho-geriatric wards of hospitals, or even simply in the solitude of their own homes where they prefer to spend their time.

As a result, it may be concluded that the data presented in the analysis is somewhat biased in that the responses of the most vulnerable elderly subjects was not gathered for this analysis, while only the data of the most psychologically robust elderly people was included. Consequently, the results suggesting that Age played no role in the results may be called into question due to this methodological issue.

It may be the case that the people most vulnerable to a deleterious interviewer effect in Grossarth-Maticek’s sample were just the type of people excluded from the current study due to the ethical concern stated above. If these people were included in the current study, a better test of the hypothesis in regards to psychological vulnerability may have been more possible. As they were excluded from the experimental aspect of the current study, it is possible that the interviewer bias effect which was hypothesized may have occurred, but had a much reduced chance of making itself evident. It is possible that the ability to frighten people into an exaggerated risk perception is only likely to occur in combination with other factors, such as:- Neuroticism and family history of cancer/CHD (as measured in the current study); traumatic life experiences; internal/bodily cues; and having cohorts experience cancer and CHD in increasing rates (not measured in the current study). The last set of these factors are more likely to occur for an older population as they generally have more bodily symptoms of ill-health and have more associates dying of cancer and CHD than do younger people. This combination of factors, along with the interviewer bias may have worked in Grossarth-Maticek’s sample simply because of the age group of his subjects and his means of accessing them, eg. sampling the oldest person in every second house in Crvenka. As the most vulnerable of this age group were excluded from the current study for the ethical concerns stated as
well as the sampling method, it would appear that the hypothesis has not been fairly tested.

The results also demonstrate that being assessed as a Type 1 or Type 2 scorer was not dependent upon gender- males and females were just as likely to be obtain Type 1 or Type 2 as their highest scores, regardless of the definition of receipt of risk information used.

The results clearly demonstrate that there are no correlations between the dependent variable (Changes in Mean Heart Rate) and any of the other study variables (Tables 33-35). It was hypothesized that there would be a relationship found between this dependent variable and such factors as Neuroticism and family history of cancer/CHD, but this has been demonstrated to not be the case (Tables 35 & 34 respectively)- it appears that Changes in Mean Heart Rate is independent of these variables.

The main question of Study 1 was to determine if subjects in the Full Interview condition would show an alarm reaction (as measured by Changes in Mean Heart Rate) on presentation of risk information as assessed by the Short IRI. It was hypothesized that subjects who receive a Type 1 or Type 2 score would show a greater Change in Mean Heart Rate score (an increase) compared to subjects who received a Type 3 or Type 4 score. A series of Univariate ANOVA tests were run to assess this hypothesis with each of the three definitions of receipt of risk information. These tests were also conducted adjusting for both age and gender. As seen in the Results section (Tables 36-38), each of the groups demonstrated a decrease in the values of mean heart rates from the pre-test administration time frame (time 1) to the post-test administration time frame (time 2)-this held true for each of the definitions of receipt of risk information. There were no significant differences between the risk information and health prone information groups. This finding fails to support the hypothesis which stated that subjects receiving risk information news would demonstrate an alarm reaction in terms of elevated heart rates for the time 2 over time 1 period. As such, the Null Hypotheses must be accepted as there is a lack of support for the Experimental Hypotheses.
The fact that both groups of subjects actually showed a decrease in average heart rates is an unexpected finding. It needs to be noted that while each of the groups analyzed in the Univariate ANOVA showed a decrease of mean heart rate in the time2 segment of the interview, this trend only makes sense in relation to the mean heart rates of the time1 segment. As already noted, during the time1 segment, subjects in the Full Interview condition were asked to reflect on and discuss positive and negative life events, and their typical reactions to these. As they were discussing real events that they had experienced, often involving trauma or emotional upset, their mean heart rates for this segment were elevated due to the salience of the topics. The time2 segment involved them receiving their Short IRI scores and discussing the implications in terms of what may happen to them. It is possible that the discussion of any potential, positive or negative, does not have the same salience and power to create a physiologically measured reaction as does the discussion of an actual negative life event. The research conducted by Huang et al (1989) strongly supports the notion that discussion of actual life events is more likely to produce demonstrable changes in measures such as heart rate, compared to tasks which are artificial challenges or have no immediate reality to the subject- the same effect has been seen in the current study. In the current study, the time1 segment of the interview did not operate as a base line measure of physiological arousal, which would usually be taken when the person was in a relaxed state. These subjects were often not relaxed, but because of what they were being asked to recount, were often in a high state of emotional arousal. Considering this, it is sensible that the next segment of the interview (time2) would lead to a decrease in mean heart rate measures as the challenge of discussing negative life events had passed. As such, the decrease in mean heart rates is not a reflection of the alarm potential of the Short IRI scores, but a reflection on the alarm/arousal potential of the time1 segment topic area, during which they were discussing actual life events. In hindsight, a baseline measure of physiological arousal during relaxation, followed by the experimental procedure would have produced more meaningful results.
Study 1-Question 2:

Study 1 was also interested in testing for the effect of risk information on the Self Report measures of risk perception and anxiety as dependent variables. Table 40 of the Results section demonstrates that when analyzed as the whole sample, subjects’ self report measures of risk perception and anxiety decreased from time1 to time2, i.e. they appear to have become less anxious about the prospect of contracting cancer or CHD, and they viewed themselves as being less at risk of either of these conditions over the course of the interview. Tables 42, 44, 46, & 48 for the Full Interview condition show that this held true for each of the definitions of receipt of risk information; Tables 43, 45 & 47 show the same pattern for subjects in the Self Administered condition. The Univariate ANOVA tests also support the conclusion that Self Reported measures of cancer/CHD anxiety and risk perception decreased, regardless of interview type and the Short IRI score obtained. The only exception to a general finding of the above stated pattern is the result for Full Interview subjects when the rri2 definition of receipt of risk information was used (Table 48). Subjects who received risk information according to the Short IRI (ie, when their Type 1 or 2 score was more than 5/10) showed less of a decrease in CHD anxiety when compared to subjects who received health prone information. This finding alone is the only one which lends any support to the hypothesis- all other results from the Self Report measures fail to support the hypothesis, and the support that this lends is not to the extent hypothesized. As such the Null Hypotheses must be accepted as there is a lack of support for the Experimental Hypotheses.

The outcome of decreases in anxiety and risk perceptions may have resulted from a ‘repeated exposure’ effect, ie. the more times people are asked the self assessments of lifetime risk and anxiety level concerning cancer and CHD, the more they are habituated to the questions and their answers reflect a trend towards less anxiety and less perception of risk merely because of exposure to the question. The first time a person is asked these questions, they may respond with higher levels of self reported anxiety and perception of risk because these are unusual questions to be asked in our society and the prospect is highly feared. Previous research has demonstrated that people in our culture are most
anxious of cancer, but also of CHD- we are all somewhat over-sensitized to issues of cancer and CHD. By the time the subjects answered the Self Report questions at time2, they had already answered the same questions on two prior occasions, i.e in the initial Health Psychology Questionnaire; at the beginning of the interview; and finally at time2 after the interview had concluded. For Full Interview subjects the interview concluded when they were given their Short IRI scores, while for Self Administered subjects the interview concluded when they handed back their completed Short IRI questionnaires. Being the third occasion of answering these questions (happening between 15 minutes and 1¼ hours after they had most recently answered them, at time1), the possibility arises that they had simply become somewhat de-sensitized to the arousing nature of the questions. This should come as no surprise, as many behaviour modification techniques (e.g. Systematic Desensitization) work because of this process of repeated exposure, either in-vivo or in-situ.

**Study 1-Question 3:**

Study 1-Question 3 was conducted in order to test the possibility of an experimenter bias in Grossarth-Maticke’s methodology. The experimenter effect hypothesis is based on the notion that Grossarth-Maticke’s interviewers were able to either overtly or covertly communicate a sense of the proband’s risk status during the course of the interview. In order to do this, the interviewer must have been able to accurately predict whether a proband was vulnerable as per the Short IRI constructs (i.e Type 1 or Type 2) on the basis of their discussion of positive and negative life events. As such, it was decided to test the current researcher’s ability to accurately predict the subjects’ eventual Short IRI score on the basis of the discussion of positive/negative life events alone, prior to the administration of the Short IRI. The inference is that if the current researcher was able to accurately predict Short IRI risk status above the rate of chance, then Grossarth-Maticke’s interviewers would also have been able to pre-empt the proband’s Short IRI scores on the basis of the conversation alone, and ultimately convey this via the course of an interviewer bias or an overt statement. If the current researcher was unable to predict above the rate of chance subjects’ risk status, then it would seem unlikely that Grossarth-
Maticek’s interviewers could have developed a sense of the proband’s risk status and then conveyed this in any accurate sense.

In the current study, the researcher made a prediction as to whether a subject would receive an assessment of risk prediction (a higher Type 1 or 2 score than Type 4 score) from the Short IRI, or an assessment of health proneness (higher Type 3 or Type 4 score). This prediction was based on the information provided by the subject in relation to questions about their positive and negative life events and their typical responses to these. The current researcher utilized his knowledge of Grossarth-Maticzek’s (1980) taxonomy of personality factors to decide where their highest score would be obtained based purely on the information which subjects provided about themselves. The prediction was recorded prior to the administration of the Short IRI questions. The sample size for this research question was less than all of the Full Interview subjects (52 out of 65 Full Interview subjects). This disparity in numbers is due to inadvertent omissions to make the predictions on the interviewer’s behalf with 13 of the subjects- the interview itself was a very ‘full’ process with much data being gathered, and there were 13 occasions where the current interviewer simply forgot to make the prediction in the context of many other tasks that needed to be attended to.

As the Results clearly demonstrate, the current researcher’s ability to predict the eventual health risk or health prone score status was accurate above the rate of chance (Table 50). Consequently, Hypothesis iii) of Study 1 is found to be supported. It is clear from this result that based on the information gained from a discussion about the subjects positive and negative life experiences, an interviewer who is familiar with Grossarth-Maticzek’s taxonomy is able to accurately predict above the rate of chance the subjects eventual Short IRI status in terms of health risk or prone to positive health. As such, the possibility that this ability would have allowed Grossarth-Maticzek’s interviewers to either overtly or inadvertently convey the risk status to his probands remains.
**Study 2:**

Study 2 was aimed at testing the hypothesis that finding oneself in a heightened risk group (as may have Grossarth-Maticek’s Type 1 & 2 subjects) is anxiety arousing over a protracted period of time. The Study 2 hypothesis is based on the notion that Grossarth-Maticek’s Type 1 & 2 subjects, being characteristically less emotionally stable, could have maintained their elevated cancer/CHD risk perceptions and anxiety levels for the length of the prospective study, so as to produce the reported results at all of the follow up times, extending to 15 years.

As it is not possible to directly test this hypothesis for reasons already stated, Study 2 examined the effects of having a first order relative with a hereditary cancer or CHD on a person’s perception of cancer/CHD risk perception and illness anxiety- having such a first order relative presents a degree of risk in a similar way as it is proposed Grossarth-Maticek’s Type 1 & 2 subjects perceived a degree of risk due to their IRI status. The power of this knowledge (of heightened risk) to effect risk perception and anxiety levels over time was evaluated in order to indirectly test the current hypothesis of a long term interviewer effect of Grossarth-Maticek’s interviews. In the current sample, subjects with a first order relative who have a ‘genetic’ cancer or ‘genetic’ CHD were tested for cancer/CHD risk perception and anxiety levels with the length of time they have known of their heightened risk being measured. It was proposed that subjects in the current study who were at-risk of cancer/CHD for family history reasons would display more cancer/CHD anxiety and risk perception when compared with subjects with no such family history. Further, it was proposed that there would be a relationship in this sub group of at-risk subjects between Neuroticism and cancer/CHD risk perception and anxiety, with higher Neuroticism scorers displaying higher scores on these measures. It was proposed that the strength of the relationship between family history, Neuroticism and risk perception/anxiety would not diminish over time.

Subjects who responded that they had first order relatives with cancers/CHD known to have a strong genetic component were expected to demonstrate higher levels of cancer/CHD anxiety and risk perception than subjects with no such family history. It was expected that Neuroticism scores would add to the variance of cancer/CHD anxiety when comparing subjects with and those without a relevant family history. It was expected that this effect would be demonstrated over time, with the
level of cancer/CHD anxiety and risk perception remaining high regardless of the amount of intervening years since they learnt of their heightened level of cancer risk.

As seen in the Results section (Table 52), the Multiple Regression Analysis demonstrated a contribution to increase in Cancer risk perception over time from the amount of years which a person has known of the family cancer history. The longer a subject has known of cancer in the family, the more likely they were to view themselves as being at risk of cancer- this was consistent with the Hypothesis 1 & 3 of Study 2. Neuroticism, however, made no contribution to explaining the variance seen in cancer risk perception, and this finding was contrary to the stated hypothesis of emotional stability as playing a role in cancer risk perception- in this case, the Null Hypothesis 2 must be accepted.

In regards to Cancer anxiety (Table 53), there was no correlation reported between family history of cancer and cancer anxiety- as such, Hypothesis 1 must be rejected in favour of the Null Hypothesis 1. The Multiple Regression Analysis found that there is a decrease in Cancer anxiety over the course of time as a function of knowing of a family cancer history- this was contrary to the hypotheses 3, as it was expected that cancer anxiety would maintain itself (i.e be unchanged) regardless of the amount of years that the person had known of the family risk factor. As such, Null Hypotheses 3 must be accepted. Also, contrary to hypothesis 2, Neuroticism played no role in explaining the variance- Null Hypothesis 2 must therefore be accepted.

A possible explanation of this finding is that the longer people are aware of a cancer risk because of family history, the more de-sensitized they become to that risk, and therefore less anxious. Table 58 of the Results section shows that there is a positive correlation between Age and family history of cancer- the longer people have been alive, the longer is their knowledge of a family history of cancer. It appears that additional years of life give a person more opportunity to de-sensitize to the fact that they have cancer in their family history. As stated, this is contrary to what was hypothesized, as it was expected that cancer anxiety would maintain itself over time, not diminish. It was also expected that Neuroticism would play a role in maintaining cancer anxiety, however, it appears
that Neuroticism plays no such role. As such, it would appear from the Multiple Regression Analyses that people are more likely to view themselves as being at risk of contracting cancer the longer they have known of a family risk factor, but this increased sense of risk does not translate into increased anxiety- they know of the risk but do not feel anxious about it, perhaps as a result of mere de-sensitization over time.

The same analysis was carried out for CHD risk perception (Table 54). A significant correlation can be seen between family history of CHD and risk perception of CHD- as such, Hypothesis 1 is supported, and Null Hypothesis 1 is rejected. Both Years of family CHD and Neuroticism do not explain any of the variance in CHD risk perception. This is against the Hypotheses 2 & 3, which expected to see both of these variables play a role in explaining the variance in CHD risk perception. In regards to CHD Anxiety, a significant correlation was also seen between family history of CHD and anxiety about that condition- as such, Hypothesis 1 must be accepted. The study variables Years of family CHD and Neuroticism do not account for any variance in Anxious about CHD at all. This is contrary to the Hypotheses 2 & 3, which expected to see Years of CHD in family and Neuroticism both accounting for variance in CHD anxiety. As such, Null Hypotheses 2 & 3 must be accepted.

In attempting to explain the above findings, it is possible that family history of cancer was simply not potent enough to influence subjects’ anxiety levels in regards to cancer. Family history of CHD, however, was related to both risk perception of CHD and CHD anxiety. Various researchers have demonstrated that cancer anxiety increases in the short term as a result of learning of a family risk factor (Frost et al 2000; Miller 1992; Lerman et al 1991), but no one has studied this effect over time. It appears from the current results that the effect of family history of cancer on cancer anxiety diminished over time- most people are unable to continue being anxious over a condition which they continue to view themselves as being at risk of. This result makes sense in that most people in the current sample fall below the mean scores for Neuroticism on the EPQ, which according to Eysenck & Eysenck (1994) is 5.68 when male and female scores are combined. The combined mean Neuroticism score for males and females in the current sample was 5.36.
As such, a characteristic of the current sample in general was that it was not highly neurotic—this would have been aided by the deliberate exclusion of the least emotionally stable of the oldest study candidates. It is suggested that this exclusion did not occur in Grossarth-Matick’s samples, and therefore it is likely that his samples had higher mean neuroticism rates than the current sample. As such, there may have been more people who were able to sustain cancer and CHD anxiety over a long period of time by virtue of their neuroticism, producing the hypothesized ‘sleeper effect’.

The current findings may also have occurred because the methodology used was not an adequate way of indirectly testing the possible explanation of Grossarth-Matick’s results. Subjects in other categories may have provided a more meaningful test of this hypothesis, e.g. current cigarette smokers. This population is unable to escape the regular health warnings that suggest smoking to be a major cause of both cancer and CHD. As such, this population could be expected to view themselves at risk— it could be expected that the most neurotic of smokers would take the health warning messages and make themselves quite anxious as a result. Research could then answer if this anxiety maintains itself over time as a function of Neuroticism. Such a test may be a better way of indirectly answering Study Question 2 than the method chosen.

In summary, there appears to be no support for Hypotheses 1 & 2 of Study 1; support for Hypothesis 3 of Study 1; and only mixed support for the Hypotheses of Study 2. What little support of hypotheses there is in Study 2 does not make for a compelling argument in favour of the possible explanations of Grossarth-Matick’s results as proposed in this paper, however it also does not disprove the possibility being proposed. There are several possible explanations of these outcomes, and each must be reviewed in turn.

Firstly, the current findings, while not disproving the possibility of a large scale interviewer bias/treatment effect in Grossarth-Matick’s studies, could be viewed as suggesting that no such effect actually occurred. It is clear from the current study that no data suggestive of a treatment effect was found in this sample. With such a finding it is always possible that this has resulted simply because no such effect actually occurs in
reality. One could only really arrive at this conclusion, however, if there were no competing explanations as to why no effect was found in the current study. This raises factors which may have biased the reported outcomes.

**Ethical constraints producing a sample bias**

As detailed in an earlier section, due to ethical considerations the current researcher decided to not test the current hypotheses with elderly subjects for whom there was an ethical concern in revealing to them their Short IRI score- their data did not enter this analysis as they were regarded by the current researcher as being too emotionally unstable to cope with negative risk information. As such, it may be considered that the current sample was biased in the sense that the elderly subjects who most impressed the interviewer as being psychologically vulnerable were excluded from learning of their health risk scores due to the ethical concerns- only the data of the most psychologically robust elderly subjects did enter the analysis, and these people would not be ‘cases’ of the treatment effect. These people were more likely to be given a prediction of positive health and were therefore not likely to show an alarm response to the Short IRI results. The types of subjects thereby excluded from the analysis were just the type of subjects (high Neuroticism, high Type 1 & 2 scorers) who would be expected to show the alarm reaction which was the dependent variable in this study. As Grossarth-Maticek’s methodology did not exclude these people from his samples, it is proposed that a large part of his results may be explained by virtue of a large alarm reaction from the most vulnerable elderly people only. The current study did not actually test the most vulnerable elderly subjects because of this ethical consideration. The raw figures in the current study are reasonably small, however small numbers such as these are important when considering how few cases of Type 1 & Type 2 subjects there were in the sample. In addition, even if the numbers were much larger, eg. 50 elderly subjects whose data was not gathered, the implications would be the same- the most psychologically vulnerable people were not sampled, while only the most robust were sampled. In addition, it is likely that there was already a sample bias operating in the current study in that elderly subjects were taken from service clubs such as Probus. These clubs are social
organizations, holding regular meetings, outings and social events. It is likely that only
the more emotionally stable elderly people ever consider joining such a club, while the
thought of joining would give the least emotionally stable elderly people ‘the horrors’. As
such, it is possible that the broad range of elderly people who Grossarth-Matichek
managed to sample (sampling the oldest person of every second household of Crvenka)
managed to enlist the participation of a much larger amount of emotionally unstable
elderly people than did the current sampling method- the two samples may have been
very different on this single important variable.

As such, it may be concluded that the current study was not a fair test of the hypothesis,
and that a different methodology would need to be developed with consideration to ethics
and subject recruitment in order to adequately test the hypothesis. In an attempt to gain
access to less emotionally stable subjects, the current researcher did obtain permission to
request participation from out-patients to an Anxiety clinic operated by the local health
service. Letters requesting participation were given to the service, however none of the
out-patients accepted the invitation. Even if they had have completed the questionnaire
and attended the interview, it is probable that the current researcher would have
experienced the same ethical dilemma and chosen to not reveal the Short IRI scores to
them as well – as such, the same ethical concern may have prevented their data from
entering the analysis.

Heart rate as the physiological measure

Another limitation to the current study was the fact that measuring heart rate responses as
the only indicator of autonomic arousal is quite limiting. Early in the research design
process, the current author was directed to consult with a research physiologist at
Southern Cross University in order to gather information about the most useful
physiological measures of autonomic arousal. After hearing of the study design and
purpose, the stated physiologist suggested that changes in heart rate would be the most
suitable dependent variable measure. There are many precedents in the research literature
for using heart rate as the physiological measure of anxiety- Kantor et al (2001) state that
it is a common choice for researchers. While Levinson et al (1992) conclude that the
weight of evidence is overwhelmingly in support of increases in heart rate as a response to fear, anger and sadness, they also note that there have been some studies which have failed to support these associations. The reasons for these contrary finding are not evident, but may indicate a lack of universality in heart rate responses as indicators of alarm- some people may respond to alarm with different physiological responses. This possibility may have impacted upon the current results as there may have been subjects who responded autonomically in ways which were not measured by the Polar Heart Rate monitor. Blumenthal et al (1985) conclude that forearm blood flow and forearm vascular resistance were more sensitive indicators of arousal than were heart rate or blood pressure measures- they caution that either heart rate or blood pressure as an index of arousal may not accurately reflect responsivity. As another alternative to heart rate measures, Bloom & Trautt (1977) report that Finger Pulse Volume is a sensitive measure of autonomic arousal associated with anxiety.

Greenfield & Sternbach (1992) state that there is no general agreement on the shape of heart rate responses, suggesting rather that mild stimuli results in decelerations of heart rate, moderate stimuli leads to a generally diphasic response, while intense stimuli leads to an accelerative response. Quigley et al (2002) state that situations that are perceived as entailing demands which are in excess of the person’s ability to cope will be experienced as threatening and will lead to little or no increases in heart rate response, whereas challenges which are perceived as within one’s ability to cope can lead to increases in heart rate as these will marshal one’s resources.

Baldaro et al (2001) found that heart rate deceleration occurred in response to threatening stimuli. Their research entailed showing subjects 10 minutes of film footage depicting graphic surgery scenes as compared to another condition in which 10 minutes of neutral footage was presented to subjects. Baldaro et al’s (2001) results indicate a greater heart rate deceleration in response to the surgery film compared with the response to the neutral film, as the former was perceived as containing threatening images for the viewers. They view these results as being consistent with Lang, Greenwald, Bradley & Hamms findings (cited in Baldaro et al 2001), which suggest that the early stages of
information processing is characterized by heart rate decelerations while the person attends to a new stimuli. It is important to point out here that the stimuli presented by Lang et al (cited in Baldaro et al 2001) was visual, i.e a six second viewing period of aversive stimuli. They concluded that subjects showed a triphasic response in that on presentation of the stimuli there was a brief initial deceleration; this was followed by a small subsequent heart rate increase, and then a modest secondary deceleration- the average of these changes is generally lower than the baseline levels of heart rate. Boldaro et al (2001) found that the bradycardia was evident throughout the 10 minute viewing period of threatening film footage. The key issue with this research is not in suggesting that the fear experienced as a result of the threatening footage will produce a reduction in heart rate; the research demonstrates that in the act of attending to new stimuli which is not imminently threatening, parasympathetically driven heart rate deceleration occurs as an early aspect of the information processing stages. Viewing threatening stimuli requires more attention than does viewing neutral stimuli, and the bradycardia appears to occur as a result of the attending to the new stimuli. As such, heart rate deceleration can be considered an index of attention in situations where new stimuli is presented. While not presenting visual stimuli in the current study, new information was presented in the form of Short IRI results- this required the subject’s attention which could have brought the heart rate response down.

Fitness levels have also been shown to effect heart rate responses. Boucher et al (1995) studied the different levels of heart rate responsiveness between subjects who were assessed as being aerobically fit (by virtue of extensive aerobic training in the four years prior to participation- mean heart rate for this group was 58 bpm) with subjects who had no history of aerobic training but who had naturally occurring low resting heart rates (mean heart rate of 58 bpm). Both groups were compared with a control group who had a mean resting heart rate of 69 bpm and no history of chronic aerobic training. Subjects were given an arithmetic stress task while heart rate data was measured during a 5 second pre-task phase, for five 4 second periods during the task, and for 5 and 10 seconds during recovery from the task. Boutcher et al (1995) found that for all of the groups in their study, there was an initial increase in the heart rates during the early phases of the task, a
leveling out near the completion of the task, and a drop in heart rate after task completion. For both the aerobically trained and the inherently low heart rate subjects, there was a significantly lower absolute heart rate during the task and after the stressor when compared with the control group. Boutcher et al (1995 p43) state that “persons with inherently low resting HR display a bradycardia response during psychological stressors that is similar to that of aerobically trained individuals”. They conclude that lower absolute heart rates during and after stressors is influenced by levels of aerobic fitness as well as genetic inheritance. The average age of subjects in the current study was 40 years old with a standard deviation of 17 years. Many of the subjects were young university students, who are in the age group where one would expect a greater degree of fitness when compared to the older age groups. Sixty of the subjects in the current study were under the age of 40.

Zahn et al (1990) studied the heart rate responsiveness of people who have a genetic loading for anxiety disorders by virtue of their parent’s anxiety disorders. These people were compared in a standard laboratory stress test with subjects who had no parents with an anxiety disorder. They found that there were many significant correlations between autonomic indices, including heart rate, and measures of state and trait anxiety in the sample at risk of developing an affective disorder. This relationship was not found amongst the normal controls, i.e indices such as heart rate did not correlate to psychological measures of anxiety in the control group of subjects’. Zahn et al (1990 p.108) conclude that in some high risk subjects, moderate stress produces increases in a psychobiological complex which includes subjective anxiety and depression as well as a generalized ANS reaction. This research demonstrates that the level of heart rate response to stress may also depend upon the level of proneness to affective disorders in subjects according to their genetic inheritance as well as their levels of fitness. Zhan et al (1990) conclude that with normal populations, the relationship between anxiety and autonomic indices have not been consistently observed, whereas it has been with subjects experiencing anxiety conditions. The mean EPQ score for Neuroticism in the current sample was 5.4 out of a possible score of 12- as such, it could be argued that this was not a population prone to neurotic conditions such as anxiety disorders. The people who were
assessed as being prone to an anxiety disorder (and who thereby would be expected to show a large heart rate response) were the elderly subjects who were excluded from the experimental phase of data gathering for ethical reasons.

Subject’s prior life experiences have been found to effect heart rate responses. In a study of Vietnam veterans, including a group who had experienced PTSD and a group that had not, Beckham et al (2002) report that heart rates were seen to increase when subjects were asked to relive a non trauma related anger experience. Such an experience is considered to be stressful, and the reliving of it was seen to stimulate autonomic arousal as measured by heart rate. Compared to the non PTSD veterans, the PTSD group were quicker to anger, reported greater anxiety and anger, and showed higher mean heart rate responses during the relived anger task. As such, it can be concluded that traumatic psychological experience predispose one to a higher resting heart rate as well as a larger heart rate response to stressful events when compared to people who have not had a traumatic episode. Buckley et al (2001) report conclusions from a meta-analysis of 34 studies which examined the role which PTSD may play in higher levels of basal cardiovascular activity. From their meta-analysis, Buckley et al (2001) found an association between PTSD and elevations of basal heart rate and diastolic blood pressure when compared to groups who had not experienced PTSD. The differences in resting heart rates were the greatest with the PTSD samples having average resting values around 5 beats per minute faster than the non PTSD group. None of the subjects in the current study reported experience in the Vietnam war. Some subjects did report traumatic life experiences, such as the deaths children or siblings. Several of the older subjects did report WWII experience, and as stated earlier were excluded from the experimental data gathering phase as a result of the PTSD like presentation which they gave. It is these subjects who could be expected to show greater heart rate responsivity to a psychological stressor.

Heart rate response is sensitive to regular behaviours, such as communicating. Lynch et al (1992) state that advances in computer technology have permitted the ongoing, non invasive monitoring of blood pressure and heart rate while the subject is speaking. They
refer to this as a ‘robust phenomenon’ which has been repeatedly demonstrated in a wide range of subjects and situations in both experimental and clinical studies. The increases in blood pressure ranges from 10-50% within 30 seconds of the initiation of speech; when speech stops, blood pressure returns back to pre-speaking levels. More accurately, this appears to not be a function of speaking per se, as the same effect has been demonstrated with deaf individuals who are monitored while using sign language- as such, it seems to be a function of communicating (Lynch et al 1992). An additional observation which has emerged from research is the finding that when people are beginning to approach, allude to or openly mention feelings of helplessness and hopelessness, their blood pressure drops by as much as 50% (Lynch et al 1992). Lynch et al (1992) suggest that this response is related to discussion of situations which involve overwhelming sadness where neither fight nor flight is possible, leading to a reflex ‘conservation of energy’ rather than autonomic arousal and preparation for challenge. It is possible that the subjects in the current study who learnt of a risk status according to the Short IRI, experienced this as a hopeless situation in which escape was not possible (more so as the risk entailed one’s own personality- how do you escape your own personality?). Lynch et al’s (1992) conclusions would suggest that this would produce as much as a 50% decrease in heart rate as the organism reflexively attempts to conserve energy in response to the threat.

It is possible that heart rate is a more sensitive measure of stress in women than in men, as some research has demonstrated greater increases in heart rate responses for women in response to stress (Valdimarsdottir 2002.) Steiner et al (2002) state that higher baseline heart rates in women is well established, but the gender effects on physiological reactivity show a mixed picture. Many studies have demonstrated larger heart rate increases in women, however the salience of the stressor to each gender may play a role. For example, a video showing hunting scenes led to larger heart rate increases in men than in women (Steiner et al 2002)- some stressors may be more salient to one or other gender. For example, Quigley et al (2002) observed that women were more likely to report feeling threatened by a verbal arithmetic task than were men.
Huwe et al (1998) report that the research literature shows an inconsistent relationship between anxiety and physiological responses, and the relationships are often related to trait anxiety rather than to state anxiety. They claim that some studies show a positive relationship between heart rate and anxiety, some show a negative relationship, while most find that no relationship could be observed. Huwe et al (1998) do state, however, that a crucial factor in the finding of relationships between heart rate and anxiety is the time at which the measures were taken. Some researchers take heart rate measures before, during and after the stressful event (eg. Huwe et al 1998); at one second intervals during a stressful event (eg. Reidbord & Redington in Port & van Gelder 1995; Simons et al 1999); at specific intervals, e.g for 5 seconds pre-task, at five four second periods during the task, and for 5 ten second periods during recover (Butcher et al 1995); after 2 minutes of a base line period, as well as 5 and 10 minutes into a speech task (Steiner et al 2002); and mean heart rate scores from one minute to the next (Harris 2001). It would make sense that each researcher evaluates what time of measurement would make the most sense in relation to the effect they are expecting and the nature of the task. Perhaps those that fail to do this adequately also fail to arrive at positive findings.

In summary, the possible heart rate confounds not controlled for in this study include:-

- heart rate response to stress may depend upon the level of proneness to affective disorders in subjects according to their genetic inheritance (Zahn 1990).
- heart rate response may depend upon subject’s levels of fitness (Boutcher et al 1995)
- heart rate response many depend upon prior life experiences which subjects have undergone, eg. PTSD (Beckham et al 2002; Buckley et al 2001).
- the salience of the stressor to each gender was not assessed (Steiner et al 2002).

Future research in this area would be well served by taking multiple measures of physiological arousal, not just relying on changes in heart rate.
As stated previously, heart rate was chosen as being the physiological dependent variable on the basis of advice provided by an exercise physiologist at the current author’s university. It may have been more profitable to invest in more extensive equipment to take a wider range of physiological measures rather than to just rely on one measure. As a result of using only a heart rate measure, it is unknown if there were more physiological indicators of alarm which would have informed the results, but were not measured. As such, the results may present only part of what actually happened for the subjects in the experiment.

The heart rate measuring device
As stated earlier, the experimental physiologist consulted at Southern Cross University suggested the measurement of heart rate as the dependent variable for this study. In addition, he recommended the purchase of a Polar Heart Monitor. The reasons for the recommendation were that it is an unobtrusive device, fitting easily over the subject’s chest; it can be self fitted by the subject; it records the heart rate measurements in a form which can then be downloaded onto computer software via an interface device; the software can produce a chart showing the heart rate measures during the interview process, and allows the heart rate readings to be converted into a form which could be transferred to an Excel spreadsheet from which the basic statistics could then be calculated. The Polar Heart Rate Monitor did prove to be very useful in these regards. At reasonable expense, the university purchased this device and the data collection commenced.

When conducting the early interviews, it became apparent that the shortest time span between heart rate measures which the Polar Heart Rate monitor could manage was 5 seconds. This would not be a problem in the application for which it was devised, physical exercise. However, in the psychological research literature as opposed to the physiological research literature, it can be seen that the measurements of heart rate in response to psychological experiences are typically taken in a much smaller time frame, eg. at one second intervals during a stressful event (Reidbord & Redington in Port & van Gelder 1995; Simons et al 1999). Midway into the data collection phase, the current
researcher further investigated studies in psycho-biology and found that most of them used devices that were able to take measures of heart rates each second- many of the studies showed ‘peaks’ or ‘spikes’ in heart rate on presentation of psychological material on a second to second basis. The spike, as an indicator of heightened emotional arousal may only last for a second before disappearing. Initially, it had been hoped that the current study would also be measuring these spikes in heart rate as the method of demonstrating a change in heart rate resulting from psychological experience. It became apparent that the Polar Heart Rate monitor was simply not sensitive enough to capture the brief spikes in heart rate which other studies show result from psychological experience.

As such, it would appear that the five second interval of sampling the heart rate may indeed be useful in exercise physiology research, but is somewhat inadequate in the psychological research of emotions. Unfortunately, many interviews with the Polar Heart Rate monitor had already been conducted by this time. An ever present difficulty in conducting this type of un-funded research is gaining access to enough subjects. As such, it was decided to press on with the device already used in dozens of interviews as it may have proven impossible to then re-recruit more subjects if the process was to begin again with a more sensitive heart rate measuring device- there is a finite amount of people willing to volunteer their time for research purposes, and the current author had already sampled many of those available.

Rather than this limitation rendering the results invalid, it does render them somewhat incomplete and provides only a partial picture of how each subject responded to risk information. For example, the Polar Heart Rate Monitor would only record an elevated heart rate if the response lasted for more than 5 seconds. As Levenson (1992) points out, it is possible for emotional reactions to come and go in a shorter time frame than 5 seconds. On realizing that the data collected was unable to detect spikes which came and went in less than 5 seconds, another method for calculating changes in heart rate needed to be developed. As such, it was decided to compare the mean heart rates from the pre-news segment of the interview with mean heart rates from the post-news segment of the interview. While this is a creative way of making use of the limited data, it is a somewhat
insensitive method and has yielded results that are less meaningful than would have the approach originally envisaged, i.e. having heart rate samples taken each second in order to detect a spike in heart rate.

Huwe et al (1998) state that a crucial factor in the finding of relationships between heart rate and anxiety is the time at which the measures were taken. Some researchers take heart rate measures before, during and after the stressful event (eg. Huwe et al 1998); some at one second intervals during a stressful event (eg. Reidbord & Redington in Port & van Gelder 1995; Simons et al 1999); some at specific intervals, e.g for 5 seconds pre-task, at five four second periods during the task, and for 5 ten second periods during recover (Butcher et al 1995); and others after 2 minutes of a base line period, as well as 5 and 10 minutes into a stressful task (Steiner et al 2002).

If a more thorough a measurement of changes in heart rate was used, the current study would have been better served by following the example set by Steiner et al (2002), Huwe et al (1998), Butcher et al (1995) amongst others and utilized a baseline period prior to the interview to establish pre-existing heart rates against which the experiment effect could be compared. The use of a baseline measure may have yielded more useful data in terms of demonstrating the experiment effect than the method employed in this study. When the methodology was being designed, the current researcher was under the impression that the Polar Heart Rate monitor was sensitive enough to detect a short term spike in heart rate as the dependent variable- as such, a baseline measure was considered unnecessary as it was anticipated that the analysis would be utilizing a measure of heart rate ‘spikes’ and troughs.

**The Sleeper Effect**

Another possible explanation for Grossarth-Maticek’s results may be termed the ‘Sleeper effect’ (Lang and Lang, in Rosenberg & Turner 1981), whereby the levels of cancer and CHD anxiety increased *over time* from the point of the interview. In effect, the ‘seed’ of anxiety may have been planted in the context of the interview via either an overt statement of the Short IRI risk status or an inadvertent interviewer effect. This perception
of increased risk may not have had an initial effect on his subjects (therefore no change observed in the current study), but this effect may have grown with time as the social and somatic cues for cancer and CHD increased with the subject’s advancing years. For example, the normal increase in health problems which occur with age may have been perceived by Grossarth-Matick’s Type 1 and 2 subjects as being evidence of cancer or CHD proneness, leading to powerful increases in anxiety (and subsequent poorer immune function) in the years after the initial interview. The perception of proneness may have been a secondary concern to more immediate life challenges at the point of the interview, only to emerge years later when the ill-health of self and others began to re-elicit the anxiety. If this were the case, there may have been either no elevations or only insignificant elevations in heart rate at the point of developing the risk perception in the interview. This possibility has not been tested in the current methodology, and the issue of the Polar Heart Rate Monitor’s inadequacy would not be relevant to this possibility.

The sleeper effect hypothesis might suggest that that an initial response to learning of risk is simply stage one in the process and the sleeper effect is stage two, leading to higher and maintained anxiety at a later time. This possibility could very well be consistent with the lack of initial response as seen in the current study.

Support for this possibility comes from Miller (1996), who states that Monitors and Blunters do not differ on initial psychological or physiological measures of arousal when exposed to threatening information. However, Blunters tend to diminish their anxiety over time by either not seeking out information or cues concerning the threat, and/or by habituating to the threat. In contrast, Monitors sustain higher levels of arousal over time as they are seeking more information and they show less habituation to the threat. Even in the absence of ongoing threat cues, monitors will still experience threatening signals while remaining unaware of safety signals. It is possible that if Grossarth-Matick’s most vulnerable probands did experience more anxiety from perceiving their risk status in the time subsequent to the interview, they were likely to have been high in this characteristic of monitoring. Age is the highest risk factor for both cancer and CHD. The mean age of the current sample was 40 years, with a standard deviation of 17 years, whereas the mean
age for Grossarth-Maticek’s Yugoslavian and Heidelberg samples were 60 and 50 respectively. People of this age group can be expected to experience more bodily cues to ill-health in the years following the interviews, i.e the 15 year follow up saw the mean ages rise in the Yugoslavian and German samples to 75 and 65 respectively. These are the stages of life when people are confronted with internal somatic cues for ill-health as well as cues relating to illness and deaths of their cohorts- ample opportunities for monitors to respond to these cues with elevated anxiety levels. To further complicate the picture, high Monitors who score low on a measure of avoidance eventually show a decrease in anxiety as they have more opportunity to fully process the threat and adapt to it with effective coping and problem solving strategies. The high Monitors who are also high on measures of avoidance are the individuals who fail to de-sensitize to the threat, fail to adapt, and consequently maintain elevated levels of sustained arousal and anxiety (Miller et al 1995).

In contrast to Grossarth-Maticek’s samples, most of the subjects in the current sample were not in an age group which poses risks for cancer and CHD- it is viewed as a distant possibility, but rarely an immediate concern to younger people. There is the possibility that due to lack of immediacy of the threat of cancer and CHD, a sleeper effect is the more likely route to heightened perception of risk and anxiety. As the current study was looking for an immediate reaction to health risk information, it could have completely missed the pathway by which Grossarth-Maticek’s subjects may have developed anxiety over a protracted period of time. In order to address this limitation of the current study, a longitudinal methodology would be required to ascertain if such a sleeper effect operates over time, or even over a life-span.

**Range of possible responses to risk information**

Emotions are complex phenomenon which involve an affective, physiological and cognitive processes. Levinson (1992 p23) makes the point that

“emotion relevant ANS activity is similarly imposed on an ongoing stream of physiological activity occasioned by the ANS acting in service of its many
masters, both internal (eg. homeostasis, response to metabolic demands) and external (eg. orientating, defense). Isolating an emotion and a segment of associated physiology for study is difficult at best.”

The attempt has been made in this study to isolate a particular physiological event and relate it to a psycho-social event, assuming the existence of a particular emotional state on the basis of an increase in heart rate. Levinson (1992) would suggest that this is a very difficult thing to do. Individuals differ in their emotional reactions to the same situations. What may deeply affect one individual may fail to move another; a stressful interview situation may elicit anger, fear or depression in different individuals, or any combination of these (Philippot 1993). It has been assumed in the current study that the only likely reaction to finding oneself with an at-risk status is alarm, however it is possible that there may be a range of responses. Quigley et al (2002) state that heart rate response, whether it be accelerative or decelerative, will depend upon the cognitive processing of the stressful situation- the cognitive processing of the nature of the stressful situation will influence the heart rate response. Situations that are perceived as entailing demands which are in excess of the persons ’ability to cope will be experienced as threatening and will lead to little or no increases in heart rate response. However, situations which are perceived as entailing demands which are within the personality to cope with will be perceived as a challenge and lead to increases in heart rate response. These conclusions were arrived at by Blasacovich et al (cited by Quigley et al , 2002) in which heart rates were measured during a goal relevant, motivated performance task. Challenge appraisals prior to the commencement of the task lead to a mobilization of resources relevant to the task action required, involving a large increase in cardiac reactivity. On the other hand, a threat appraisal is associated with a poorer energy mobilization and a pattern of modest cardiac reactivity. In their own study, Quigley et al (2002) found that as their subject’s perceptions changed from threat to challenge appraisals, cardiac reactivity increased accordingly. New information about the task was presented to subjects in the anticipation stage of the procedure, leading to alterations in perceptions from threat to challenge.
In relation to the receipt of a poor health prognosis from the Short IRI, some people may respond with: relief (“I will finally be looked after and absolved of my responsibilities”); some with satisfaction (“I always thought I was prone to cancer/CHD, and I was right!”); some with optimistic determination (“this will be my biggest challenge but I will overcome it”); some with skepticism (“why would anyone believe this nonsense?”); some with pessimistic resignation (“now it is just a matter waiting until I become unwell”); some with fatalism (“it is all in the lap of the gods- whatever will be, will be”); and others with problem solving coping (“what do I need to do from now on to ensure that I stay healthy?”). The self report measures may not have captured this range of responses.

All interactional models of stress emphasize the importance of individual perceptions of stressors, rather than assuming that one event means the same thing to all people. By way of example, one subject in the study who was given her Short IRI score (Type 1) was not surprised or alarmed by this, as she had for many years been convinced that she was in fact cancer prone due to psychological factors- this was a very resilient belief as she resisted the current author’s attempts to challenge the perception with research findings contrary to her strongly held views.

**Perceptions of the interviewer**

It is possible that few of the hypothesized outcomes were found in this study as the interviewer was simply not perceived by subjects as being a credible authority. Covelo (1992) suggests that in order to be able to successfully convey a perception of risk, the following characteristics need to be perceived of the communicator:

- perceived caring and empathy
- perceived competence and expertise
- perceived honesty and openness
- perceived dedication and commitment.

Any of these characteristics may not have been perceived by the subjects of the interviewer, leading to a lack of acceptance of the Short IRI scores. It may be that the purpose of the study design was too apparent to the subjects and they were aware that
their emotions were being manipulated for the purpose of heart rate measurements. As such, while all of the Full Interviews only proceeded when the subject stated that they felt an adequate degree of trust and understanding in the research purposes, they may have been privately suspicious but overtly acquiescent.

The interviewer in this study obviously harboured some strong views about what may have produced Grossarth-Maticek’s results, as seen in the hypotheses. These views may be seen as being somewhat sceptical about the nature of Grossarth-Maticek’s methodology, results and the conclusions he drew from them. It is possible that at some level of perception, subjects in the current study detected that the current interviewer did not necessarily believe in Grossarth-Maticek’s results and conclusions. This may have differed to the 100 interviewers used in Grossarth-Maticek’s study. As they were post graduate psychology students, and most likely relatively young, it is possible that they were impressed by Grossarth-Maticek and his theory, especially if he was seen as being somewhat charismatic as Eysenck (1988) claimed. In contrast, the current experimenter is in his 40’s, has been practicing psychology for the past 19 years, and is therefore perhaps not as impressionable as were Grossarth-Maticek’s interviewers. Of course, this is only conjecture, but it may have provided a subtle extraneous variable in Grossarth-Maticek’s studies which were not present in the current study. If his interviewers sincerely believed that the Short IRI results were able to predict incidence of and death by cancer and CHD, their ability to inadvertently convey this impression to subjects, or their ability to sincerely inform subjects of their risk status would have been enhanced. This may have been a factor in the production of a treatment effect in his studies which was not present in the current study.

In summary, the hypotheses of the current study were largely unsupported. The reasons for this outcome have been discussed above. They include:- the nature of the sample, ie. generally younger than Grossarth-Maticek’s sample, and for the most part with no life experience of being a combatant or a civilian in a war zone; the ethical dilemma for the current experimenter, which saw him exclude from the study the most vulnerable elderly subjects who were those most likely to display the effects hypothesized, ie. a selection
bias in the current sample; the heart rate monitor used, being unable to detect changes in heart rate with a second by second measurement; the reliance on one autonomic measure only, ie. heart rate as the measure of autonomic arousal rather than a range of measures; a range of possible confounds with heart rate measures; the possibility that changes in risk perception and disease anxiety occur over a period of time, rather than as an instant reaction to new information, ie. a sleeper effect; possible questions around the current interviewer’s credibility with subjects in providing a health prediction; it is possible that there are a range of possible reactions to perceiving oneself as being at risk of chronic disease, other than just an alarm reaction. In regards to Study 2, there is the possibility that a chance of chronic disease for reasons of family history may not seem real enough to relatively young subjects- a different naturally occurring at risk population could have been sampled, such as cigarette smokers.

As the current research project has been an attempt to make sense of Grossarth-Maticek’s reports, the Conclusion that follows will examine fundamental differences between the current sample and Grossarth-Maticek’s samples. This difference may account for his remarkable results, and the lack of results reported in the current study. In addition, recommendations for future studies will be made.
Chapter 16
Conclusion

As made clear in the Discussion section of this paper, the hypotheses for the current study were largely unsupported. A possible contribution to this outcome could relate to differences in Grossarth-Matichek’s sample to the current one on a range of factors, including cultural ones. Terada et al (2000) found that Grossarth-Matichek’s Rationality/antiemotionality scale did not correlate with a range of chronic health conditions in a cross sectional community study in Japan as they were reported to do so in Yugoslavia and Heidelberg. The authors believe that cultural differences between these European and Japanese cultures were responsible. As such, it would appear that culture plays a role in determining the applicability of Grossarth-Matichek’s findings. It is unknown by the current author how similar or different German and Yugoslavian people are to Australians.

Other cultural factors may have made a difference between the current sample and Grossarth-Matichek’s samples. In his first study Grossarth-Matichek (cited in Eysenck 1988) sampled the oldest person in each household of a Yugoslavian town of 14,000 people, Crvenka.. As this sampling occurred in the late 1960’s, the demographics of the sample would necessitate that each of these people had been affected first hand by events during the Second World War as well as the turbulent political/military experiences of the Cold War era. Eysenck (1988) stated that the age range of the Yugoslavian probands was between 50-65, meaning that in 1940, these people were aged between 20-35. In 1941, Yugoslavia was invaded by German forces and thereafter militarily occupied (Roberts 1996). Few invasions are un-traumatic to a population. Following the German invasion, a three-way civil war between Croations, mainly Serb communists (led by ‘Tito’) and royalists erupted. The communists proved victorious in 1945 and Tito reigned as dictator for the ensuing decades. In the immediate aftermath of WWII, many parts of Europe saw large scale massacres as retributions for war time allegiances. Roberts (1996 p. 509) states that,

“ it was said that in France more perished in the ‘purification’ of liberation than in the great Terror of 1793, but even that comparison is dwarfed by
vengeance taken in Yugoslavia, where old enmities between communities opened up by wartime decisions to co-operate with or fight the Germans were now driven home yet more deeply by new massacres”.

Under Tito’s dictatorship, the above mentioned fissures within Yugoslavia were contained until 10 years after his death when, in 1990 they erupted with a re-emerging civil war. These tensions had apparently never been far from the surface in the Yugoslavian national psyche. The population sampled by Grossarth-Maticke in 1969 were people who had experienced the traumas of German invasion and occupation; the civil war which ensued; the retribution massacres following WWII; life under Tito’s dictatorship and the Cold War; and the tensions of an inherently fractured and divided country. In discussing the negative life events of his probands during the full interview condition, it can only be imagined that they had much to reveal to the interviewer.

Grossarth-Maticke’s second sample (reported in Eysenck 1988) was taken from Heidelberg, West Germany in the early 1970’s. The age range for this sample was younger than that of the Yugoslavian sample, being 40-60 years of age (average age was 50- Eysenck 1988). At the commencement of data gathering, it would have been around 25-30 years since the events of the Second World War, meaning that in 1940 these probands would have been aged between 10-30, with an average age of 20. Ten year olds in 1940 Germany were primary school aged children, while the 20-30 year olds were their parents as well as participants in the armed forces. Like the Yugoslavian sample, it is possible that many of those in Grossarth-Maticke’s Heidelberg sample were also war traumatized in a way that the current subjects were not. Many of the men would have been active combatants and experienced associated war stress, and many of the women could also have been war traumatized (by their proximity to armed conflict and aerial bombing, their fear of destruction, and grief from the loss of loved ones). In regards to the effects on combatants, Grossman (1999) studied war trauma amongst survivors of 20th century conflicts. He states that during World War II, after 60 days of continuous combat, 98% of all soldiers became psychiatric casualties. This level of psychological damage began in World War I with the unprecedented magnitude of horror due to sustained violence, threat and trauma for millions of people. Grossman (1999) states that the remaining 2% of servicemen
that were not psychiatric casualties after 60 days of continuous combat were psychologically disturbed to begin with.

There is considerable evidence from studies conducted on Vietnam veterans suggesting that such stressful war time experiences can have long-term, lasting effects on the person’s stress reaction and associated endocrine system as well as neural functioning and structure. PTSD is a relatively recent construct and therefore largely unrecognized in the WWII population, although the same phenomenon was known under such terms as ‘shell shock’ and ‘combat fatigue’ (Comer 1995). PTSD can result from experiences in which an actual or threatened serious injury or death occurs to oneself or associate. Comer (1995) states that as many as 31% of Vietnam veterans experienced psychological harm after combat serious enough to qualify as PTSD, while a further 23% suffered at least some of the symptoms of PTSD; in addition, 26% of women who served in Vietnam (as non combatants) suffered from PTSD, and a further 21% suffered from some symptoms. As can be seen from these figures, the experience of war is extremely stressful. Khalsa (1997) describes the difficulty which Vietnam veteran PTSD sufferers have in turning off their biological responses to stress. He posits a ‘feedfoward’ mechanism in which, due to traumatic experiences, an excessive amount of cortisol is released into the blood supply which interferes with the hippocampus’ ability to instruct the hypothalamus to ‘switch off’ the secretion of cortisol via a feedback process. This results because the hippocampus is especially sensitive to the damaging effects of excessive cortisol levels, resulting in a 20-25% loss of neurons in that brain structure in older people as a result of excessive cortisol. The degenerative cascade which ensues means that excessive amounts of cortisol damages the brain’s ability to reduce the secretions of cortisol, leading to an ever increasing level of secretions. McEwan (1998) states that this reduces the hippocampus’ ability to reliably and accurately contextualize memories, resulting in a decreased ability to access the information required to determine if a situation constitutes a threat or not. The net effect is that the stress response becomes less regulated and inhibited, and more situations are perceived by the sufferer as being threatening and thereby stressful- anxiety increases as a result, and the person experiences a decreasing ability to ‘switch off’ cortisol and to relax, even when not in a stressful situation. Khalsa (1997) reports research in which Vietnam veterans suffering from PTSD demonstrated this difficulty in ‘turning off’ their biological
responses to stress as assessed by a range of measures, e.g. they produced 40% less of the chemicals required to switch off the feedforward mechanism. McEwan (1998) reports that the failure to turn off the sympathetic activity after a stressful event has passed is a feature of age in laboratory animals, and there is accumulating evidence of this occurring in humans. He speculates that the allostatic load over a lifetime, reflective of stressful experiences, may cause the regulation of the HPA axis to wear out or become exhausted.

In Grossarth-Maticek’s Yugoslavian sample, the female proband’s ages put them in the demographic of those who experienced the emotional strain of caring for children at a time when war was endangering all of their lives. For the non combatants in Grossarth-Maticek’s Heidelberg sample, their experience of WWII would have been as either terrified children (as young as 10 years of age), or as young women (aged between 20-30 in 1940), also with parenting responsibilities. Their trauma would have increased particularly in the final stages of WWII as the Allies advanced and bombing raids on Germany became more prevalent and unpredictable. Roberts (1996 p.498) states that

“…terrible destruction had been inflicted on German cities by a great air offensive…. much of historic Europe was literally and figuratively in ruins”.

Heidelberg itself somehow managed to not be bombed during WWII, however as the nearby cities of Frankfurt, Dusselldorf and Cologne were being bombed, it would have been an anxious time for the inhabitants- fear of impending bombing would have been intense and the threat of destruction ever present. In addition to raising their children under these conditions, like many women in other warring countries, German women were also living with the stressor of their men (husbands, boyfriends, fathers, sons and brothers) being conscripted into the war effort and dying in large numbers. Other non combatants from WWII were found to have experienced PTSD reactions- Kuch and Cox (1992) report a study which examined 124 survivors of Nazi concentration camps, concluding that 46% met the diagnostic criteria for PTSD many years after their experiences. Is it at all possible that Yugoslavian and German civilian populations were not traumatized by WWII?
In considering the effects of war zone experience on civilian populations, de Jong et al (2004) state that a number of studies have demonstrated the effects of multiple exposures to traumatic events that are associated with higher levels of symptoms of PTSD. They cite intensity of a traumatic event, the severity of the incident, and the extent of the physical injury as contributing factors to PTSD in civilians. de John et al’s (2004) study of lifetime events and PTSD in 4 specific post-conflict settings (Cambodia, Gaza, Ethiopia, and Algeria) demonstrated that conflict-related events after age of 12 years were significantly related to PTSD in all 4 samples. Further evidence of trauma to civilians in war zones is seen in the aftermath of the Soviet invasion of Afghanistan in 1979- a camp of over 110,000 civilian refugees formed in a northwest province of Pakistan near the city of Peshawar (Grinfeld 1999). As many as 90% of the refugees were assessed as suffering symptoms linked to post-traumatic stress such as nightmares, night terrors, sleeplessness, memory loss, physical and behavioral problems, depression and anxiety. Michultka et al (1998) studied the effects of war trauma on a civilian population from El Salvador. They report that 68% of the refugees studied met the diagnostic criteria for PTSD. These effects of trauma occurred to a non combative population as a result of witnessing atrocities including the deaths of wives, children, neighbors and friends. Although studying the war zone trauma amongst certain ethnic populations, de Jong et al (2004) state that research has revealed a ‘universal vulnerability’ to this trauma amongst both Western and non Western populations. In light of findings such as these, a question worth pondering is how much (undiagnosed) PTSD was prevalent in the populations from which Grossarth-Matick drew his samples? It may have gone largely unnoticed simply because the whole population was more or less traumatized. Could this factor allude to the unique but unknown differences in Grossarth-Matick’s sample which Amelang (1991) suggests may account for his results?

How badly affected by the ‘feedforward’ mechanism described by Khalsa (1997) a person becomes is dependent upon the amount and severity of chronic stress which they have experienced. Apart from the stress factor, people in the middle age to older age group are considered to be more vulnerable to this degenerative process for a variety of reasons.
“the more damage to the brain a person has suffered, the harder it is for him or her to "turn off" stress. The part of the brain that "shuts off" cortisol production- thereby reducing the detrimental effects of stress- commonly deteriorates with age. When this happens, the person reacts even more strongly to stress-and therefore suffers even more damage to the shut off mechanism. Thus a deadly downward spiral occurs.” (Khalsa 1997 p 52)

It is possible that simply due to demographics, Grossarth-Maticek’s samples were derived from a population of people who had experienced a heightened level of trauma due to their proximity to WWII- in modern parlance, sufferers of PTSD. Considering the lifetime experiences which his Yugoslavian and German subjects endured, it is almost inconceivable that the most psychologically vulnerable of these people were un-affected in terms of the degenerative processes described by McEwan (1998) and Khalsa (1997).

As stated earlier, most of the subjects in the current sample have not had the same life experiences by virtue of their ages and nationality. The only subjects that did have similar life experiences to Grossarth-Maticek’s probands were the most elderly subjects from the Probus clubs- some of these people revealed WWII service experience, and some commented that their nerves had been ‘shot’ ever since. These were the people who were excluded from the experimental data gathering and full interview for the ethical considerations described earlier. Their data did not enter the experiment, but they are likely to have been the most similar to Grossarth-Maticek’s subjects.

Obviously, not all of Grossarth-Maticek’s subjects were emotionally unstable or war traumatized. However, the current hypothesis has stated the possibility that it is only the most emotionally vulnerable subjects who could have been affected by the proposed treatment effect. Those who were more psychologically resilient to the effects of WWII experiences would not have been the subjects assessed as at risk of cancer and CHD; would not have been the most emotionally unstable probands; and would not have developed a sense of impending risk as a result of the interview process; nor were they likely to have gone away with an altered risk equation for
cancer and CHD, and therefore were not the people likely to have ruminated and worried over this possibility in the ensuing years.

Had Grossarth-Maticek sampled the youngest adult in each home, he may have been sampling a far less traumatized population, with less risk of developing a treatment effect from the data collection process. The responses of his probands may have looked more like the typical responses of subjects in the current study, which was minimal and inconsistent (albeit measured by a less than adequate heart rate monitor device). Had this been the case, Grossarh-Maticek may have found less remarkable results, only a moderate role for psychological factors in the contribution to cancer and CHD. Without well designed research methodologies, this must remain conjecture only.

**Future studies**

In order to more fairly test the hypothesis raised in this study, a superior methodology to the current one would need to be devised. In regards to the sample, the subjects in the study should all be in the elderly age group. There may be age dependent factors related to perceptions of risk and cancer/CHD anxiety which are not captured when studying a younger age group, e.g. the deaths of partners and friends from cancer and CHD; the increase in bodily cues relating to potential symptoms of cancer and CHD; approaching the age of one’s parents who died of cancer or CHD. Again, in order to more fully replicate Grossarh-Maticek’s study, subjects should be taken from a population who have had active war experience (either as a combatant or as a civilian in a war zone). A second sample could be taken from a population who have not had such a life experience, so that comparisons of the effects of that life experience could be measured and quantified.

The ethical dilemma stated in an earlier section would not be eliminated by this methodology however. The fact would remain that it is hypothesized that in conducting the experiment, some subjects would suffer as a result, leaving the interview with an enhanced or introduced perception of risk and subsequent anxiety. The subjects who would be expected to suffer this the most (the least emotionally stable, highest Neuroticism scorers) are just those for whom a treatment effect is expected. The treatment effect itself is potentially psychologically damaging. One
wonders how this treatment effect could be assessed without causing psychological damage? The researcher may confidently claim, as did the current one prior to administering the interviews, that s/he can ‘un-do’ the perception of risk created in the interview. However, the experience of the current researcher is that when it came to debriefing the least emotionally stable elderly subjects, he was so unconfident of his ability to do this (after 19 years of experience in applied psychology) that he decided against presenting the disturbing information to particular subjects. His ethic was that the potential for information gained in following the intended methodology was outweighed by the prospect of creating a sense of risk in an elderly person- and that this perception of risk could in fact be a factor in producing excessive amounts of anxiety which may damage the immune function of the person, and thereby make them more vulnerable to cancer and CHD. Who would approve of their elderly parents being subjected to a stressful experience for the sake of a research project, regardless of the scientific value of the findings? Without a new methodology to solve this dilemma, it would seem very difficult to test for this treatment effect with the population for whom it is resumed to operate.

A possible answer to this dilemma could entail a double blind study in which naïve interviewers conduct the interviews. In order to faithfully replicate Grossarth-Matick’s research, ideally the interviewers would be recruited from the same demographic as Grossarth-Matick’s interviewers, i.e relatively young post graduate psychology students. As Eysenck (1988) states, the interviewers who were most able to predict incidence of cancer and CHD by Short IRI measures were those who endorsed Grossarth-Matick’s theory- in order to test the current hypothesis it would be important to replicate this influence. That is, recruit post graduate psychology students who are favourably disposed to the notion of psychological factors playing a role in the causation of cancer and CHD, and then train them in Grossarth-Matick’s theory before administering the interviews. As stated, it would be important to have them blind as to the real purpose of the study, i.e to test for an interviewer bias and therefore treatment effect.

As with the current study, subjects would be tested for their self reported perceptions of cancer/CHD risk and anxiety prior to the administration of the interview. In regards to measures of physiological arousal, subjects would need to be connected to a range
of physiological measures such as heart rate monitor (which takes a measure each second), a blood pressure measure as well as Finger Pulse Volume. This would ensure a far more sensitive assessment of changing levels of emotional arousal and would capture the very peaks of arousal in the small time frames involved. Measures of fitness, traumatic life experiences, genetic predispositions to anxiety disorders, and the presence of anxiety disorders would need to be taken. Relying on a wider range of more sensitive measuring equipment, a baseline measure of autonomic arousal may be gathered prior to the tasks involved in the interview. As a such, a simple ABA approach could be used in which the baseline measures are compared with measurements from the ‘treatment’ period (administration of the test and learning of Short IRI scores); and then compared with measures taken from a debrief period where the perceptions of risk are ‘undone’ with information counter to the induction information. Subjects would then be randomly allocated to either a Full Interview condition or the Self Administered condition as in the current study- this is still considered important as a comparison of the effects of each of the conditions is of interest. Subjects in the Full Interview condition would then be administered the same interview process as was done in the current study; as would subjects in the Self Administered condition.

Rather than have the interviewers tell subjects of their Short IRI scores, in order to more adequately test for an interviewer bias, the interview would finish with the final questionnaire item being asked. Subjects would then be interviewed by the researcher (not the student interviewer) and assessed on issues such as whether their sense of risk to cancer or CHD and levels of anxiety changed during the course or as a result of the interview; and if they got the sense that the interviewer saw them as being at risk for cancer and CHD. This would be a direct test of the interviewer bias hypothesis. If subject’s perceptions of risk and anxiety changed as a result of the interview process, this would strongly suggest that the process itself produced this change. This conclusion would be strengthened where the subject also stated that they obtained the impression that the interviewer saw them as being at risk. Obviously, this approach would be labour intensive and therefore expensive., but it would go further in explaining how Grossarth-Maticek arrived at his remarkable results.
An additional feature of such a methodology, in order to minimize the level of health risk to subjects, would be to offer them the same level of psychological intervention which Grossarth-Maticzek offered his probands in the intervention study, i.e. a cognitive behavioural therapy along the lines of his Creative Novation Behaviour Therapy (Grossarth-Maticzek & Eysenck 1991). From their reports, it appears that this model of therapy ‘inoculated’ his probands against the deleterious effects of their cognitive and behavioural style. It is just as plausible that this therapy inoculated his probands against the deleterious effects of the interview process and bias itself. Regardless of what the therapy was protecting his probands from, there would seem to be a good ethical case for including a similar intervention in a superior methodology, so as to minimize the health risks to subjects.

Another possible approach would be a naturalistic methodology with a population who is experiencing the problems of interest (cancer or CHD), so that the researcher runs no risk of creating these problems. However, the further the researcher goes down this path, the further s/he moves away from the question of interest and begins to answer other, less pertinent questions. Also, studies such as this would be quasi retrospective in addition to being quasi prospective, and issues of recall bias become relevant limitations.

Another possible study would be to psychologically screen a population at risk of chronic disease in a prospective approach. Such a population may be probands who are more vulnerable to an HIV diagnosis than the general population by virtue of their sexual or drug use behaviours, e.g. gay men or IV drug users. This type of study would take us away from a direct attempt at solving the Grossarth-Maticzek ‘puzzle’, but it would address the underlying phenomenon of interest. Some people who receive an HIV diagnosis remain healthy and never actually develop fully blown AIDS; whereas other people are seen to become very unwell very quickly and often die within a short time from the initial diagnosis. Is it possible that psychological differences are at play here in terms of personality, health beliefs, expectations, and the messages communicated by health professionals? Are certain personality types more resilient on receipt of information about a life threatening condition? What role do basic beliefs about HIV/AIDS shared by the community and medical professionals play, and how do these feed into expectations of survival or demise for the patient? What role does
the health professional play, in terms of styles and content of threat communication, in regards to the patient’s expectations. If a role is established, how do these psycho-social factors interact with physical factors- in a multiplicative or additive manner? This type of research could address the broader question of how belief may play a role in determining biology.

In regards to Study 2 of the current research, it would appear that the prospect of obtaining cancer/CHD by virtue of family history is not quite ‘real’ enough to disturb subjects beyond what Frost et al (2000) has found by way of a short term effect. It would perhaps be better to sample a population of people for whom the perception of risk is more ‘real’ and current. Cigarette smokers would be a likely population to sample. They are subjected to repeated health warnings and messages that smoking is a major risk factor for both cancer and CHD. This population may be more comparable to Grossarth-Maticek’s subjects who potentially came to view themselves as at risk of cancer and CHD by virtue of a perceived Short IRI risk status. It would then be possible to determine if their cancer/CHD risk perception and anxiety does in fact maintain itself over time; or if it maintains itself only for the most psychologically vulnerable subjects (ie. high Neuroticism, Type 1 & 2 scorers).

Ultimately, the best test of the current hypothesis would be to conduct a long term prospective study, replicating Grossarth-Maticek’s methodology. It can be argued that Amelang did not conduct a true replication study as he relied on a different method of data collection to Grossarth-Maticek (and the current author contends that the method of data collection is an essential issue for different reasons that Eysenck 1991). A full and thorough replication study would be expensive and time consuming. It would also not solve the ethical dilemma- how can researchers administer a process to subjects which they suspect may either accelerate or create life threatening conditions such as cancer and CHD? The only answer to this is to create methodologies which either minimize or eliminate this risk by indirectly testing the hypothesis, such as those already suggested. The other partial answer is to bring results from different research projects together so that inferences may be made for the question of interest. This is somewhat inadequate and will never provide more than possibilities. However, due to the nature of the topic area and the ethical constraints which most researchers are
likely to apply to themselves, not to mention the constraints imposed by ethics committees, this may ultimately be all that is possible.

The issue of psychosocial factors in the causation of chronic disease remains an important and controversial one. Answers to the questions posed in the current study have large implications for medical and health care practice, not just in terms of the role which psychology may play. Broader implications include the information, and style of information delivery which health professionals may give to people about their prospects of contracting chronic disease. Is it possible that health professionals cast modern day scientific ‘hexes’ on people when they inform them of risk factors? (Many indigenous cultures would not find this prospect so strange). If so, what is the extent of the effect of such a hex, and how can this be weighed up against the possible benefit of being informed? Can such a ‘hex’ help to create a self fulfilling prophecy and form part of the multi-causal chain of factors in the production of cancer and CHD? Can a psychological study actually result in the premature deaths of many of its participants, just by the mere power of suggestion? And alternately, can health enhancing effects be produced by the same process, by suggesting a prognosis of positive health? Unfortunately, the current study has failed to shed more light onto these questions, however it has raised a question which appears to have not been asked by other researchers in regards to making sense of Grossarth-Maticek’s results. Perhaps more answers can be provided by subsequent study which may follow this one.

The introductory chapters of this paper have detailed claims by a range of researchers which, taken together could allow one to conclude that there is at least moderate evidence that psycho-social factors do play a role in the causation of cancer and CHD. The suggested strength of that role varies according to the claims, with some researches suggesting that there is very little role (Amelang et al 2003) while others (Grossarth-Maticek & Eysenck 1990) suggest there is a very large role. While there does appear to be incontrovertible evidence that psychosocial factors play an important role in the multi-causal equation for general health issues (Cooper 2006), and also convincing evidence of a role for psychology in the causation of CHD (see Bunker et al-2003), the role for psychology in the causation of cancer is far less clear and remains controversial. At this stage, any conclusion of psycho-social factors
playing a large role in the causation of cancer, or indeed the major causal role may still be considered ‘the faith of an agnostic’, as suggested by Binik (1991).

Psychology may remain conflicted on a large role for psycho-social factors in cancer for some time to come. Within many psychologists, the ever optimistic practitioner wants to believe that such a role will ultimately be found, as the prospects for prevention and treatment that arise from this possibility are profound. Despite this fervent wish, the ever skeptical scientist within many psychologists is still yet to be convinced. Psychology clearly plays an important role in clarifying this issue with well constructed research which will inform the ongoing debate.
Appendix I: Letter of Introduction

Dear Sir/Madam,

I am writing to ask for your help in a medical research project. We are studying the relationship between a range of life experiences and behaviours and illhealth. Our aim is to improve the understanding of the causes of illnesses so that it may be possible to prevent some of them from happening in the future. To do this we need to get information from a range of people.

Would you be willing to help us by completing a questionnaire about yourself? This may take around 30 minutes, depending on how long it takes you to answer each question. I will attend your group venue to administer this test to a group of participants.

The questionnaire asks you about such things as experiences that you have had in your life and how you have responded to those situations; how you think and typically react in certain situations.

I would be most grateful if you could find the time to complete the questionnaire. The information that you provide will be kept strictly confidential and used only in the preparation of statistical reports in which you will not be identified.

The questionnaire will entail you volunteering your name and contact phone number to allow me to follow up participants and invite people to attend a second assessment interview in my office at the university. Again, your participation would be entirely voluntary. The second assessment interview will be more in depth and will entail around 1 1/2 hours of your time. In addition, I will be using some bio-feedback equipment which will measure your heart rate. This will be a completely painless procedure.

If you have any questions, we would like you to ask us. Either myself, or my research supervisor, Dr Paul Gannon (Psychology Lecturer, Southern Cross University 02- 66203761) will be happy to answer them.

Thank you in anticipation of your help.

Yours Sincerely,

James Alexander: Registered Psychologist
PhD candidate- School of Human Services
Appendix 2: Informed Consent Form (for Interview 1)

Consent to Participate in Research Study Form: (EPQ)
(Ethics clearance number XXXX)

We are currently conducting research with people to measure what aspects of life may be related to health and illness.

This questionnaire (the Eysenck Personality Questionnaire) is for the purpose of conducting scientific research into the question of whether peoples family history of illness, and their experiences, actions, attitudes and feelings may have some relationship with health and illness.

Your participation in this study is completely voluntary- you may choose to withdraw at any time.

It should take around 30 minutes to complete.

If you choose to participate in answering these questions, your answers will remain totally confidential and you will be helping in a scientific study.

All information you may provide is entirely confidential, and will not be used for any purpose other than this study. Your name will not be entered on any computers. For the purposes of data analysis each participant will be allocated a code number which will in no way be cross-referenced with any details of the participants. The data gathered will be destroyed after it has been analysed.

You may be invited to participate in a second assessment interview at a later date. This will entail answering some more questions while having physiological measures taken, such temperature, heart rate, blood volume pulse and skin conductance. If you are willing to participate in this second assessment interview, would you please state your name and phone number below.

Name: ___________________________
phone number: _______________________

The only time that your name will be used in relation to the study is when I follow up people who have provided me with their names and phone numbers if they are willing to participate in a second assessment interview.

If you have any questions, we would like you to ask us. If you have any additional questions later, Dr Paul Gannon (Psychology Lecturer, Southern Cross University 02- 66203761) will be happy to answer them.

I have read the information above and agree to participate in this study. I am over 18 years of age.

Your Signature _______________________________ (date) __/___/1998.
Appendix 3: Informed Consent Form (for interview 2)

Consent to Participate in Research Study Form:
(Ethics clearance number XXXX)

This questionnaire (the Short Interpersonal Reactions Inventory) is for the purpose of conducting scientific research into the question of whether peoples experiences, actions, attitudes and feelings may bear some relationship with health and illness.

Your participation in this study is completely voluntary- you may choose to withdraw at any time.

It should take around 90 minutes to complete, as we will spend time discussing various aspects of the questionnaire and of your life experiences before the questionnaire items are asked.

If you choose to participate in answering these questions, your answers will remain totally confidential and you will be helping in a scientific study.

All information you may provide is entirely confidential, and will not be used for any purpose other than this study. Your name will not be entered on any computers. For the purposes of data analysis each participant will be allocated a code number which will in no way be cross-referenced with any details of the participants. The data gathered will be destroyed after it has been analysed.

If you have any questions, we would like you to ask us. If you have any additional questions later, Dr Paul Gannon (Psychology Lecturer, Southern Cross University 02- 66203761) will be happy to answer them.

I have read the information above and agree to participate in this study. I am over 18 years of age.

Your name_________________________________

Your Signature ______________________(date)__/___/1998.

Thank you.

James Alexander: Registered Psychologist.
(No. PS0030544 NSW Psychologists Reg. Board)
Appendix 4: The Health Psychology Questionnaire.

Southern Cross University Health Research Questionnaire:

Thank you for agreeing to participate in this study. Please answer the following health related questions as accurately as possible.

1. Your gender: (place an * next to your answer)   Male
   Female

2. Your age ____

3. Have you ever had a major operation or minor surgery? (eg. the removal of a mole or cyst)
   Yes (place an * next to your answer)
   No

4. Have you ever been diagnosed with diabetes? Yes (place an * next to your answer)
   No

5. Have you ever been diagnosed with a cancer? Yes (place an * next to your answer)
   No
   5a. If 'Yes', what type of cancer? ___________
   5b. How many years ago did you receive this diagnosis? ______

6. Have you ever been diagnosed with a coronary heart diseases? (that is any of the following conditions:- angina pectoris, heart attack, weak heart,'heart asthma')
   Yes (place an * next to your answer)
   No

7. As far as you know, has any member of your immediate family (birth mother/father, blood brother/sister, son or daughter) ever been diagnosed with one of the following conditions listed below?
   Yes (place an * next to your answer)
   No
   breast cancer, colorectal (bowel) cancer, melanoma, leukaemia, ovarian cancer, prostate cancer.
   (please highlight or underline the type of cancer)
   7a. If 'yes' for question 7, we need to know how many of your immediate family members (birth mother/father, blood brother/sister, son or daughter) have had one of these diagnoses. Please write in the number here _____
7b. If 'yes' for question 7, think of the first time when you learnt that an immediate family member (birth mother/father, blood brother/sister, biological son or daughter) was diagnosed with one of these conditions. How long ago did you learn of these conditions or understand what it meant?
   Indicate the number of years here. ______

8. As far as you know, has any member of your immediate family (birth mother/father, blood brother/sister, biological son or daughter) ever been diagnosed with one of the following heart conditions listed below?
   Yes (place an * next to your answer)
   No

   angina pectoris, heart attack, weak heart ('heart asthma')

   (please highlight or underline the type of above condition if you know)

8a. If 'yes' for question 8, we need to know how many of your immediate family members (birth mother/father, blood brother/sister, son or daughter) have had one of these diagnoses.
   Please write in the number here _____

8b. If 'yes' for question 8, think of the first time when you learnt that an immediate family member (birth mother/father, blood brother/sister, son or daughter) was diagnosed with one of these conditions. How long ago did you learn of these conditions or understand what it meant?
   Indicate the number of years here. ______

9. As far as you know, has any member of your immediate family (birth mother/father, blood brother/sister, son or daughter) ever been diagnosed with diabetes?
   Yes (place an * next to your answer)
   No

9a. If 'yes' for question 9, we need to know how many of your immediate family members (birth mother/father, blood brother/sister, son or daughter) have had this diagnosis.
   Please write in the number here _____

9b. If 'yes' for question 9, think of the first time when you learnt that an immediate family member (birth mother/father, blood brother/sister, son or daughter) was diagnosed with this condition. How long ago did you learn of this condition or understand what it meant?
   Indicate the number of years here. ______
10i) How likely do you think you are to experience a condition with cancer in the course of your life? (place an * on the dotted line)

0......1......2......3......4......5......6......7......8......9......10
definitely will not 50-50 definitely will

ii) How anxious or worried are you about the possibility of obtaining a condition with cancer over the course of your life? (place an * on the dotted line)

0......1......2......3......4......5......6......7......8......9......10
not at all worried moderate worry very worried

11 i) How likely do you think you are to experience a coronary heart disease in the course of your life?

0......1......2......3......4......5......6......7......8......9......10
definitely will not 50-50 definitely will

ii) How anxious or worried are you about the possibility of obtaining a coronary heart disease over the course of your life?

0......1......2......3......4......5......6......7......8......9......10
not at all worried moderate worry very worried

12 i) How likely do you think you are to experience a blood born disease (eg. HIV, Hepatitis C) disease in the course of your life?

0......1......2......3......4......5......6......7......8......9......10
definitely will not 50-50 definitely will

ii) How anxious or worried are you about the possibility of obtaining a blood born disease (eg. HIV, Hepatitis C) over the course of your life?

0......1......2......3......4......5......6......7......8......9......10
not at all worried moderate worry very worried
Eysenck Personality Questionnaire:

Please answer each question by eliminating the Yes or No which does not apply to you following each question. There are no right or wrong answers, and no trick questions. Work quickly and do not try to think too long about the exact meaning of the question.

eg. I often feel rather stressed

1. Does your mood often go up and down? Yes/No
2. Are you a talkative person? Yes/No
3. If you say you will do something, do you always keep your promise no matter how inconvenient it might be? Yes/No
4. Do you ever feel 'just miserable' for no reason? Yes/No
5. Are you rather lively? Yes/No
6. Were you ever greedy by helping yourself to more than your fair share of anything? Yes/No
7. Are you an irritable person? Yes/No
8. Do you enjoy meeting new people? Yes/No
9. Have you ever blamed someone for doing something you knew was really your fault? Yes/No
10. Are your feelings easily hurt? Yes/No
11. Can you usually let yourself go and enjoy yourself at a lively party? Yes/No
12. Are all your habits good and desirable ones? Yes/No
13. Do you often feel 'fed-up'? Yes/No
14. Do you usually take the initiative in making new friends? Yes/No
15. Have you ever taken anything (even a pin or button) that belonged to someone else? Yes/No
16. Would you call yourself a nervous person? Yes/No
17. Can you easily get some life into a rather dull party? Yes/No
18. Have you ever broken or lost something belonging to someone else? Yes/No
19. Are you a worrier? Yes/No
20. Do you tend to keep in the background on social occasions? Yes/No
21. Have you ever said anything bad or nasty about anyone? Yes/No
22. Would you call yourself tense or 'highly strung'? Yes/No
23. Do you like mixing with people? Yes/No
24. As a child, were you ever cheeky to your parents? Yes/No
25. Do you worry too long after an embarrassing experience? Yes/No
26. Do you like plenty of bustle and excitement around you? Yes/No
27. Have you ever cheated in a game? Yes/No
28. Do you suffer from nerves? Yes/No
29. Have you ever taken advantage of someone? Yes/No
30. Are you mostly quiet with other people? Yes/No
31. Do you often feel lonely? Yes/No
32. Do other people think of you as being very lively? Yes/No
33. Do you always practice what you preach? Yes/No
34. Are you often troubled about feelings of guilt? Yes/No
35. Do you sometimes put off until tomorrow what you ought to do today?  
36. Can you get a party going?

**Miller Behavioural Style Scale:**

Please read each of the following scenarios and try to vividly imagine yourself in those situations. Place an * on each of the _____ lines which you think would apply to you.

1. Vividly imagine that you are afraid of the dentist and have to get some dental work done. Which of the following would you do? Check all of the statements that might apply to you with an *.

   _____ I would ask the dentist exactly what work was going to be done
   _____ I would take a tranquilliser or have a drink before going
   _____ I would try to think about pleasant memories
   _____ I would want the dentist to tell me when I would feel pain
   _____ I would try to sleep
   _____ I would watch the dentist’s movements and listen for the sound of the drill
   _____ I would watch the flow of water from my mouth to see if it contained blood
   _____ I would do mental puzzles in my mind.

2. Vividly imagine that you are being held hostage by a group of armed terrorists in a public building. Which of the following would you do? Check all of the statements that might apply to you with an *.

   _____ I would sit by myself and have as many fantasies and day dreams as I could
   _____ I would stay alert and try to keep myself from falling asleep
   _____ I would exchange life stories with the other hostages
   _____ If there was a radio present, I would stay near it and listen to the bulletins about what the police were doing
   _____ I would watch every movement of my captors and keep an eye on their weapons
   _____ I would try to sleep as much as possible
_____ I would think about how nice it’s going to be when I get home

_____ I would make sure I knew where every possible exit was.

3. Vividly imagine that, due to a large drop in sales, it is rumoured that several people in your work department will be layed off. Your supervisor has turned in an evaluation of your work for the past year. The decision about lay-offs has been made and will be announced in several days. Which of the following would you do? Check all of the statements that might apply to you with an *

_____ I  would talk to my fellow workers to see if they knew anything about what the supervisor’s evaluation of me said

_____ I would review the list of duties for my present job and try to figure out if I had fulfilled them all

_____ I would go to the movies to take my mind off things

_____ I would try to remember any arguments or disagreements I might have had that would have resulted in the supervisor having a lower opinion of me.

_____ I would push all thoughts of being layed off out of my mind

_____ I would tell my spouse that I’d rather not discuss my chances of being laid off

_____ I would try to think which employees in my department the supervisor might have thought had done the worst job

_____ I would continue doing my work as if nothing was happening.

4. Vividly imagine that you are on an aeroplane, thirty minutes from your destination, when the plane unexpectedly goes into a deep dive and then suddenly levels off. After a short time, the pilot announces that nothing is wrong, although the rest of the ride may be rough. You, however, are not convinced that all is well. Which of the following would you do? Check all of the statements that might apply to you with an *

_____ I would carefully read the information provided about safety features in the plane and make sure I knew where the emergency exits were

_____ I would make small talk with the passenger beside me

_____ I would watch the end of the movie, even if I had seen it before
_____ I would call for the flight attendant and ask what exactly the problem was

_____ I would order a drink from the flight attendant or take a tranquilliser

_____ I would listen carefully to the engines for unusual noises and would watch the crew to see if their behaviour was out of the ordinary

_____ I would talk to the passenger beside me about what might be wrong

_____ I would settle down and read a book or magazine or write a letter.

Please continue to the next (final) page

Thank you for filling in this questionnaire- your assistance is much appreciated and may help us to reach a more scientific understanding of some the causes of certain illnesses.

Would you be willing to participate in an interview if called to do so in order that we can collect more information? (eliminate the answer which does not apply to you)

YES / NO

If YES, please add you name and phone number here so that I can arrange a time that may suit.

Your name.................................. Your phone number.............................

It may take me 2-3 months before I can contact you in regards to the follow up interview.

Thanks again,

James Alexander
Registered Psychologist.
PhD candidate- School of Human Services
Lismore Campus
Appendix 5 - The Short Interpersonal Reactions Inventory (Short IRI)

SHORT INTERPERSONAL REACTIONS INVENTORY
by R.Grossarth-Maticek, Ph.D & H.Eysenck, Ph.D, D.Sci.

Here are a number of questions covering your attitudes and emotions in relation to people and situations which have great significance for you. Try to answer 'Yes' or 'No' to each question according to your first impression; do not think too long about the precise meaning of the question. Leave the answer blank only if it is quite impossible for you to answer 'Yes' or 'No'. Eliminate the answer which does not apply to you, leaving the answer which does apply,

eg. I am able to get some life into a dull party

1. I find it very difficult to stand up for myself. YES NO
2. I have been complaining for years about various unfavourable conditions but I am not able to change them. YES NO
3. I am mainly concerned with my own well-being. YES NO
4. I am usually content and happy with my daily activities. YES NO
5. I cannot live happily and contentedly with or without a particular person. YES NO
6. I prefer to agree with others rather than assert my own views. YES NO
7. Certain people are the most important causes of my personal misfortunes. YES NO
8. I alternate to a great degree between the positive and negative evaluation of people and conditions. YES NO
9. When I cannot achieve closeness with someone who is emotionally important to me, I have no difficulties in letting them go. YES NO
10. I cannot live happily and contentedly in the absence of certain states or conditions, eg. I need my work but I am unhappy doing it. YES NO
11. I tend to act more to fulfil the expectations of people close to me rather than look after my own needs. YES NO
12. Certain conditions or situations are the most important cause of my personal misfortunes. YES NO
13. With people I love, I keep changing from keeping them at a great distance to stifling dependence, and from stifling dependence to excessive distancing. YES NO
14. I can usually arrange things so that people who are emotionally important to me are as close or as distant from me as I wish. YES NO
15. I often have thoughts which terrify me and make me unhappy. YES NO
16. I tend to give in and abandon my own aims in order to achieve harmony with other people. YES NO
17. I feel helpless against people or conditions which cause great unhappiness for me, because I cannot change them. YES NO
18. When I am in a situation which I experience as threatening, I immediately try to get other people to help and support me. YES NO
19. When I fail to achieve my objectives, I can easily change tack. YES NO
20. Relations with certain people are always pretty unsatisfactory but there is nothing I can do about it. YES NO
21. I am unable to express my feelings and needs openly to
22. I always seem to be confronted with the undesirable aspects of people and conditions. YES NO
23. When someone who is emotionally important to me hurts me, even slightly, I immediately dissociate myself from that person YES NO
24. I can manage to live fairly contentedly with or without someone who is emotionally important to me YES NO
25. Certain situations and states (e.g. at my place of work) tend to make me unhappy, but there is nothing I can do to alter things. YES NO
26. I tend to accept conditions which work against my personal interests without being able to protest. YES NO
27. Certain people keep interfering with my personal development. YES NO
28. I expect others to live up to the highest moral standards but do not feel that these are binding on myself. YES NO
29. I can usually change my behaviour to suit conditions. YES NO
30. Certain bodily conditions (e.g. being overweight) make me unhappy, but I feel unable to do anything about them. YES NO
31. I often feel inhibited when it comes to openly showing negative feelings such as hatred, aggression, or anger. YES NO
32. Certain conditions keep interfering with my personal development. YES NO
33. I seek satisfaction of my own needs and desires first, regardless of the rights and needs of others. YES NO
34. I am usually capable of finding new points of view and successful, sometimes surprising solutions for problems. YES NO
35. I can relax bodily and mentally only very rarely; most of the time I am very tense. YES NO
36. I am not inclined to be demonstrative when emotional shocks upset me. YES NO
37. I cannot control excitement or stress in my life because this is dependent on the actions of other people. YES NO
38. When I make emotional demands on another person, I require immediate satisfaction. YES NO
39. I am independent in what I do, and do not depend on other people if it this would be to my disadvantage. YES NO
40. I have great difficulties in entering into happy and contented relations with other people. YES NO
41. When I feel emotionally let down, I tend to be paralysed and inhibited. YES NO
42. I cannot control excitement or stress in my life because this depends on conditions over which I have no control. YES NO
43. I usually find fulfilment in everyday situations which are not subject to ordinary rules, regulations, and expectations. YES NO
44. When things don't work out, this does not make me give up but rather makes me change my way of doing things. YES NO
45. I am helpless when confronted with emotional shocks, depression or anxiety. YES NO
46. When something terrible happens to me, such as the death of a loved one, I am quite unable to express my emotions and desires. YES NO
47. I can express my aims and desires clearly but feel that it is quite impossible to achieve them. YES NO
48. As soon as someone becomes emotionally important to me
I tend to place contradictory demands on them, such as "Don't ever leave me" or "Get away from me".

49. When things lead to harmful results for me, I have no trouble in changing my behaviour to make for success.

50. I am seldom able to feel enthusiasm for anything.
Appendix 6 - Additional Statistical Tables:

Table 59 shows positive correlations for Full Interview subjects between Short IRI Type 1, Type 2 and Type 3 scores (p<.01); a significant negative correlation between Type 1 and Type 4 scores (p<.05). It also shows significant negative correlations between Type 2 and Type 4 scores, as well as between Type 3 and Type 4 scores (all p<.01).

Table 59. Short IRI scale correlations for the Full Interview condition subjects (n=65).

<table>
<thead>
<tr>
<th></th>
<th>Type 1</th>
<th>Type 2</th>
<th>Type 3</th>
<th>Type 4</th>
</tr>
</thead>
<tbody>
<tr>
<td>Type 1</td>
<td>Correlation</td>
<td>1</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Sig. (2-tailed)</td>
<td>.</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Type 2</td>
<td>Correlation</td>
<td>.470(**)</td>
<td>1</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Sig. (2-tailed)</td>
<td>.000</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Type 3</td>
<td>Correlation</td>
<td>.403(**)</td>
<td>.579(**)</td>
<td>1</td>
</tr>
<tr>
<td></td>
<td>Sig. (2-tailed)</td>
<td>.001</td>
<td>.000</td>
<td></td>
</tr>
<tr>
<td>Type 4</td>
<td>Correlation</td>
<td>-.270(*)</td>
<td>-.371(**)</td>
<td>-.453(**)</td>
</tr>
<tr>
<td></td>
<td>Sig. (2-tailed)</td>
<td>.029</td>
<td>.002</td>
<td>.000</td>
</tr>
</tbody>
</table>

** Correlation is significant at the 0.01 level (2-tailed).
* Correlation is significant at the 0.05 level (2-tailed).

In Table 60, correlation coefficients are shown for Short IRI scores and the personality variables used in this study for Full Interview subjects. Type 1 scores significantly negatively correlate with Extroversion (p<.05), meaning that Type 1 scores correlate significantly with Introversion in a positive direction. There is a significantly positive correlation between Type 1 scores and Neuroticism (p<.01). Type 2 scores show a negative but non significant correlation with Extroversion scores, and a significant positive correlation with Neuroticism (p<.01). Type 3 scores show a significant positive correlation with Neuroticism scores (p<.01). Type 4 scores show a positive but non significant correlation with Extroversion, and a significant negative correlation with Neuroticism (p<.01). Extroversion shows a significant negative correlation with Type 1 scores.
scores p<.05) and a non significant negative correlation with Neuroticism, meaning that Introversion is positively correlated with Type1. Extroversion also shows non significant negative correlations with Type 2 and Type 3 scores, and a non significant positive correlation with Type 4 scores. Neuroticism is positively correlated with Type 1 scores, Type 2 scores and Type 3 scores (all: p<.01); and negatively correlated with Type 4 scores (p<.01). There is also a non significant negative relationship between Neuroticism and Extroversion. Finally, Monitor and Blunter scores show no significant correlations with any of the Short IRI scores.

Table 60. Correlations of personality variables and Short IRI Type scores for Full Interview condition subjects (n=65).

<table>
<thead>
<tr>
<th></th>
<th>Extroversion</th>
<th>Neuroticism</th>
<th>Monitor score</th>
<th>Blunter score</th>
</tr>
</thead>
<tbody>
<tr>
<td>Type 1</td>
<td>Correlation</td>
<td>-.319(**)</td>
<td>.392(**)</td>
<td>-.052</td>
</tr>
<tr>
<td></td>
<td>Sig. (2 tailed)</td>
<td>.010</td>
<td>.001</td>
<td>.682</td>
</tr>
<tr>
<td>Type 2</td>
<td>Correlation</td>
<td>-.200</td>
<td>.582(**)</td>
<td>.193</td>
</tr>
<tr>
<td></td>
<td>Sig. (2 tailed)</td>
<td>.110</td>
<td>.000</td>
<td>.124</td>
</tr>
<tr>
<td>Type 3</td>
<td>Correlation</td>
<td>-.131</td>
<td>.595(**)</td>
<td>.157</td>
</tr>
<tr>
<td></td>
<td>Sig. (2 tailed)</td>
<td>.298</td>
<td>.000</td>
<td>.212</td>
</tr>
<tr>
<td>Type 4</td>
<td>Correlation</td>
<td>.196</td>
<td>-.434(**)</td>
<td>-.162</td>
</tr>
<tr>
<td></td>
<td>Sig. (2 tailed)</td>
<td>.117</td>
<td>.000</td>
<td>.196</td>
</tr>
</tbody>
</table>

** Correlation is significant at the 0.01 level (2-tailed).
* Correlation is significant at the 0.05 level (2-tailed)

Table 61 shows the correlations between the Short IRI Types and the Short IRI scores and cancer history and CHD history in the family for the Full Interview sample. There are no additional significant correlations between the variables reported.
Table 61. Correlations between Short IRI Types and cancer/CHD in family history for Full Interview condition subjects (n=65).

<table>
<thead>
<tr>
<th>Type</th>
<th>Correlation</th>
<th>Cancer history</th>
<th>CHD history</th>
</tr>
</thead>
<tbody>
<tr>
<td>Type 1</td>
<td>-.054</td>
<td>.672</td>
<td>.111</td>
</tr>
<tr>
<td></td>
<td>-.199</td>
<td>.111</td>
<td></td>
</tr>
<tr>
<td>Type 2</td>
<td>.072</td>
<td>.571</td>
<td>.590</td>
</tr>
<tr>
<td></td>
<td>-.068</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Type 3</td>
<td>-.029</td>
<td>.817</td>
<td>.300</td>
</tr>
<tr>
<td></td>
<td>-.131</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Type 4</td>
<td>.128</td>
<td>.308</td>
<td>.368</td>
</tr>
<tr>
<td></td>
<td>.114</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

** Correlation is significant at the 0.01 level (2-tailed).
* Correlation is significant at the 0.05 level (2-tailed).

Table 62 shows the correlations between Short IRI scores and the self report measures used in this study for the Full Interview subjects.

Table 62. Correlations between Short IRI Types and self report measures of cancer/CHD risk perception and anxiety for Full Interview condition subjects (n=65)

<table>
<thead>
<tr>
<th>Type</th>
<th>Cancer risk perception</th>
<th>Anxious about cancer</th>
<th>CHD risk perception</th>
<th>Anxious about CHD</th>
</tr>
</thead>
<tbody>
<tr>
<td>Type 1</td>
<td>.233</td>
<td>-.076</td>
<td>.069</td>
<td>.060</td>
</tr>
<tr>
<td></td>
<td>.062</td>
<td>.545</td>
<td>.584</td>
<td>.634</td>
</tr>
<tr>
<td>Type 2</td>
<td>.261(*)</td>
<td>-.177</td>
<td>.072</td>
<td>.012</td>
</tr>
<tr>
<td></td>
<td>.036</td>
<td>.160</td>
<td>.570</td>
<td>.927</td>
</tr>
<tr>
<td>Type 3</td>
<td>.376(**)</td>
<td>-.169</td>
<td>.111</td>
<td>-.024</td>
</tr>
<tr>
<td></td>
<td>.002</td>
<td>.177</td>
<td>.380</td>
<td>.847</td>
</tr>
<tr>
<td>Type 4</td>
<td>-.181</td>
<td>.238</td>
<td>.090</td>
<td>.083</td>
</tr>
<tr>
<td></td>
<td>.150</td>
<td>.056</td>
<td>.473</td>
<td>.511</td>
</tr>
</tbody>
</table>

** Correlation is significant at the 0.01 level (2-tailed).
* Correlation is significant at the 0.05 level (2-tailed).
Table 63 shows correlations between self report variables and personality variables for the Full Interview condition subjects. As the entire table is too large to display on an A4 format, Table 63 shows only the correlations which demonstrated a level of significance. There is a significant positive correlation between Anxious about CHD and Anxious about Cancer (p<.01); a positive correlation between Likely to get CHD and Anxious about CHD ((p<.01); and a negative correlation between Likely to get CHD and Monitor score (p<.05).

Table 63. Correlations between the potential predictor variables for the Full Interview condition subjects (n=65)

<table>
<thead>
<tr>
<th>Likely to get CHD</th>
<th>Anxious about cancer</th>
<th>Anxious about CHD</th>
<th>Likely to get CHD</th>
<th>Monitor</th>
</tr>
</thead>
<tbody>
<tr>
<td>Correlation</td>
<td>.228</td>
<td>.542(**)</td>
<td>1</td>
<td></td>
</tr>
<tr>
<td>Sig. (2 tailed)</td>
<td>.068</td>
<td>.000</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Monitor</td>
<td>Correlation</td>
<td>-.027</td>
<td>-.137</td>
<td>-.249(*)</td>
</tr>
<tr>
<td>Sig. (2 tailed)</td>
<td>.831</td>
<td>.277</td>
<td>.046</td>
<td></td>
</tr>
</tbody>
</table>

** Correlation is significant at the 0.01 level (2-tailed).
* Correlation is significant at the 0.05 level (2-tailed).

Table 64 displays the correlations between cancer and CHD in family history and Short IRI scores for the Self Administered sample. There are no additional correlations to report.
Table 64. Correlations between Short IRI Types and cancer and CHD in family for Self Admin. Subjects (n=40)

<table>
<thead>
<tr>
<th>Type</th>
<th>Correlation</th>
<th>Cancer history</th>
<th>CHD history</th>
</tr>
</thead>
<tbody>
<tr>
<td>Type 1</td>
<td>-.135</td>
<td>-.161</td>
<td></td>
</tr>
<tr>
<td></td>
<td>.408</td>
<td>.327</td>
<td></td>
</tr>
<tr>
<td>Type 2</td>
<td>.006</td>
<td>.067</td>
<td></td>
</tr>
<tr>
<td></td>
<td>.970</td>
<td>.686</td>
<td></td>
</tr>
<tr>
<td>Type 3</td>
<td>-.207</td>
<td>.052</td>
<td></td>
</tr>
<tr>
<td></td>
<td>.200</td>
<td>.752</td>
<td></td>
</tr>
<tr>
<td>Type 4</td>
<td>.205</td>
<td>.290</td>
<td></td>
</tr>
<tr>
<td></td>
<td>.204</td>
<td>.073</td>
<td></td>
</tr>
</tbody>
</table>

** Correlation is significant at the 0.01 level (2-tailed).
*Correlation is significant at the 0.05 level (2-tailed).
Figure 7. Scatterplot showing outlier in Changes in Mean Heart Rate:
Appendix 7: Transcript of interviewer statements recorded during a standard interview:

Thanks very much for taking the time out to come along here today- I really appreciate it. The first thing I need to do is to ask you to attach this devise- it’s a heart rate monitor- what you put on is the transmitter, and this device here is the receiver which picks up the frequency from the transmitter and gives a continuous reading (demonstrate how to attach the transmitter).

Confidentiality:
OK, the first thing I need to tell you is that each participant in my study has been given a study number- you are number …… (show this to the subject). All of the information which you have given me so far in the questionnaire, and any information you give me today is located only with this study number. I take all of the information and enter it into my computer system, but no where in it is your name entered, only the study number. So, if someone was to steal my computer, all they would find would be a bunch of numbers which wouldn’t mean anything to them (show them to SPSS spread sheet).
This is pretty important for people to know because I suppose the quality of the results I wind up with are to a certain degree dependent on the quality of the information people are willing to give me. This is an unusual situation to find yourself in- you have never met me before and have never been in this office before, and I will be asking you questions about aspects of your personal life and asking that you provide me with as honest answers as possible. So, in order to be willing to provide me with this information, you really need to be able to trust that any information you give me is handled properly. I’ve been working as a psychologist for around 15 years, so I am very well aware of the need to keep people’s personal information confidential- I certainly don’t go blabbing your information to anybody else.

Introduction to the area of study:
The area that we will be looking at today is called Health Psychology- its dealing with the idea that psychological factors may have a role to play in health and ill health. This idea
has been around probably ever since humans have been on the planet. All traditional cultures in the world believe that non physical factors play a large role in physical health. Even in the history of western culture, this idea has been around for a long time. Around 2 ½ thousand years ago, Hippocrates was making statements about certain illnesses seeming to be related to certain personality types. Until around 100 years ago, this idea was viewed as being a fairly self evident fact- the early medical journal literature often referred to psychological factors in ill health. As an example, the man who is referred as being the ‘Father of British medicine, Sir William Osler is quoted as saying “it is more important to know what kind of person has the disease rather than what kind of disease the person has”. Around the turn of the last century, the germ theory of disease shot to prominence as the major explanatory system within western medicine. Pasteur had discovered the germ several decades early, but it was around the turn of the century that germ theory became the major way of explaining ill health. At that point, anything that could not be seen under a microscope or measured in a test tube was pretty well ignored as being unscientific. Some researchers and practitioners stayed interested in psychological factors in ill health, but any professional discussion of this tended to be based on anecdotal observations rather than on empirical, quantitative research.

In the early 1950’s, a British psychologist by the name of Hans Eysenck began approaching this question from an empirical research basis. He was primarily a personality researcher, but he was obviously interested in mind-body health issues, perhaps because Hippocrates had described certain conditions as being related to certain personality types, and Eysenck did a lot of research with developing Hippocrates personality types. So, Eysenck began the fist empirical research into health psychology issues. He teamed up with a Scottish oncologist, and together they studied the personality types of male lung cancer patients- they did find statistically significantly different personality characteristics to male lung cancer patients compared to the rest of the population. Not far into this research program, the oncologist was killed in a car accident and Eysenck had to find another medico who was going to help him with the research program. He couldn’t find anyone to help- young oncologists were warned off by the old guard, saying things like it would be a bad career move to get caught up in this sort of
stuff. So, eventually, Eysenck had to let it go and focused his interest on other aspects of personality research.

In the late 1960’s and early 1970’s, a Yugoslavian psychologist named Ronald Grossarth-Maticek began researching the same question. He obviously had read some of Eysenck’s reports from the 50’s and was also interested in whether there might be any such thing as the health prone, cancer prone or heart disease prone personality types. In the early 70’s in Heidelberg, West Germany, he began what must be one of the largest research studies conducted in psychology. He recruited nearly 20,000 people to participate in what would become a long term study. He had a team of 100 interviewers who amassed large amounts of information from these people- they had all been assessed as being free from any signs of cancer or heart disease, so they were basically all healthy. He got from them information about many of the standard medical risk factors for cancer and heart disease. So, he asked them about such things as cigarette smoking history, alcohol consumption, dietary factors, levels of exercise, family history information, cholesterol levels, etc. In addition to all of this, he developed a 200 item questionnaire that was designed to measure people in terms of whether, according to his theory, they were cancer prone, heart disease prone, or health prone on the basis of their personalities. He followed these people up at the 7, 9, 13, and 15 year marks to find out if they had experienced any of these conditions. What he claims to have found was an 81% accuracy of prediction of the incidence of cancer, heart disease or health based on the psychological factors alone. Further, his statistical analysis suggested that the psychological factors were 6 times more predictive of cancer or heart disease than were any of the physical risk factors either on their own or taken together. Now, this is obviously a very big claim- it lead to some controversy in the literature with many people suggesting that it couldn’t be true. Its even more remarkable when you consider that its around 1 in every 10 heavy cigarette smokers can expect to die of lung cancer- cigarette smoking is one of the best predictors of death by lung cancer and heart disease. So, basically, if you are a heavy smoker, you can say that you have about a 10% chance of dying of lung cancer- its about a 10% accuracy of prediction. Compare this to Grossarth-Maticek’s claim of 81% accuracy of prediction based on psychological factors, and you
can see what a big claim it is. Other researchers in health psychology have demonstrated that psychological factors are at least a moderate predictor of ill health, but Grossarth-Maticzek was saying that it is the most overriding important one.

Now, even though it’s a very impressive claim with a large amount of people, its hard to have a whole lot of confidence in it on its own. There needs to be other researchers who independently come to the same conclusion before you could really say there is clear evidence of what Grossarth-Maticzek was saying. His results may have something to do with unique characteristics of his sample- they were German people in their 40’s, 50’s and 60’s, so they had all had experience in WWII- maybe this had something to do with the findings; or maybe it has to do with his particular methodology. In the absence of independent replication studies, we really don’t know whether to have a lot of confidence in his results. So, at the moment there are a range of independent studies happening in various parts of the world, and mine is one of those. Any questions at this stage?

What I’ll be doing today is administering Grossarth-Maticzek’s questionnaire with you- he has since reduced it to 50 items rather than 200- his statistical analysis suggested that you could get the same measures with a shorter questionnaire. I’ll be working out what your answers mean in terms of whether it suggests you are cancer prone, heart disease prone or health prone- adding all of this to the information which you have already provided me with in the original questionnaire, and see if there are any relationships between it all across my whole sample.

**Prospective study question:**

I’m asking all of my participants if they’d be willing to have me contact them in 2 years time and each 2 years subsequent to that, to find out if there have been any major health issues pop up over that time- would that be ok if I did that, gave you a call in 2 years? It would just be a matter of a 5 minute phone chat. I don’t anticipate that there would be any health issues pop up in 2 years, but if I left it for 20 years I wouldn’t be able to find anybody- its much easier to track people every couple of years. (Obtain their phone numbers, or parent’s phone numbers or email addresses).
Cancer/CHD anxiety question 1:
OK, the next thing I need to do is to ask you some questions that you have already answered on the first questionnaire. (Obtain subject’s answers to the questions).

Positive/Negative life experiences discussion:
Before I administer the questionnaire with you, I’d like to discuss with you some aspects of your life. If you can think about a couple of things:- a positive life experience or event which you’ve had and your response to that situation; or more generally your typical responses to happy situations in your life. And also a negative life event or experience and your response to it; or again, more generally your typical responses to negative events in your life. I wont be taking any notes during this discussion.
(Subject recounts positive and negative experiences)
Prompting questions:-
What did you do at that time, when ……. happened? Was it a private internal type of happiness, or did you share it with other people? How do other people around you know when you are happy, what do they see you doing? Do you tend to share that type of thing with others? Is that a typical response for you?
Can you remember what you did with the upset? Did others around you know how it affected you? How did they know? Was it a private internal kind of upset, or did you let others know what you were going through? Is that typical for you? Do you usually share your upsets in that way?
Have you had any experiences with depression? How do you usually respond to stress? What do you do with it? Do you tend to systematically work out what to do about a stressful situation, or just stay with the upset?
Do you see yourself as being a person who can stand up for themselves, or do you prefer to just let things go? Are you willing to go through conflict in order to get somewhere better, or do you prefer to avoid conflict if possible?

Gauge trust & understanding:
OK, before I begin to administer the questionnaire with you, I’d just like to check out with you if you feel the need for any more information. Like I said before, this is an
unusual situation to find yourself in- you’ve never met me before, and here I am asking you questions about your personal life. I suppose to feel ok about discussing these things with me, its important to feel that you have enough trust that the information you provide will be treated properly, and in this whole situation; and enough understanding in what the research is all about. So, if you feel that there are questions you need to ask in order to have enough trust and understanding of this research, then feel free to ask them and I’ll do the best I can to answer or clarify any questions. OK, so you feel that you have enough trust and understanding to proceed with the questionnaire? If there are any questions that pop up, feel free to ask them.

**Introduction to Short IRI:**

OK, this is called the Short Interpersonal Reactions Inventory- it was devised by Grossarth-Maticek and Eysenck, who met up with him in the mid 80’s.

These are the instructions (read them off the Inventory).

Most of the questions are pretty straight forward; some on the second side get a little bit curly with some potential double negatives in there- it can be a bit hard to work out, ‘well, if I answer yes to this, does it mean yes or no to that’. Some people find it a bit easier to think of these in terms of a bunch of statements which for you are either true or false. So, you can answer either yes or no, or true or false. Also, this was translated from German, so some of the language is a bit odd to us- it reads like a literal translation from German; I could have re-written it into the sort of English we speak, but then I’d be open to criticisms of tampering with the questionnaire, so I thought it was just better to leave it as it is. If you want me to repeat any questions, I can do that; or if you want me to explain what a question is actually asking, then I can do that as well. As it says in the instructions, what we are after here is your first response as soon as you understand what the question is asking- we don’t want the edited answer, which may come a nanno second after your first response.
**Gauge interest in IRI answers:**
(on completion of administering the Short IRI)
OK, so are there any questions that you’d like to ask; anything that you want to know about the research?
(if the subject says s/he would like to know what the results of the IRI mean for them)
Would you like me to work out your score now so I can tell you? OK- it will take me just a few minutes.
(if subject does not inquire about the scores- ‘Would you like to know what your score suggests for you?’)

**Provision of Short IRI score:**
Alright, what this says about you is that you are more prone to good health than to anything else. The questionnaire is made up of 4 different scales, each with 10 items. Scale 1 is the cancer prone type- you scored 2 out of 10 on that, so that means according to Grossarth-Maticek that you aren’t psychologically prone to cancer. On the Type 2 scale, which is proneness to heart disease, you only scored 1 out of 10 on that- this means that according to Grossarth-Maticek, you aren’t psychologically prone to heart disease either. The type 4 scale is the health prone personality type, and you scored 7 out of 10 on that scale, so overall this means that Grossarth-Maticek would conclude from this that you are health prone. I’ll explain all of this in more detail to you in a moment, but right now I need to ask you the questions that I asked you before you answered the questionnaire.

**Cancer/CHD Anxiety questions 2:**
Questions are read from the sheet again, subject’s answers are obtained and recorded.

**Discussion/debriefing/interpretation of results 1:**
Right, now lets have more of a look at what these results mean for you. Firstly, I wouldn’t say from this that you will never experience cancer or heart disease. The fact is, the older people get, the more likely they are to experience either of these conditions- it just seems to be a normal part of the aging process; for example, your immune system
naturally slows down when you get old- this makes people more susceptible to cancer and other conditions. What most people are concerned about are the premature cancers and heart diseases, not so much the type that people get in their 80’s or 90’s. So, even though you scored high on health proneness, I wouldn’t say you will never experience cancer or heart disease. If Grossarth-Maticek is right, then he would say that he could be 81% confident, based on your answers, that you are not likely to experience premature cancer or heart disease, regardless of all the other risk factors. He says that the psychological factor is by far the most important one, and your psychological factor suggests that you are not prone to cancer or heart disease. If he is wrong, and at this stage we don’t really know whether he is right or wrong, but if he is wrong, it probably means that psychological factors are a moderate risk factor, just like all the other moderate risk factors. Cancer and heart disease are multi factorial equations- you have life style factors like smoking and drinking, levels of exercise; you have family hereditary factors; exposure to carcinogens, etc. And you have a psychological contribution to that equation. So, if Grossarth-Maticelk is wrong, it means that you simply don’t have to worry about the psychological contribution to the equation- you still need to work out where you stand with all the other risk factors I mentioned, but you don’t need to worry about the psychological part of that equation. Do you have any questions about this?

Discussion/debriefing/interpretation of results 2:
OK, so what this score suggests about you, according to Grossarth-Maticek is that you are more psychologically prone to cancer than to heart disease or to optimal health. The questionnaire is made up of 4 different scales, each with 10 items in them. Grossarth-Maticek would look at your answers here and say that because you scored 7 on the T1 scale, which is the cancer prone scale, and because this is your highest score, you are psychologically prone to cancer. You can see that your optimal health scale, the T4 scale here, only adds up to 4 out of 10, so the T1 scale is your highest scoring one. So, according to Grossarth-Maticek your personality would make you more vulnerable to cancer.
Cancer/CHD Anxiety questions 2:
Questions are read from the sheet again, subject’s answers are obtained and recorded.

Debriefing a negative score.
Now, there’s a few things we need to keep in mind about Grossarth-Maticek’s research. Firstly, by his own figures, his predictions were wrong 19% of the time, so you may be one of these ‘errors’ of prediction. Secondly, we just don’t know how much confidence to have in his original research. As I said earlier, there are allegations of scientific fraud and other questions about his research, so until independent researchers can replicate his findings, it all has to be viewed as a hypothesis at this stage. Also, some really interesting research was conducted by a German professor of psychology called Amelang. You would think that if someone has cancer, their scores on this questionnaire would show them as being cancer prone; and if someone had heart disease, their scores would show them as being heart disease prone; and if they were healthy, their score would show them as being health prone. Amelang got a sample of people who were healthy, another group of people who were cancer patients and a group of people who were heart disease patients, and administered this questionnaire to them. What he found was that people’s answers on the questionnaire did not indicate which group they belonged to- there was no necessary relationship between having cancer and being assessed as cancer prone; the same thing with heart disease patients and with healthy subjects. So, the questionnaire was not able to differentiate between those who had cancer, heart disease or good health. This suggests that it is not such an accurate predictor of cancer or heart disease. What it did find though was that it could broadly distinguish between two main groups of subjects- those that were healthy and those that were unhealthy, because of either cancer or heart disease. So, the questionnaire answers could distinguish between the broad categories of healthy or unhealthy, but it couldn’t actually distinguish between different types of unhealthy, cancer or heart disease. Also, the biggest predictor for both cancer and heart disease is age. As you are still quite young, this means that you have many years to look at and change the whole range of risk factors such as dietary issues, levels of exercise, smoking, drinking- all that sort of stuff.
You also have plenty of time to modify some of the characteristics that Grossarth-Maticke was talking about.

**Reviewing the Types.**

Now, let's have a look at the personality types (share with subject the prepared description sheets). This is what Grossarth-Maticke calls the cancer prone personality type. Its important here to emphasize that what we are talking about are hypothesized personality types, because in the absence of independent confirmations, all this has to be viewed as a hypothesis- it hasn’t been demonstrated to be correct. So, the hypothesized cancer prone personality traits are these (read off sheet). Now, all of us have these characteristics within us- everyone does these things from time to time. This type is really describing a person who is like this pretty well all the time- their personality can be characterized by these terms. Because you scored high on this type, you can probably relate to some of these characteristics? (discussion)

Read from sheet and discuss the CHD personality type as well as the Optimal Health type.

**Implications for prevention of illness.**

Now, there’s a few things I need to tell you about some other research which Grossarth-Maticke did. As part of his study, he had a sample that were assessed as being cancer or heart disease prone, and he gave them the option of some short term psychological intervention to help them change those aspects of their behaviour and personality. About half of the people accepted the offer and the other half didn’t, so there he had a control group and a treatment group. With those that did accept the offer, he saw them on average around 6 times and provided his own style of cognitive-behavioural therapy. He helped people, for example if they were assessed as being cancer prone, to learn how to recognize strong negative feelings; how to express these to others and realize the world wouldn’t fall apart if they did; how to cope with stress, develop strategies to solve problems; things like that- reasonably basic stuff. When he followed these people up years later, he found that in the control group, those who had no treatment at all, there was a fairly high incidence of cancer and heart disease, as well as other serious illnesses,
and even things like more fatal car accidents. When he followed up the treatment group, he found that very few of them had experienced cancer or heart disease, or other serious illnesses--they were much more healthy. So, it seems that what he managed to do was to help the people who were assessed as being at risk to reverse their risk status--this suggests that you can do things to minimize the effects of the psychological risk factor. If you begin to focus on the aspects of personality which he describes and try to change them, then it seems that there is a good chance that you can actually improve your health status years later. So, for example, you may begin to look at ways of expressing your concerns or upsets to other people; or becoming more assertive; or developing new problem solving strategies. The other thing worth noting is that the greatest predictor of both cancer and heart disease is age--the older you get, the greater chance you have of getting either cancer or heart disease. The at risk age group is generally 60 plus, so you have around 35 or 40 years before you enter that age group, and all that time to change some of these tendencies you've got!

Counselling can be an effective way of addressing some of these behaviours--there is a free counseling service here at the uni, and at places like community health centres. The basic rule of thumb with counseling is that if it makes you feel more helpless and hopeless, then it isn’t working for you and may be of no use for your health; but if you feel less helpless and hopeless because of it, then it is most likely good for you. Self help books can be useful as well--I’ve included a reference here for a really good one by American psychologist Martin Seligman called ‘Learnt Optimism’. It has lots of stress reducing strategies in it.

So, overall, if you are able to address some of the personality characteristics described by Grossarth-Maticek, either by just being aware of them and counteracting them, or by counseling or self help books, then, if Grossarth-Maticek is right, there should be a really good chance for you to reverse this risk factor over time.

If Grossarth-Maticek is wrong, then it probably means that the other researchers in health psychology are right and that psychological factors are only moderate risk factors, in which case personality is only one of a range of factors which may make up health or
cancer or heart disease. This means that you would do well to look at these other factors, for example smoking, drinking, diet, levels of exercise- those life style factors which you can actually do something about. It might also help you in life to look at the personality aspects which Grossarth-Maticek described, but if he is wrong, changing them may not have much effect on your overall health status.

So, how do you feel about all that? Is there anything more you would like to know, and questions to ask?

OK- thanks very much for your time- I really appreciate it, and I’ll be in contact with you in 2 years times.
Appendix 8- Interview format and data recording form:

Interview format: date…./…./2001 HRM file ____

- Subject number ________ check that interval is set to 5 sec.
- Self administered / full interview format.
- Apply heart rate monitor equipment- obtain a reading-begin
- Allow 5-10 minutes before recording is begun- discussion
- Begin recording HRM reading- *begin stop watch*.

Note stopwatch ______
- Ask if the subject is willing to have the interviewer contact him/her in 2 years time and each subsequent 2 years in order to ask questions about health issues.
- If ‘yes’: note contact ph. No........................... other ph. No......................

Note stop watch: ______ Ask cancer/CHD anxiety questions

i) How likely do you think you are to experience a condition with cancer in the course of your life?

0......1......2......3......4......5......6......7......8......9......10

- definitely will not
- probably will not
- probably will

How anxious or worried are you about the possibility of obtaining a condition with cancer over the course of your life?

0...........1...........2...........3...........4...........5...........6...........7...........8...........9...........10

- not at all worried
- moderate worry
- very worried

ii) How likely do you think you are to experience a coronary heart disease in the course of your life?

0......1......2......3......4......5......6......7......8......9......10

- definitely will not
- probably will not
- probably will

ii) How anxious or worried are you about the possibility of obtaining a coronary heart disease over the course of your life?

0...........1...........2...........3...........4...........5...........6...........7...........8...........9...........10

- not at all worried
- moderate worry
- very worried

Note stop watch: _____
**Full interview condition:** explain the purpose of the study and answer any questions.

establish trust in the study and interviewer.

*Note stopwatch: _____ ask subject about positive and negative life experiences and typical reactions.*

- ensure understanding and trust before proceeding with questionnaire.
- Prediction of IRI risk status: T1  T2  T3  T4

*Note stopwatch: _____ and begin administering the IRI.*

- Does the subject inquire as to the implications of their scores? Yes/No
- require prompting? Yes/No

*Note stopwatch: _____ If 'Yes' work out score and note it here: T ___ T ___ T ___ T ___*

*Note stopwatch: _____ and provide score to subject*

- Ask cancer/CHD anxiety questions: note stopwatch time ______.

i) How likely do you think you are to experience a condition with cancer in the course of your life? (place an * on the dotted line)

0......1......2......3......4......5......6......7......8......9......10

definitely will not 50-50 definitely will

probably will not probably will

How anxious or worried are you about the possibility of obtaining a condition with cancer over the course of your life? (place an * on the dotted line)

0...........1...........2...........3...........4...........5...........6...........7...........8...........9...........10

not at all worried moderate worry very worried

ii) How likely do you think you are to experience a coronary heart disease in the course of your life?

0......1......2......3......4......5......6......7......8......9......10

definitely will not 50-50 definitely will

probably will not probably will

How anxious or worried are you about the possibility of obtaining a coronary heart disease over the course of your life?

0...........1...........2...........3...........4...........5...........6...........7...........8...........9...........10

not at all worried moderate worry very worried

*Note stopwatch time ______ begin debrief*

- Note HRM reading when debrief has been successful ______
**Self Admin Condition:**
note stopwatch time ____ and give subject questionnaire
note stopwatch time ____ and receive questionnaire

Note stopwatch _____ obtain anxiety scores above, Note stopwatch ____ Discussion?

Ask cancer/CHD anxiety questions

How likely do you think you are to experience a condition of cancer in the course of your life?

0….1…..2…3…..4…..5…..6……7…..8…..9…..10
not at all anxious moderately very anxious

How likely do you think you are to experience a condition of CHD in the course of your life?

0….1…..2…3…..4…..5…..6……7…..8…..9…..10
not at all anxious moderately very anxious

How anxious are you about the prospect of obtaining cancer?

0….1…..2…3…..4…..5…..6……7…..8…..9…..10
not at all anxious moderately very anxious

How anxious are you about the prospect of obtaining CHD?

0….1…..2…3…..4…..5…..6……7…..8…..9…..10
not at all anxious moderately very anxious

Does the subject inquire as to the implications for their scores?  Yes/ No If ‘Yes’ work out the score.

Begin debrief: note HRM reading when debrief has been successful.
Appendix 9: Implications for prevention of illness.

A German professor of psychology, Manfred Amelang of Heidelberg University has conducted research in order to assess the strength of Grossarth-Maticke’s reports. In one study, he had health people, people with cancer and people with heart disease complete the questionnaire which you have just done for me, the Short Interpersonal Reactions Inventory. If this questionnaire was actually able to predict who would remain healthy and who would get cancer or heart disease, you would expect it to be able to predict in his sample who actually had cancer or heart disease, and who was healthy- just based on people’s answers to the questions. What he found was that the IRI was able to distinguish between those that were unwell and those that were healthy; but of the unwell people, if was unable to distinguish who had cancer and who had heart disease. This finding severely limits confidence in Grossarth-Maticke’s reports. It also creates the possibility that people’s scores were influenced by their health condition, ie. people with cancer and heart disease were struggling psychologically, so their answers to questions were different to people who were healthy and therefore not struggling in the same way. Amelang’s result lead to a lot of uncertainty about Grossarth-Maticke’s claims.

If Grossarth-Maticke’s claims are accurate, another aspect of his study is very important. In one study, he had a sample of people that were assessed as being cancer or heart disease prone, and gave them the option of some short term psychological intervention to help them change those aspects of their behaviour and personality. About half of these people accepted the offer and the other half didn’t, so these constituted a control group and a treatment group. With those that did accept the offer, Grossarth-Maticke saw them on average around 6 times and provided his own style of cognitive-behavioural therapy. He helped people, for example if they were assessed as being cancer prone, to learn how to recognize strong negative feelings; how to express these to others and realize the world wouldn’t fall apart if they did; how to cope with stress, how to develop strategies to solve problems, etc. And for people assessed as heart disease prone, he taught them also how to solve problems and to learn how to cope, as well as how to manage their anger and hostility towards others. When he followed these people up years later, he found that in
the control group, those who had no treatment at all, there was a fairly high incidence of cancer and heart disease, as well as other serious illnesses. When he followed up the treatment group, he found that very few of them had experienced cancer or heart disease, or other serious illnesses- they were much more healthy. So, it seems that what he managed to do was to help the people who were assessed as being at risk to reverse their risk status- this suggests that you can do things to minimize the effects of the psychological risk factor. If you begin to focus on the aspects of personality which he describes and try to change them, then it seems that there is a good chance that you can actually improve your health status years later. So, for example, you may begin to look at ways of expressing your concerns or upsets to other people; or becoming more assertive; or developing new problem solving strategies.

The other thing worth noting is that the greatest predictor of both cancer and heart disease is age- the older you get, the greater chance you have of getting either cancer or heart disease. The at risk age group is generally 60 plus, so if you are a young adult, you have around 35 or 40 years before you enter that age group, and all that time to change some of these tendencies you’ve got!

Counselling can be an effective way of addressing some of these behaviours- there is a free counseling service at the university for both staff and students, and at places like community health centres. The basic rule of thumb with counseling is that if it makes you feel more helpless and hopeless, then it isn’t working for you and may be of no use for your health; but if you feel less helpless and hopeless because of it, then it is most likely good for you. Self help books can be useful as well- a very good one is by American psychologist Martin Seligman called ‘Learnt Optimism’. This book is available at the university library, Lismore city library and local bookshops. It has lots of stress reducing strategies which are very similar to those which Grossarth-Matick taught his subjects.

So, overall, if you are able to address some of the personality characteristics described by Grossarth-Matick, either by just being aware of them and counteracting them or by counseling or self help books, then, if Grossarth-Matick is right, there should be a really good chance for you to reverse this risk factor over time.
As stated earlier, however, it is as yet unknown if Grossarth-Matischek’s conclusions are actually correct or not. There may be other factors which added to his results that are not yet fully understood. As such, there is no reason to have confidence in his predictions at this time, and they cannot be viewed as a prognosis of health or illness.

If Grossarth-Matichek is wrong, then it probably means that the other researchers in health psychology are right and that psychological factors are only moderate risk factors, in which case personality is only one of a range of factors which may make up health or cancer or heart disease. This means that you would do well to look at these other factors, for example smoking, drinking, diet, levels of exercise- those life style factors which you can actually do something about. It might also help you in life to look at the personality aspects which Grossarth-Matichek described, but if he is wrong, changing them may not have much effect on your overall health status.

Please feel free to contact me if you wish to discuss any of these issues.

James Alexander- Psychologist.
Index:

THE ROLE OF PSYCHOSOCIAL EXPERIENCE IN CHRONIC DISEASE:

**Chapter 1. Introduction:** p.1-20
Psychology and traditional medicine:
The Biomedical Model of Medicine:
Early psychological research on cancer:

**Chapter 2. Reports of Grossarth-Maticek:** p.21-55
Enter Grossarth-Maticek:
Assessment of the cancer and CHD personality:
Grossarth-Maticeck’s Yugoslav study:
Grossarth-Maticeck’s Heidelberg study:
Grossarth-Maticeck’s therapeutic intervention study:
The role of cigarette consumption:

**Chapter 3. Psychoneuroimmunology:** p.56-77
The role of Psychological stress in PNI:
The immune system and stress:
Eysenck’s theory of causal links:
The neurological component of chronic stress

**Chapter 4. Personality and CHD:** p.78-97
Type A and the measurement of the CHD personality:
Heart Rate Reactivity and CHD:
Anxiety & CHD:
Depression & CHD:
Heart Rate Variability, hostility and CHD:
Depression & CHD:
National Heart Foundation of Australia position paper:
The Stress-Diathesis and Cascade theories of disease:

Chapter 6. The Role of Learned Helplessness in Cancer: p.106-111
Learned Helplessness:

Chapter 7. Gender, stereotypes, personality & disease: p.112-131
Men’s emotional and interpersonal styles, cancer & chd:
Depression, gender and cancer:
The male gender role and disease:
Type A and sex role identity:

Chapter 8. Empirically based challenges to Grossarth-Maticek & Eysenck:p.132-145
Amelang’s cross sectional analysis:
The anomaly of Neuroticism and ill-health:
The role of repression:

Chapter 9. Experimenter Bias: p. 146-173
Did Grossarth-Maticek help belief to become biology?
Methodological issues:
The clue:
Experimenter bias:
Making sense of Grossarth-Maticek’s results:
The effects of stressful life events:
Belonging to a cancer risk group:
Grossarth-Maticek’s interview as a stressful life event:

Chapter 10. The power of expectations: p.174-186
Evidence suggestive of self fulfilling prophecies:
A Psychological Model of Expectations:

Risk perception and response to risk notification:
Cognitive processing models of risk perception:
Characteristics of the risk communicator:
Characteristics of the message recipient.
First order relatives as a natural risk group:
Psychological consequences of being in an ‘at-risk’ group:
Synthesis

Chapter 12. The Current Study p. 225-252

Testing an alternate explanation of Grossarth-Maticek’s results:
Study 1:
Study 2:
Heart Rate reactivity to psychological experience:
Possible confounds in heart rate research:
The use of Self Report measures:


Study 1 & 2
Study Protocols:
Participants
Steps involved:
Interview sequences:
The Measures in Studies 1 & 2:
Reliability and Validity data for Study 1 measures:
Study 1 measures:
Study 1 Hypotheses:
Study 2 measures:
Study 2 Hypotheses:
**Chapter 14. Results:** p.272-320

Descriptive analysis:
Analysis of Grossarth-Maticek’s taxonomy:
Study 1: Hypothesis 1- Responses of Heart Rate to risk information:
Study 1: Hypothesis 2- Responses of Self Report measures to risk information:
Study 1: Hypothesis 3- Predictions of Subjects’ Short IRI scores:
Study 2- Effects of familial history, Neuroticism and time on risk perception/anxiety:

**Chapter 15. Discussion:** p.321-355

Section A- The Sample:
Section B- Study 1- Question 1
Study 1 Question 2:
Study 1 Question 3:
Section C- Study 2:
Section D- Possible confounds:
Future Studies:

**Chapter 16. Conclusion:** p.356-368

**Appendices:** p.369-405

**References:** p.406-435
REFERENCES:

*Australian Institute of Health and Welfare*. Canberra: AGPS.


Australian Society for Posttraumatic Mental Health (1999) Posttraumatic Stress Disorder (PTSD) and War-Related Stress. ACPMH, Department of Psychiatry, University of Melbourne, Australia.


de Jong, K; van der Kam, S; Ford, N; Hargreaves, S; van Oosten, R; Cunningham, D; Boots, G; Andrault, E. (2004) The Trauma of ongoing war in Chechnya. Quantitative assessment of living conditions, and psychosocial and general health status among war displaced in Chechnya and Ingushetia. MSF report. 

Derogatis, L (1991) Personality, Stress, Disease and Bias in Epidemiologic Research. 


Fox, B (1999) Personal communication-Wednesday 11th August.


http://www.humanconcern.org/pdfs/Plight_of_War.pdf


Lerman, C; Daly, M; Sands, C; Balshem, A; Lustbader, E; Goldstein, L; James, J; and Engstrom, P (1993) Mammography adherence and psychological distress among women at risk for breast cancer. *Journal of the National Cancer Institute*. 85: 1074-1080.


[www.abc.net.au/news/newsitems/200209/s677093.htm](http://www.abc.net.au/news/newsitems/200209/s677093.htm)


Schwartz, MD; Lerman, C; Miller, S; Masny, A. (1995) Coping disposition, perceived risk, and psychological distress among women at increased risk of ovarian cancer. Health Psychology. 14; 232-235.


2006http://www.abc.net.au/rn/healthreport/stories/2006/1571396.htm#


