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## CANCER, PERSONALITY AND STRESS: PREDICTION AND PREVENTION

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**Abstract** — This paper reviews the history and present position of the theory that there exists a cancer-prone (Type C) personality which succumbs more readily to cancer, and dies more quickly after contracting cancer, than other types of personality. In particular, Type C is characterized by (a) a tendency to suppress emotions like anxiety and anger, and present a bland surface, and (b) to find it difficult to cope with stress, to develop feelings of hopelessness and helplessness, and finally depression. Modern work supports this theory quite strongly, both by controlled comparisons between cancer and other types of patients, and by prospective studies in which healthy cancer-prone subjects are followed up for up to 15 years and compared with subjects who are not cancer-prone, for mortality from and incidence of cancer. Intervention studies show that psychological therapy can (a) prevent cancer from arising, and (b) prolong life in terminal cancer patients. Theories have been developed to identify the way in which psychosocial factors can influence cancer production through affecting the workings of the immune system, and much experimental support has been found for these theories. There appears little doubt that psychosocial determinants constitute an important risk factor for cancer, and interact synergistically with other risk factors such as smoking, genetic influences, etc.

### THE CANCER-PRONE PERSONALITY: HISTORICAL INTRODUCTION

Sir William Osler, often called the father of British medicine, wrote in 1906: "It is many times much more important to know what patient has the disease than what kind of disease the patient has" (pp. 258-259). This belief goes back over 2,000 years, and has been influential in medicine until quite recently, when Pasteur's germ theory of disease persuaded physicians to concentrate on physical causes. Claude Bernard, on the other hand, stressed the important contribution of the host, the soil on which the germ might grow. It is becoming more widely realized now that both are important, and that part of the 'soil' is contributed by psychosocial factors, including personality. The notion of the cancer-prone personality was widely accepted since Galen in his book *De Tumoribus*, suggested a connection between cancer and the *melancholic*, as opposed to the *sanguine* temperament, in the second century AD. (It should be noted here that the meaning of terms like 'melancholia' for ancient Greeks

and Romans may not be identical with what we might attribute to it — Schwarz, 1987.) Greer (1983) and Rosch (1979, 1980*a,b*) have given accounts of the growth of the belief in 'Type C' (Temoshok & Dreher, 1991), to take its place beside the Rosenman and Friedman 'Type A' (coronary disease-prone) and 'Type B' (healthy). Mettler and Mettler (1947) have traced this belief of a possible link between psychological factors and cancer to its ancient origins, and Kowal (1955) has discussed the eighteenth and nineteenth century contributions. Cooper and Payne (1991) also give an historical introduction, as well as a review of modern works.

In connection with the cancer-prone personality, early writers followed Galen in referring to stress and negative emotional reactions, and suggesting difficulties in coping with stress, leading to feelings of hopelessness, helplessness and depression (Gendron, 1701; Guy, 1759; Barrows, 1793; Paget, 1870; Nunn, 1882; Snow, 1893); some noted that cancer was more common in sensitive and frustrated individuals (Stern, as quoted in Suess, Kinzel & Scribner, 1973), liable to suppress their emotions. Walshe (1846) summed up the results of many careful observations by experienced clinicians as follows:

"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."

The first half of the twentieth century showed a lack of interest in the psychological precursors of cancer, probably due to the non-experimental, non-statistical nature of the observational evidence; medicine was entering into its scientific stage. Interest revived around 1950, when Miller and Jones (1958) observed in six patients with chronic myelocytic leukemia "marked emotional stress" before the onset of the disease, and Greene and Miller (1958) and Le Shan (1959) reported that the appearance of cancer was frequently preceded by personal loss. Others reported that depression was a frequent precursor of cancer (e.g., Ramecker *et al.*, 1963; Givvaschini & Maslin, 1965; Le Shan, 1966; Bahnson, 1969*a,b*). This period also marked the emergence of research on specific personality correlates of cancer (Apse *et al.*, 1974; Bahnson & Bahnson, 1966; Bahnson 1976, 1980; Baltrush, Stangel & Waltz, 1988; Le Shan, 1961; Le Shan & Reznikoff, 1960). The various traits mentioned include: Being over-co-operative, appeasing, unassertive, over-patient, avoiding conflict, suppressing emotions like anger and anxiety, using repression and denial as coping mechanisms, self-sacrificing, rigid, predisposed to experience hopelessness and depression. One might sum these traits into two major

categories: (1) *Suppression of emotions* of fear and anger, and behaviours appropriate to these emotions, e.g., assertiveness, aggression, confidence, dominance, selfishness, and (2) *inappropriate coping mechanisms*, leading to failure, feelings of hopelessness and helplessness, and finally depression and despair (Eysenck, 1991a). Greer (1983) justly adds: "It must be said that these findings were based largely upon uncontrolled or inadequately controlled studies in which, moreover, the investigators know the diagnosis. Although such methodological shortcomings make it hazardous to draw any conclusion, the reported findings provided a valuable source of hypotheses which could be tested by more rigorous methods." (p. 536).

Before turning to better controlled studies it may be useful to say a few words about linguistic and methodological difficulties in this field. The first problem arises from the fact that while we treat stress and personality as distinct and separate concepts, they are really closely related. As I have postulated elsewhere (Eysenck, 1975), when we say 'stress' we usually mean 'strain', i.e., not the events perceived as stressors, but the individual reactions of the persons so stressed. Stress may be regarded as an objective concatenation of circumstances, e.g., dismissal from one's job; divorce; death of one's parents. But these objective events may produce quite different emotions in the person so afflicted, depending on his or her *personality*. This is just another way of stating the diathesis-stress theory (Grossarth-Maticek, Eysenck & Boyle, 1994). Strain is the combined effect of stress (objectively defined) and diathesis (personality). Personality here refers to traits (primary factors), types (higher order factors), attitudes, coping mechanisms and other non-cognitive aspects of behaviour and its underlying biological and environmental causes (Eysenck & Eysenck, 1985). If this be so, it follows that any distinction must be somewhat artificial; if disease is a function of diathesis and stress, there are obvious difficulties in disentangling diathesis from stress, seeing that strain (the *perception* by the individual of the stressful nature of precipitating events) is the crucial factor, dependent on both stress and personality. This problem has been discussed at great length elsewhere (Grossarth-Maticek, Eysenck & Boyle, 1994) in the context of being sent to an Hitlerian concentration camp as the stress; here I can only mention it *en passant*. In addition, it has been shown that life events are themselves a function of personality differences (Magnus, Diener, Fujita & Pavot, 1993).

Several discussions and investigations of the diathesis-stress theory are available (Munroe & Simons, 1991; Metalsky & Joiner, 1992; Rende & Plomin, 1992). Mostly the diathesis component is taken to be some form of negative emotionality (neuroticism, anxiety, depression), which interacts with stress/hassles to produce the final psychological or physical disease effect (Aldwin, Levenson, Spiro & Bosse, 1989). One obvious problem

in some studies must be that both the diathesis and the diathesis–stress effect are identical; e.g., depression constitutes both the diathesis and the effect. This problem vanishes when the effect is cancer, but then it can be argued that the effect may produce the alleged diathesis, i.e., cancer causes depression (Scherg, 1986). Only prospective studies can solve this problem. This criticism also applies to other diathesis-factors which have been found important, e.g., extraversion–introversion (Duckitt & Broll, 1982). What may be most important for future research is the experimental analysis of the way in which diatheses determine reactions to stress. Thus Bolger and Schilling (1991) have shown that exposure and reactivity to stressors explained over 40% of the distress difference between high- and low-neuroticism subjects. However, neuroticism may not always act as a moderator variable for stress in predicting health status (Denny & Frisch, 1981). Another problem is that personality variables may appear to act indirectly through such factors as social support, which alleviates stress but is in turn partly a function of such variables as extraversion, neuroticism, locus of control, etc. (Kessler, Kendler, Heath, Heath & Eaves, 1992). This paper also raises the important question of the relative importance of genetic and environmental contributions to diathesis (Rende & Plomin, 1992). Clearly what is needed is an *integrated model of personality, coping style, emotion and cancer*.

### EARLY STUDIES OF THE CANCER-PRONE PERSONALITY

It may be useful to look at a few of the early studies designed specifically to test the personality theories derived from 18th and 19th century observers. Several of the early workers were at first attracted by psychoanalytic notions, but soon discovered that these added little to the empirical findings, and in the main served to confuse the issues; modern work has not found it useful to return to these outmoded ‘dynamic’ theories for the most part. Good summaries will be found in Scherg (1986) and Temoshok and Dreher (1991). Thus Bacon, Renneker and Ertler (1952) obtained psychiatric case histories from 40 women with breast cancer, and found that they shared “a masochistic character structure”; 30 had no techniques for discharging anger directly or in sublimated fashion, and 25 had never experienced orgasm. Atypical case histories belonged to women in the oldest age groups; it makes sense that dispositional personality factors would strike at younger women who otherwise would have encountered cancer at a more usual later age. Blumberg, West and Ellis (1954) reported that they were able, with an accuracy of 78%, to make intuitive predictions as to how well a patient

would respond to treatment, compared to other patients with similar clinical pictures. "The fast growing cases may be described as having more defensiveness, a higher anxiety level, and less ability to reduce tension through motor discharge than the slow group . . .". They concluded that patients whose disease progressed rapidly showed "high defensiveness or a strong tendency to prevent the appearance of serenity in the presence of deep inner distress".

Greene, Young and Swisher (1956) reported on 37 women suffering from lymphoma or leukemia, stating as one of the conditions determining the development of the disease "separation from a key object or goal, with ensuing depression". Le Shan and Worthington (1956a, b) studied retrospectively the personality of 250 patients with malignancies, finding a life-history pattern of misfortune in 62% of their neoplastic subjects but only 10% of matched but not cancerous controls. This pattern included childhood experiences which made intense interpersonal relationships appear difficult and dangerous; strong, meaningful relationships became extremely important in the person's life, and when the relationship was lost, the person underwent a period of intense despair which was later repressed. Greene and Miller (1958) found that in 21 out of 23 children with leukemia there occurred separation from parents followed by depression.

Le Shan (1959, 1977, 1990) has published many papers and books, some of which I have already quoted. His work became more rigorous over time, and he began to use control groups, a relatively rare design feature in this early work. Thus he claims to have found 'loss of hope' in between 70 and 80% of his cancer patients, and in only 10% or so of his control groups. Le Shan summarizes his finding by saying that there were certain general features characterizing the typical cancer patient, in addition to 'loss of hope'. These are: (1) Loss of a crucial relationship. (2) Inability to express hostility on one's own behalf. (3) Feelings of despair. (4) Bottling-up of emotion.

It is not difficult to criticize these early studies (Scherg, 1986; Fox, 1978, 1981; Fox & Temoshok, 1988). Personality assessments are subjective and post-hoc, and may be influenced by the investigator's theories. Proper control groups are usually lacking. To speak of 'cancer' as a unitary concept neglects the evidence that different types of cancers may not share particular relations with psychosocial factors. (Some of the early workers actually compared patients with different cancers; thus Stevenson & Grace (1954) found indicators of sexual maladjustment far more frequently in the cervical cancer than in the control group suffering cancer in non-sexual sites.) These studies serve as a transition from the purely observational work of the 18th and 19th centuries to the better controlled studies that were to follow.

## THE BEGINNING OF THE MODERN PERIOD

The work of David Kissen may serve as the point where this transition began (Bahnon, 1969b, 1976). His empirical work began in collaboration with me (Kissen & Eysenck, 1962) and continued until his untimely death (Kissen, 1963a, b, 1964a, b, 1966a, b, 1967, 1968). The crucial features of his work are already apparent in the early joint study: (1) Putting up a specific hypothesis to be tested, in this case the relationship between lung cancer and suppression of emotion. (2) Choice of a suitable control group. (3) Double-blind procedure. (4) Choice of an objective measuring device. (5) Proper statistical analysis. These factors, now commonplace, were quite alien to workers in psychosomatics at the time, and Kissen is rightly regarded as a pioneer in this field. Subjects in our study were 239 patients attending Kissen's chest clinic; they were tested with the Maudsley Personality Inventory, designed as a measure of neuroticism-emotionality (Eysenck, 1959). The hypothesis was that low scores would indicate suppression of emotion, and that those diagnosed later as suffering from lung cancer ( $n = 116$ ) would have lower scores than those in whom cancer in any organ was excluded ( $n = 123$ ). I was ignorant of the diagnosis, which was made *after* administration of the inventory, and Kissen was ignorant of the scores made by the patients when he made his diagnosis.

It was found that the control group had much higher neuroticism scores than the cancer group, significant at the  $p < 0.01$  level. Smoking was ruled out as a likely intermediary. Kissen in his later work (Kissen & Rowe, 1969) repeated this study several times, always with similar results. Altogether, as he stated, the probability of a person, having a low score on the  $N$  scale, being diagnosed as suffering from lung cancer was 6 times as high as a person with a high score being so diagnosed. This estimate, based on several large samples, suggests that even a single personality factor may be quite strongly related to the occurrence of lung cancer. It is interesting to note that Kissen (1964a) already predicted, and found, evidence for a *synergistic* relationship between smoking and personality. As he said, it would appear from his studies that "*the poorer the outlet for emotional discharge the less the exposure to cigarette smoke required to induce lung cancer*" (p. 213). (Emphasis in original). This important point will be taken up again later on.

Many investigations have taken up this paradigm and replicated our findings, extending them to other forms of cancer, e.g., cancer of the breast in women (Berndt, Gunther & Rahte, 1980; Eysenck, 1981). A summary of all this work is given by Eysenck (1985); it is notable that practically all give positive results supporting the hypothesis. We may conclude that there is some acceptable evidence for the hypothesis that cancer is *correlated substantially with suppression of emotion*.

It will be obvious that this approach by Kissen and Eysenck (1962) has a serious weakness which makes the discrimination achieved appear lower than it probably is. A low score on *N* can be produced in two ways. (1) The low *N* scorer is very stable, and genuinely experiences little anxiety and depression. (2) The low *N* scorer suppresses his experiences and feelings of strong anxiety and depression, and denies them. Our methodology combines these two quite different groups, and the fact that the result of the study supported the hypothesis linking cancer with suppression suggests that had we linked cancer with a *pure* measure of suppression, the differences observed between cancer and no cancer groups would have been much larger. Methods to purify our measures of the suppression of *N* have been elaborated, and will be discussed later.

How about the second trait suggested by the early workers in this field, namely a tendency to fail to cope with stress, give up, and develop feelings of hopelessness and helplessness? Here the crucial initial study is one by Schmale and Iker (1971). The population consisted of women recommended for a diagnostic cone biopsy because of repeated evidence of suspicious cervical cells; of 68 women so recommended, 28 were found to have cancer of the cervix, 40 not to have cancer. Interviews held *prior to diagnosis* based prediction of diagnostic outcome "on the presence or absence of reported evidence of a high hopelessness potential and/or a reaction of hopelessness six months prior to the first abnormal (suspicious) smear" (p. 96). Of the 28 women with cancer, 19 had been accurately predicted to have cancer, 9 erroneously not to have cancer, while of the 40 non-cancerous women, 31 had been correctly predicted to be non-cancerous, while 9 had been erroneously predicted to have cancer. Thus 50 out of 68 had been correctly predicted to have cancer, i.e. 74%, significant at  $p < 0.001$ . Other later studies have usually borne out this finding, mainly with other types of cancer (e.g., Scherg, 1986; 1987; Wirsching, Shirlin, Weber, Wirsching & Hoffman, 1981; Goodkin, Antoni & Blaney, 1986; Hislop, Waxler, Coldman, Elwood & Kau, 1987; Horne & Picard, 1979; Greer & Morris, 1975).

Of particular interest is a recent set of results that has been reported by Cooper and Faragher (1992, 1993), Cooper, Cooper and Faragher (1989), and Faragher and Cooper (1990). In a quasi-prospective study they investigated women attending a breast screening out-patient clinic, as well as women attending a general out-patient clinic. Each of the 1596 women had presented to their general practitioners complaining of breast lumpiness or tenderness. Questionnaires were filled-in prior to diagnosis, as in the Kissen and Eysenck (1962) study. In addition a symptom-free group of 567 women was included in the study. Comparisons were made between women with breast cancer, benign tumours, and normal breasts. The main findings were that women with cancer had more

inter-personal problems rated by the individual as having high impact. "Denying the existence of the problem proved to be counter-productive, being associated with an increased risk of cancer" (Cooper & Faragher, 1993, p. 660). "The ability to express anger as a mechanism for handling the stress event again proved to be positive in the sense that it reduced the risk of a poor diagnosis (i.e., cancer)" (*ibid.*). 'Denial coping strategies' *increased* the risk of a woman being found to have breast cancer (p. 659). Interestingly, "women diagnosed as having benign breast disease were most likely to be cigarette smokers".

An interesting finding in this study provided evidence for Eysenck's (1983) 'inoculation' theory. Based on numerous animal studies (e.g., Sklar & Anisman, 1981; Newberry, 1978; Justice, 1985), which had shown inhibitory effects of chronic stress on tumour cell proliferation, and adaptation to the effects of the stressor with repeated exposure, Eysenck (1983) had argued that adaptation to chronic stress had an 'inoculation' effect on the organism. As Sklar and Anisman (1981) put it: "Acute stress results in depletion of catecholamines and increased ACh, increased synthesis and secretions of hormones, and immune-suppression. Adaptation to these biological mechanisms is observed with chronic stress, such that normal levels of functioning or alteration opposite to those induced by acute stress are apparent" (p. 391). In good agreement with this 'inoculation' hypothesis, Cooper and Faragher (1993) found that "a high number of interpersonal problems continued to be related to a non-malignant diagnosis" (p. 660). In contrast, "women who experienced a loss-related event which they perceived as having a major impact on their lives had a significantly increased risk of being diagnosed as having a malignancy relative to all other women in the study" (p. 660). In assessing research on the effects of stress on cancer, the existence of such adaptation or inoculation factors should always be taken into account.

### CRITICISM OF EARLY STUDIES

How do these studies stand up to criticism? The main criticism that remains is that nearly all the studies are of patients already suffering from cancer, so that it may be that the causal link is from cancer to personality, rather than the other way about. It can no longer be denied that there are strong links between personality and cancer, but only prospective studies can eliminate this type of criticism, i.e., studies in which *healthy* subjects are given interviews or personality tests, and are then followed up to see who dies of cancer. I will consider such studies later. Another type of justified criticism relates to the question of *causality*. We have demonstrated a correlational link between personality and cancer, but it may not be a causal one. Causality can only be demonstrated by



intervention studies, i.e., by studies demonstrating that assumed causation can be reversed by prophylactic therapy aimed at changing the personality traits shown to act as risk factors for cancer. If such treatment is successful, a causal relation seems indicated (Eysenck, 1991a). Another way to indicate causality is by demonstrating experimentally the nature of the link between personality and cancer, i.e., by showing how it is possible for personality to act as a risk factor for cancer (Eysenck, 1991a). In later sections I will try to indicate to what extent such demonstrations of intervention and linkage have been successful.

Critics of more recent studies, like Scherg (1986) and Fox (1978) often argue that there are a number of negative studies which should be taken to counterbalance the positive studies reported, and carry out what is a kind of meta-analysis of all published work. Thus Scherg lists details of 40 separate studies; by now this number could be doubled. I have several times argued against the use of meta-analysis (e.g., Eysenck, 1992a), for the simple reason that it is not reasonable to argue that a good study, properly conducted and analysed, should be averaged with a bad study, using inappropriate methodology and statistics. Consider the following example, discussed at some length by Eysenck (1990b). It concerns the Schmale and Iker study already mentioned, in which they had found that cancer could be predicted by means of interviews concerning 'hopelessness' feelings. They also used the MMPI and the Rorschach, and failed to find any correlations with cancer! This is hardly surprising — the MMPI is a multi-purpose questionnaire having little relevance to the theory being tested, and the Rorschach lacks reliability and validity (Zubin, Eron & Schumer, 1965), and is equally irrelevant. Yet any busy meta-analyst would have scored the study a 'failure' if only these two tests had been used!

The problem with so many published papers is precisely this: they are not designed to test a specific theory, they use tests that are not relevant to the purpose, and often apply these to badly chosen groups. The frequent use of the MMPI or the Rorschach is a case in point; neither test is designed to test the major theories in the field, so that any failure to distinguish cancer patients or cancer-risk probands from controls is irrelevant to the theories that have held the stage for such a long time. When a proper measure is being used, as in the Schmale and Iker study, high levels of prediction can be attained. To combine good and bad studies in a meta-analysis, and conclude that there is inconsistency, is not very illuminating; no psychologist knowledgeable in the field would have expected anything else from the use of badly chosen tests. Unfortunately epidemiology is a very immature discipline (Burch, 1986; Eysenck, 1991a; Feinstein, 1988), and particularly in relation to psychological matters it has not always made wise choices in respect to the measures used.

There is another point. In the Schmale and Iker study the successful technique used a focussed interviewing technique, and this has also been found significantly more successful than questionnaires in prediction studies of the Type A–Type B concept (Eysenck, 1990a); possibly an interviewing procedure elicits better co-operation than merely handing out questionnaires. Grossarth-Maticek, Eysenck & Barrett (1993) have put this hypothesis to the test in a large-scale prospective study, and found marked differences in predictive accuracy for the eventual occurrence of cancer and coronary heart disease, depending on the degree of interviewer participation. The use of interviewers may be more expensive and time-consuming than simply handing out questionnaires, but if theories of cancer–personality correlation are to be tested properly, this clearly is the method of choice; negative results not using optimal methods of data collection cannot be used to discredit positive results achieved by using trained interviewers.

#### THE EMPIRICAL STUDY OF SUPPRESSION OF EMOTION

Even more incisive than interviews and questionnaires are experiments specifically designed to test hypotheses. As an example, consider the Kneier and Temoshok (1984) study of suppression of emotion in cancer patients. As a contrast to ‘Type C’ cancer patients they chose ‘Type A’ coronary heart disease patients, whose personalities are supposed to be governed by the ‘AHA’ trio of anger, hostility and aggression, i.e. traits opposite to the emotion-suppressive personality of the cancer-prone patient. Also included was a healthy control group, predicted to be intermediate between the other two. Participants were shown 50 slides designed to disturb the subjects emotionally, and provoke anger, sadness, anxiety, threats to self-esteem, or threats to interpersonal needs. Psychophysiological measures of autonomic arousal were taken, and subjects were asked how ‘bothered’ they had been by the slides. Subjects were scored as *repressive*, if they denied being bothered but had strong autonomic reactions; as predicted, cancer patients had the highest score on this emotional repression index, CHD patients had the lowest, with normals, (healthy subjects) right in the middle. Such experimental designs lend much-needed support to such studies as the Kissen and Eysenck one, and immeasurably strengthen one’s belief in the correctness of the theory in question. Note also that the choice of control group was dictated by theory, a design much better than the usual choice being dictated by availability! (There is of course a good deal of evidence regarding the differential reactions of cancer-prone and CHD-prone people — e.g., Dixon and Dixon, 1991; Eysenck, 1985, 1991; Grossarth-Maticek, Bastiaans & Kanazir, 1985.)

Another interesting study making an empirical comparison of behavioural differences between 20 cancer and 24 CHD patients was reported by Beck (undated). It followed earlier attempts (Spence, 1978; Lebovits & Holland, 1983) to use the verbal behaviour of patients as an indicator of their specific illness. Hypotheses were constructed, e.g., that cancer patients would use more words that indicated 'death', and that they would use more words concerning 'hopelessness'. An open interview generated the verbal material used. It was found that word clusters denoting emotions like anxiety, death, hostility, victim, hopelessness, shrouds, clearly differentiated between the groups; 'hope' in particular, had a mean of 2.55 in the CHD group, but only 0.19 in the cancer group ( $p < 0.0001$ ). Discriminant analysis showed a complete differentiation between the two groups; "all patients could be correctly classified in the disease group they belonged to" (p. 14). This study would well repay replication.

There is another important point. Theory predicts that the cancer-prone personality suppresses or represses emotion (though of course not in the Freudian sense of the term); how can one measure this tendency by means of questionnaires? In the Kissen and Eysenck (1962) study we used low scores on a neuroticism questionnaire as an indication of such suppression, but of course people low on neuroticism would also have such low scores, as pointed out previously. Gudjonsson (1981) has shown that repressors *combine* low scores on neuroticism/anxiety scales with high scores on lie/social desirability scales; he was able to verify this hypothesis, originally suggested by Weinberger, Schwartz and Davidson (1979) and Weinstein, Averill, Upton and Lazarus (1968), by comparing verbal and psychophysiological responses to emotionally loaded questions. This would sort out low *N* scorers into *true* non-anxious and defensive suppressors of anxiety in terms of low and high *L* scores respectively. Kreitler and Kreitler (1990) give a detailed history and discussion of the 'repression' concept. The correlation between cancer-proneness and *N* has not always been found to be negative, as in the Kissen and Eysenck (1962) study; it appears to be positive in the recent study by Schmitz (1992). But in the Schmitz study the subjects had come for 'autogenic training' therapy, and might therefore be considered to be more likely to drop their defences and admit to their true emotions, without the need to suppress them.

As pointed out above, the indication of suppression of *N* can be measured with greater clarity than just using low *N* scores by using *L* scores in addition to *N* scores. This would give us 4 groups. High *N*/Low *L* are the truly anxious persons. Low *N*/Low *L* are the truly low anxious persons. Low *N*/High *L* are the repressors. High *N*/High *L* are defensive high anxious, a rather rare breed, often left out of experimental

studies. (Myers, 1993). Instead of the *L* scale, the Marlowe–Crowne Social Desirability Scale (Crowne & Marlowe, 1964) has often been used, but this may not be a good choice because the MC scale correlates significantly with *N*; a correlation of  $-0.47$  is reported by Gudjonsson (1981). Plante and Schwartz (1990) have noted that the two scales (*L* and MC) should not be used interchangeably, and the lower correlation of *L* with *N* would seem to give it the advantage. There is strong support for this way of measuring suppression of emotion (e.g., Asendorpf & Scherer, 1983; Boor & Schill, 1967; Holroyd, 1972; Jamner & Schwartz, 1986; Newton & Contrada, 1992; Schwartz, 1990; Weinberger, 1990; and many others; for a review see Myers, 1993).

Another scale that has sometimes been used is the Byrne Repression–Sensitization scale (Byrne, 1964; Bell & Byrne, 1978). Unfortunately this scale is highly correlated with measures of *N*, such as the Manifest Anxiety Scale, with which it shares many items (Sullivan & Roberts, 1969). These authors report a correlation of 0.91, and similarly high correlations are found between the R–S scale and such measures of anxiety–neuroticism as the Spielberger Trait Anxiety scale and the Eysenck Neuroticism scale (0.88 and 0.84, respectively — Slough, Kleinknedt & Thorndike, 1984). Clearly the R–S scale is just another measure of *N*, rather than of repression, in spite of its name.

Several studies have successfully correlated repressive-coping personalities with cancer (e.g., Canning, Canning & Boyce, 1992; Jensen, 1987), but coronary heart disease has given ambivalent results (Myers, 1993), some negative, like the Kneier and Temoshok (1984) study already discussed, some positive, but the latter usually measure indirect physiological correlations of CHD, and the results are difficult to interpret (Temoshok, Van Dyke & Zegans, 1983).

The usual run of studies reporting negative results in this area pays surprisingly little attention to psychological considerations of the kind mentioned; perhaps this is not unexpected in epidemiologists without any training in psychology. But no rejection of the theories here considered is justified on the basis of mumpsimus studies of this kind. It is unfortunate that we have on the one hand writers like Le Shan, Bahnson and others mentioned earlier, who carefully consider each individual case, with great psychological insight, but little concern for methodological and statistical requirements, while on the other, we have workers with little psychological insight or concern who automatically apply irrelevant measures to groups of patients not studied personally. However methodologically sound their designs, they do not usually test the theories in question. Clearly what is needed is some regard for the spirit as well as the letter of the theories to be tested; some psychological insight into the subjects' reactions to the testing process; some concern with the psychological meaning of the testing

process for individuals who may be severely stressed, ill, or suspicious of the whole testing procedure. To administer *tout court* an inventory of anxiety or depression and expect this to test complex psychological theories is not a useful procedure.

### MODERN CONCEPTS OF VULNERABILITY

We have two major theories in need of evidence. The first is that there is a general factor of personality structure which makes for health, as opposed to sickness; let us call this H, for health. We also have a second factor opposing cancer and CHD; let us call this D, for difference. Such findings as the Seltzer and Jablon (1977) and Friedman *et al.* (1993) demonstrations, which show that longevity can be predicted successfully from army rank, even with socio-economic factors controlled, and from childhood personality, support the hypothesis of a healthy personality. A paper by Friedman and Booth-Kewley (1987) on the disease-prone personality supports this notion on the basis of a review of the available evidence: "there is sufficient evidence to argue for a key role for psychological research on the prevention and treatment of disease" (p. 539).

There has been much interest in the psychological nature underlying this H concept, and several hypotheses have been put forward. One of these concepts is that of alexithymia (Lesser, 1981; Taylor, 1984), a construct whose essential features are a difficulty in identifying and describing feelings, a difficulty in distinguishing between feelings and bodily sensations, and restricted imaginative processes. The scale correlates positively with neuroticism, negatively with extraversion, and positively with somatic complaints; it seems to be an H-scale, correlated with non-health (Parker, Bagby & Taylor, in press). Affect intensity (Larsen & Diener, 1987) seems related to alexithymia, and has been found positively correlated with somatic complaints. Self-directedness (Rodin, Schooler & Schaie, 1990) is another such concept, in some sense the opposite of affect intensity and alexithymia. Ambivalence over emotional expression (King & Emmons, 1990) rather resembles the latter. Low sympathetic arousal (Dienstbier, 1989) in response to intermittent stress is regarded as a *positive* health variable. Hardiness (Kobasa, Maddi & Kahn, 1982) is also regarded as a positive predictor of H (Roth, Wiehe, Fillingim & Shay, 1989). Optimism (Scheier & Carver, 1985) is also H-positive, as are fighting spirit (Nelson *et al.*, 1989), and self-efficacy (Bandura, Cioffi, Taylor & Brouillard, 1988). Finally, we have the concept of the 'health-oriented individual' by Kreitler & Kreitler (1991), which is also H+ oriented. The concept of the 'dependent personality' (Greenberg & Bornstein (1988) is another H-trait complex. Most of these concepts have

only been linked with minor stresses and psychosomatic disorders, usually in students. It would be interesting to study their relation, prospectively and contemporaneously, with cancer.

An indication of the possibilities inherent in this approach is given in a prospective study by Grossarth-Maticek and Eysenck (in press), covering a random sample of 5,716 people aged from 40 to 68, carried out in Heidelberg, Germany. The questionnaire was constructed to measure the concept of *self-regulation*, or autonomy; it was applied to healthy probands in 1973, and mortality was ascertained in 1988, i.e., 15 years later. Self-regulation (S-R) or autonomy refers to the ability of the individual to adapt to circumstances, control his or her emotions, cope adequately with external stressors, not to repress the expression of his feelings, being physically active, being assertive but not aggressive, flexible rather than rigid, and able to alter his or her behaviour to achieve satisfaction of

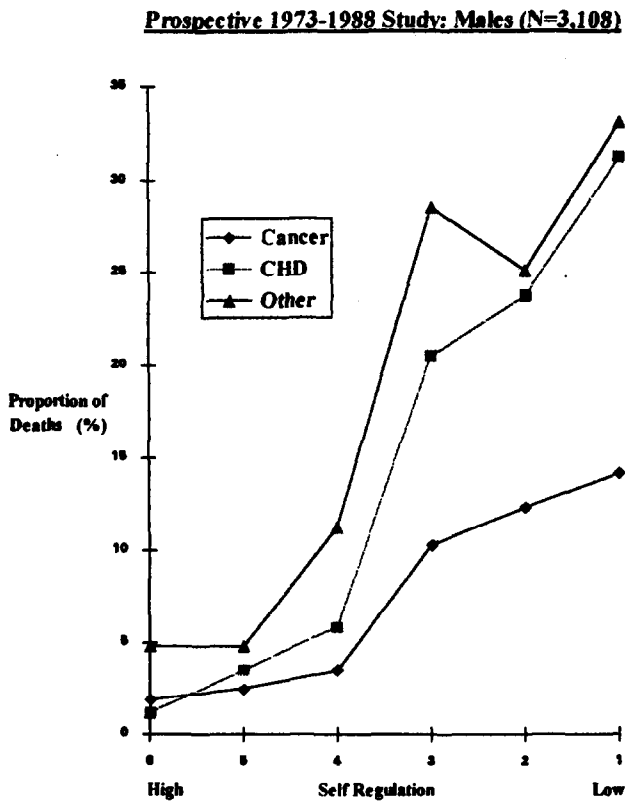


FIG. 1. Death from cancer, coronary heart disease and other causes as a function of degree of self-regulation (Grossarth-Maticek & Eysenck, in press). Data for males.

important life aims. A 105-question inventory is answered on a 6-point Likert-type scale for each question, and then reduced to a total score ranging from 1 (low S-R) to 6 (high S-R). Figures 1 and 2 show the inverse relation between mortality and S-R; as S-R decreases from 6 to 1, mortality increases for cancer, CHD and other causes. Results are similar for both sexes, and achieve a very high level of statistical significance. This is a personality component of H whose contribution cannot be attributed to factors like differential consumption of cigarettes, alcohol, etc., all of which have been found quite incapable of explaining the results.

### PROSPECTIVE STUDIES: SURVIVAL

Prospective studies, involving follow-up of groups studied at point  $T_1$ , and analysed for mortality at point  $T_2$ , are the 'gold standard'

#### Prospective 1973-1988 Study: Females (N=2,608)

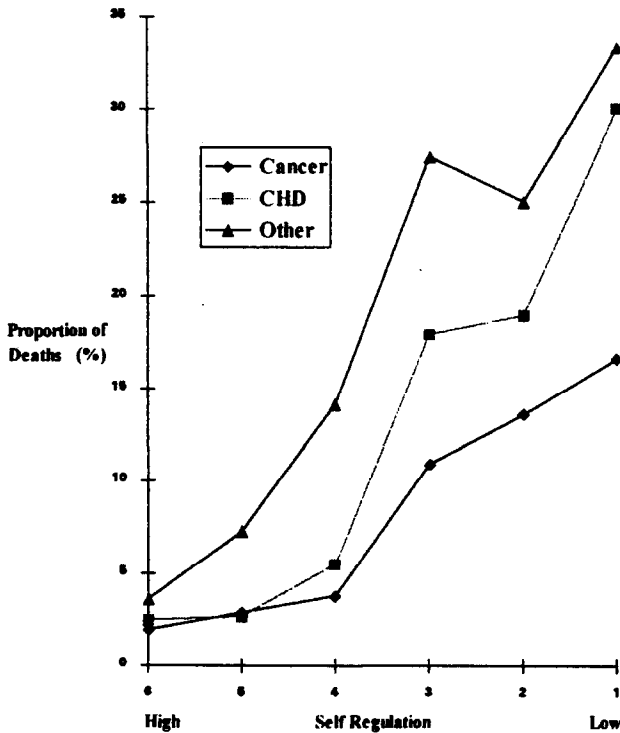


FIG. 2. Death from cancer, coronary heart disease and other causes as a function of degree of self-regulation (Grossarth-Maticek & Eysenck, in press). Data for females.

Table 1. Survival Time of Cancer Patients as a Function of Score on the Self-Regulation Inventory (SR)

N	Survival time in years	Mean survival time	Type of carcinoma									
			Mammary		Colorectal		Stomach		Bronchial		Other tumours	
			N	S-N:	N	S-R	N	S-R	N	S-R	N	S-R
156	1-2	1.4	10	2.1	21	2.2	31	2.1	45	2.3	49	2.4
156	3-5	4.3	15	2.7	31	2.6	27	3.7	37	3.8	46	3.7
117	6-9	7.8	20	3.6	21	3.5	12	4.1	10	4.0	54	3.9
103	10-17	14.7	19	4.1	17	4.0	5	4.2	6	4.5	56	4.8
89	18-27	23.9	10	5.2	23	5.1	3	5.6	2	5.6	51	4.9
Total: 621			74		113		78		100		256	

of mind-cancer research (Temoshok & Dreher, 1992). There are two clearly differentiated types of prospective studies. One, to be discussed in this section, studies cancer patients at  $T_1$ , measuring theoretically relevant personality and stress factors and at  $T_2$  determines how many have survived. Alternatively survival time might be the dependent variable, so  $T_2$  is not fixed. As an example of the latter type of analysis, consider the prediction of cancer from scores on the self-regulation questionnaire (Grossarth-Maticek & Eysenck, in press) results from which were shown in Figs 1 and 2. Table 1 shows the self-regulation scores of 5 groups of patients suffering from different types of cancer, related to years of survival. It will be seen that patients only surviving one or two years had low S-R scores (between 2.1 and 2.4), while those surviving 18 years or more had S-R scores between 4.9 and 5.6. There is a linear relation between S-R scores and duration of survival in each of the 5 groups, suggesting that S-R has a positive influence on survival.

There are two problems with data of this kind. In the first place, many of the traits suggested to favour survival may be correlated with known physical risk factors. Thus trait X, supposedly favouring survival, may be correlated with low frequency of drinking and smoking, good eating habits, etc. One might think that a simple partial correlation procedure would be able to sort out problems of this kind. This is not so. Consider Table 2, which shows the relationship between smoking and drinking, on the one hand, and self-regulation scores, on the other; data are given separately for the part of the sample that died, and the part that survived. For those who survived, drinking and smoking actually went up with greater self-regulation; for those who died, drinking and smoking actually went down with greater self-regulation! Overall there



Table 2. Consumption of Tobacco and Alcohol in Cancer Patients, According to Self-Regulation Scores

	1	2	3	4	5	6	
	%	%	%	%	%	%	
Live	15.3	15.6	14.7	24.6	21.7	22.0	Cigarettes per diem
Deceased	26.9	25.6	24.3	23.9	21.3	21.3	
Live	21.6	23.6	39.8	48.7	42.6	44.6	Alcohol: grams per diem
Deceased	75.8	79.4	69.6	28.3	24.2	25.3	

was a slight, irregular downward trend for both groups. It is possible to conclude that contradictory effects of this kind make statistical treatment difficult, but the data do not suggest that much if any of the S-R effect is due to smoking or drinking.

A second problem is the fact that the effects of drinking (and probably smoking, too) are dependent on motivational factors (Grossarth-Maticek & Eysenck, 1991a). People drinking alcohol suffer greater mortality if they drink to drown their sorrows than if they drink to celebrate, or for fun. And finally, some types of non-alcoholic drink, i.e., coffee, have opposite effects on the probability of developing cancer and CHD, increasing the probability of CHD and lowering that of cancer (Grossarth-Maticek & Eysenck, 1990a); presumably alcohol has the opposite effect. We have tried to explain these effects as far as cancer is concerned (Grossarth-Maticek, Eysenck & Rakic, 1991), but whatever the explanation clearly these effects are very complex, and taken together with the evidence later to be presented that psychological and physical factors in cancer genesis are synergistic it must be obvious that simple presentation of single factor correlations, as is customary in the literature, is not sufficient for proper causal analyses. Unfortunately, many of the studies reported in the literature are of this sort.

Greer, Morris and Pettingale (1979) found that survival 5 years after the diagnosis of breast cancer was significantly related to psychological traits assessed at 3 months. Women considered on the basis of a structured interview to show 'fighting spirit' had a better prognosis than those displaying stoic acceptance or helplessness and hopelessness. Thus cancer-prone women, using the traditional conception of that term, survived less well than those showing the opposite type of personality. Similarly, Derogatis, Abeloff and Melisaratos (1979) found that women with breast cancer who survived more than one year had higher ratings on measures

of hostility or anger ('fighting spirit') than those who died within the first year. In an even earlier study, Blumberg, West and Ellis (1954) studied two groups of cancer patients matched for age, intelligence and stage of cancer, administering the MMPI following initial treatment. The study was more focussed than many using this multi-purpose instrument; they found that those dying in less than 2 years, as compared with those dying after more than 6 years, had higher depression scores and lower neurotic outlet scores, as well as very low acting-out scores at the time of first assessment. Stravaky, Buyck, Loft and Wancklin (1968) found their long survivors angrier, but without loss of control; they showed an underlying hostility or aggressiveness.

In the original Greer, Morris and Pettingale study (1979) the authors had categorized coping styles of their patients, in addition to the 'fighting spirit' category, such as denial, stoic acceptance, and hopelessness-helplessness; 81% of 'fighting spirit' patients were still alive, but only 20% of the hopeless/helpless group. Follow-up studies were done at 10, and again at 15 years from the outset of the study (Greer, Pettingale, Morris & Haybittle, 1985; Greer, Morris, Pettingale & Haybittle, 1990), finding that the original results held strongly. Fighters and deniers were more than twice as likely to be alive than helpless/hopeless patients. This is one of the most persuasive studies in the field. The main results were replicated in a similar study by Di Clemente and Temoshok (1985), using patients suffering from malignant melanomas. Among women, stoic acceptance made for a low or bad prognosis; among men, hopelessness/helplessness made for a relapse.

What is particularly interesting in these studies, to which many others could be added, e.g., Cella and Holland (1988), Pettingale (1984), Temoshok (1985) and Temoshok *et al.* (1985) is that results are in good agreement with prediction from the other studies reviewed in preceding sections, and with the theories and observations described. It seems that the cancer-prone person is not only more likely to develop cancer, but if ill with cancer is less likely to survive. Of course there are many problems inherent in this type of study; how, for instance, can we be sure that the patients less likely to survive are equal with the longer survivors with respect to stage of illness? Personality factors are known to influence when in the development of cancer the sufferer seeks medical help (Berndt, Gunther & Rahte, 1980); perhaps this is another factor linking personality and survival? But it seems unlikely that these difficulties can be entirely responsible for the congruence observed. This conclusion will be strengthened when we consider the results of prospective studies of the second kind, in which healthy subjects are interviewed and/or given questionnaires, and then followed up for lengthy periods to check on mortality.

### PROSPECTIVE STUDIES: MORTALITY

Studies in this section are devoted to efforts to predict mortality from cancer (and/or other diseases) in healthy individuals investigated at  $T_1$ , and followed up for a period of years to a point  $T_2$ , when mortality and incidence are ascertained, i.e., who has died of cancer (or whatever), and who has been so diagnosed, but is still alive. In the nature of things there are few such studies, for obvious reasons.

(1) Large numbers of probands are required at the beginning if sufficient numbers dying of a specified type of cancer (e.g., bronchial carcinoma) are to be found at  $T_2$ . Even specifying 'bronchial carcinoma' may not be specific enough; we may have to distinguish between epidermoid and adeno-type cancers. Even more refined sub-classes may be asked for by oncologists. In one of our studies dealing with cancer of the breast (not yet published) we started with 8,051 women; of these, 108 died of mammary carcinoma after a 15-year follow-up. Clearly investigators must decide just how specific a diagnosis to investigate. The more specific, the more likely that the group will be homogeneous, but equally, the larger the  $T_1$  population tested will have to be. A compromise is essential, and will inevitably be criticized. If you look at cancer in general, you may get away with a starting group of 1,000; if you look at a specific type of cancer, you may need 5,000–10,000. Success in finding a connection between personality/stress at  $T_1$  and mortality/incidence at  $T_2$  will justify your choice; failure is ambiguous.

(2) Another problem is the selection of your original sample. It is of course necessary to have as random a sample as possible, but limitations are needed as far as sex and age are concerned. If you are concerned with cancer of the breast, or the cervix, you obviously require only women. As far as age is concerned, probands under 40 are out; with the average life expectancy between 70 and 80, it would take too long for any reasonable percentage to die in the life-time of the investigator! But choosing the age limits of one's sample has other problems. If cancer-proneness bears a dose-response relationship to mortality/incidence, as it probably does, then the most prone would be expected to die relatively young; this suggests a relatively young sample. But that would require a much larger sample, because not many would die of cancer! Decisions of this kind are difficult to make, but they may determine outcome. A very old sample will have high mortality in minimum time, but may show much less connection between cancer-proneness and personality/stress than a relatively young sample. Such differences in selection may lead to 'failures to replicate', where different age samples are being compared.

Some of these studies are relatively restricted in their coverage, but nevertheless relevant. Often they are unplanned outcomes of analyses

carried out because the data were available; these might be called 'convenience' studies, without a strong theoretical basis. Kaplan and Reynolds (1988), in a 1-year follow-up study of 6,848 healthy people, found an increased risk for cancer incidence and mortality among those who were 'socially isolated'. Shekelle *et al.* (1981) and Persky, Kempthorne-Rawson and Shekelle (1987) in the Western Electric Study, discovered that those found on the original test to be depressed had twice as high a risk of death from cancer as those low on depression. At first this seems to contradict the Kissen and Eysenck results, showing a *negative* relation between neuroticism and cancer, but Temoshok and Dreher (1992, p. 115) point out that the correlation was with death from cancer, not with onset. Hence, it is related negatively to the lack of fighting spirit shown in the previous section to prolong life.

More in line with expectation are the findings of Dattore, Shontz and Coyle (1980) who followed up 200 disease-free veterans who had been tested upon entry into a VA hospital. Comparing the records of 75 who went on to contract cancer with 125 who remained healthy, or developed other diseases, the cancer patients were far *less depressed*, and significantly *more repressed*, than the control subjects. This agrees with the Kissen and Eysenck (1962) results, and, as Temoshok and Dreher puts it, is "compelling evidence that Type C is a cancer-risk factor" (p. 116).

One of the oldest and longest-continued studies was begun by C. B. Thomas (Shaffer, Graves, Swanck & Pearson, 1987) and was continued for 40 years; Temoshok and Dreher (1992) gave a list of references to successive reports. Thomas never suspected a correlation of personality with cancer, being concerned with coronary heart disease; thus her findings are all the more convincing, confounding the chance of any possible prejudice dictating results. She 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! The fact that this and other prospective studies support the Kissen and Eysenck (1962) result, and indeed produce even better discrimination between cancer and non-cancer patients, suggests strongly that it is not cancer that causes personality changes, but personality-stress that causes cancer.

### THE PROSPECTIVE STUDIES OF GROSSARTH-MATICEK

We must finally turn to the work of Grossarth-Maticek, which is more voluminous than most of the other prospective studies (Eysenck, 1991a, has presented the major results in book form). Grossarth-Maticek has published 3 major follow-up studies, one from Yugoslavia (Grossarth-Maticek, Kanazir, Schmidt & Vetter, 1982), and two from Heidelberg

(Grossarth-Maticcek *et al.*, 1985; Grossarth-Maticcek, Eysenck & Vetter, 1988). In all cases, healthy individuals were selected on a randomized basis, interviewer-applied questionnaires were used, medical tests applied, and information collected on smoking, drinking and other life-style habits, by trained interviewers. Mortality and incidence were assessed after a ten-year follow-up, with independent supervision. Two types of data were collected to assess personality/stress. The first was by means of a set of trait inventories theoretically based to predict cancer or coronary heart disease. Particularly relevant to the concept of Type C are two questionnaires, namely (1) Number of traumatic life-events evoking chronic helplessness, and (2) rational-antiemotional behaviour (suppression of emotion). A third questionnaire deals with anger, and is predictive of CHD, as opposed to cancer: (3) number of traumatic life-events evoking chronic excitement. A path model with cancer as the dependent variable was constructed, and these 3 variables had standardized partial regression coefficients of 0.43, 0.41, and  $-0.32$ , exactly as predicted from theory (Grossarth-Maticcek, Kanazir, Schmidt & Vetter, 1982, p. 297). Four other questionnaires added very little, and the explained variance for the seven variables combined, in the prediction of cancer, is 0.55, with the contribution in the first 3 components amounting to 0.49. In other words, about half the cancer variance is due to personality/stress factors (Eysenck, 1988) (see Table 3). Psychological predictors were found to be more important in

Table 3. Determinants of Cancer: Seven Scales Measuring Personality Traits (Eysenck, 1988)

$x_1$	Number of traumatic life events evoking chronic hopelessness	0.43	
$x_2$	Number of traumatic life events evoking chronic excitement	$-0.32$	
$x_3$	Rational and anti-emotional behaviour	0.41	
$x_4$	Tendency towards self-abnegation for the sake of harmonious social relationships	0.18	
$x_5$	Lack of hypochondriasis	0.17	
$x_6$	Absence of psychopathological symptoms such as anxiety	0.08	
$x_7$	Lack of positive emotional contact	0.13	
			e ↓ 0.67 Cancer incidence Y

the prediction of cancer than physical predictors (Grossarth-Maticek *et al.*, 1985). These results from a prospective study again support the Kissen and Eysenck (1962) and the Schmale and Iker (1971) studies.

As an alternative to this *normative* type of scale construction, Grossarth-Maticek also used a *semi-ipsative* type which resulted in four scales identifying four types of personality: Type 1 = cancer-prone; Type 2 = coronary heart disease-prone, Type 3 = hysterical; Type 4 = autonomous (healthy). Type 4 is similar to the self-regulating type already described; Types 1 and 2 embody the characteristics historically ascribed to the cancer-prone and the CHD-prone individual. Type 3 is of no particular interest here, but is predicted to be healthy. A person is identified as belonging to that 'type' for which he has the highest score on the questionnaires. This is rather a clumsy kind of statistic, adopted presumably because it tends to appeal to members of the medical profession; some form of profile analysis would be much more appropriate and predictive. These 'types' are of course correlated with the normative questionnaires already described (Grossarth-Maticek *et al.*, 1988). Correlations between 'types', cancer and coronary heart disease are very significant and in the predicted direction, even when smoking and other physical risk factors are partialled out (Eysenck, 1991a).

Figures 3, 4 and 5 show results from the original Yugoslav, and the two Heidelberg studies. The Yugoslav sample was 60 years-old on average, the two Heidelberg samples 10 years younger. One of the Heidelberg populations was randomly selected, the other was composed of persons judged to be 'stressed' by relatives and friends. Mortality is clearly greater in the older sample than in the younger, random sample, and greater in the 'stressed' than in the normal sample. Clearly in all samples cancer mortality is highest in Type 1, CHD mortality in Type 2 probands, with mortality quite low in Types 3 and 4, as expected. Prediction is clearly successful *both* for general mortality, as well as for the cancer-CHD difference, although it is clear that the latter differentiation is less successful.

These studies have been much criticized (see Eysenck, 1991b, for a target article describing the Grossarth-Maticek studies, and Eysenck's 1991c, replies to invited critics). Some of the criticisms are well taken, but do not impair the evidential value of the final results. But running through much of the criticism seems to be a belief that results are 'too good to be true'; this is not easy to understand because the results in Figs 3, 4 and 5 are less good than those reported in the follow-up of the Thomas studies, in spite of the fact that her data collection was not theoretically based, had far more restricted material to work on, and covered a much larger life-span. However, at my suggestion, C. R. Reynolds funded a thorough re-analysis of existing data, and a continuation of the Heidelberg study for

YUGOSLAV STUDY

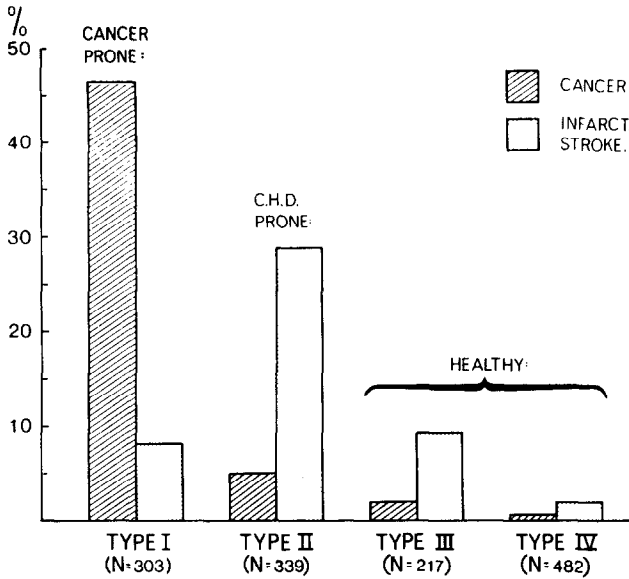


FIG. 3. Death from cancer and coronary heart disease for cancer-prone, CHD-prone, and 'healthy' subjects: Yugoslav study (Eysenck, 1991a).

HEIDELBERG STUDY  
(normal group)

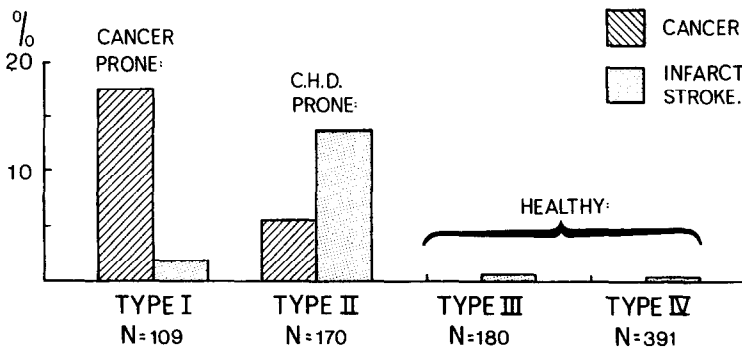


FIG. 4. Death from cancer and coronary heart disease for cancer-prone, CHD-prone, and 'healthy' subjects: Heidelberg study, normal group (Eysenck, 1991a).

### HEIDELBERG STUDY (stressed group)

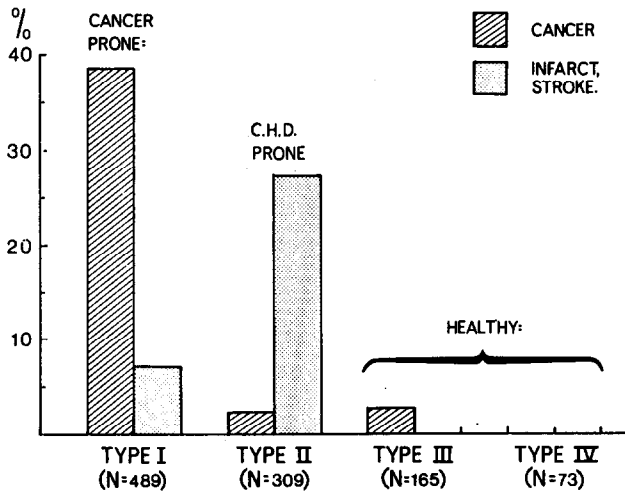


FIG. 5. Death from cancer and coronary heart disease for cancer-prone, CHD-prone, and 'healthy' subjects: Heidelberg study, stressed group (Eysenck, 1991a).

another 4½ years, under my supervision. (The re-analysis was supervised by C. Spielberger.) The detailed results of the extended follow-up have been published elsewhere (Eysenck, 1993) and are shown in Fig. 6; they show a continued significant effect along the same lines, and caused the main critic, who had available all the accumulated data, to withdraw his criticisms, after carrying out his own analysis. These data seem to be quite definitive.

There are further studies extending the list of types to 6, using a new inventory, adding 2 more to the original 4 types, and a different population (Grossarth-Maticek & Eysenck, 1990b). Results continue to be supportive of theory, but a detailed discussion would not be appropriate. Instead it may be useful to discuss quite briefly a number of independent replication studies, because nothing is more convincing than successful independent replication. Among the more interesting of these studies are those of Amelang and Schmidt-Rathjens (1992, 1993), Brengelmann, (1993), Ploeg, Kleijn, Mook, Hunge, Pieters and Leer (1989), Quander-Blaznik (1991), Ranchor, Sanderman and Bouma (1992), Sandin, Chorot, Jimenez and Santed (1993a, b), Schmitz (1992, 1993), Shigehisa (1991), Shigehisa, Fukui and Motoakis (1989, 1991), Shigehisa and Oda (1993),



HEIDELBERG STUDY  
(1982-1986)

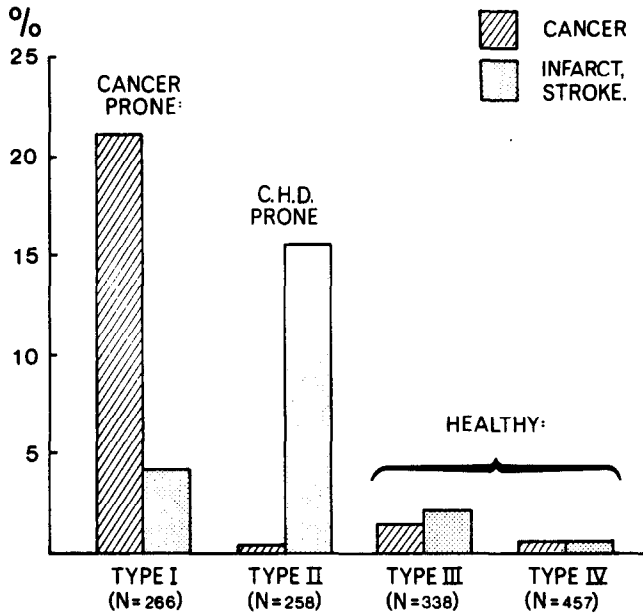


FIG. 6. Follow-up of the combined Heidelberg studies for another 4½ years (Eysenck, 1993).

Bleiker, Ploeg, Hendriks, Leer and Kleijn (1993), Spielberger (1993). Schmitz used the 6-type questionnaire (Grossarth-Maticek & Eysenck, 1990) and found high predictability for both cancer and CHD. He also found high correlations between the types and the three components of the Eysenck personality model (Eysenck & Eysenck, 1985). Types 1, 2 and 3 correlated positively with neuroticism, while type 4 correlated  $-0.57$  with N. The pattern was similar for extraversion, while psychoticism correlated positively with type 2, and negatively with type 5, as expected. Psychosomatic complaints correlated massively with types 1 and 2, but negatively with type 4. Many other correlations with personality and behaviour are given, including coping styles; as expected emotion-oriented, avoidance-oriented and distraction-oriented are characteristic of types 1 and 2, while type 4 is task-oriented. Practically all the reported findings support the Grossarth-Maticek typology. It should be noted that the sample was made up of people coming to Schmitz for autogenic

training, i.e. for psychological treatment, hence they would be expected to be more ready than most to overcome their reluctance to discuss their emotional hang-ups. (The results are given in tabular form in the Appendix, Table A.1.) A later study extends the sample used, and the tests given (Schmitz, 1993).

Quander-Blaznick (1991) "found a statistically significant multivariate association of low expression of anxiety and unfulfilled need for closeness with lung cancer" (p. 125). Amelang and Schmidt-Rathjens (1992) found a link between Types 1 and 2 and disease, but could not distinguish between cancer and CHD, probably because they did not use interview procedures (Grossarth-Maticek *et al.*, 1993). Ploeg *et al.* (1989) attempted to improve the psychometric qualities of the rationality-antiemotionality scale; their findings "tentatively support the view that rationality/antiemotionality may be an important distinctive personality characteristic in patients with cancer" (p. 217). Spielberger (1993) reported excellent psychometric properties for some of the Grossarth-Maticek scales, as well as predictive validity. Schmitz (1992) also reported good psychometric properties of the scales, in good agreement with the original analyses by Grossarth-Maticek and Eysenck (1990b). The results of the Sandin *et al.* (1993a, b) studies are shown in the Appendix, Table A.2. So far attempts at replication have been almost universally favourable to the conception underlying the Grossarth-Maticek types.

The latest replication study, as yet unpublished, is by Fernandez-Ballesteros. She compared 210 healthy college students, 90 healthy women, 90 women with benign growths, and 122 women with breast cancer, using two Grossarth-Maticek scales, rational-antiemotional and harmony-seeking, each consisting of 12 items. The cancer patients were very significantly differentiated from all the other, healthy groups in the predicted direction. Comparing the cancer patients with the benign tumour group, the differences on the two scales were two SDs, which is a very large effect size. In fact every single item in the two scales gave results in the predicted direction! This is an unusual decisive finding.

## SYNERGISTIC INTERACTION OF CANCER RISK FACTORS

The theory endorsed in this article considers that psychosocial factors like personality reactions to stress constitute an important risk factor for cancer, whether through initiation or (more probably) propagation. It has never been asserted (as critics sometimes pretend) that this is the *only* risk factor, or that psychosocial factors *cause* cancer. There are very many risk factors (genetic, smoking, drinking, unhealthy eating habits, radon gas inhalation, air pollution, etc.), and an important question that arises concerns the *mode of interaction* of these risk factors. I have already

mentioned that the results of the early Kissen–Eysenck work suggested a *synergistic* relationship between lung cancer and smoking, and this was borne out in the Eysenck (1988) paper comparing smokers and non-smokers with high or low scores on rationality–antiemotionality; the table given there shows that of those who never smoked only 1 died of cancer. Of those who smoked, but had low scores on R–E, none died of cancer. But of those who smoked *and* were high on R–E, 31 died of cancer (p. 459). Clearly, it is the *combination* of smoking and personality that is important. This point is amplified in the Grossarth-Maticek *et al.* (1988) paper.

A more detailed discussion of our own work is given by Eysenck, Grossarth-Maticek and Everitt (1991), and a more general discussion by Eysenck (in press). A good deal of evidence is cited in both studies to show that physical risk factors for cancer act synergistically rather than additively, and that small-scale early studies suggest that a similar mode of interaction can be observed when physical and psychosocial risk factors interact. Also noted are problems of statistical analysis which are serious, but cannot be discussed here for lack of space. I will here simply quote a few of the better and larger studies, trusting that the very obvious multiplicative effects will demonstrate the reality of the relationship.

Consider Table 4, which shows the lung cancer mortality of 2,374 healthy probands followed up over a ten-year period (Eysenck *et al.*, 1991). ‘No stress’ here means that the proband at the beginning of the experiment was of any other type than Type 1, the cancer-prone type; ‘stress’ means that he was of Type 1. Clearly, the use of the term ‘stress’ here is an abbreviation for “having a personality that reacts in a certain manner to stress, and is therefore cancer-prone”, but as a heading the term may be acceptable.

Table 4. Lung Cancer Mortality of 2,374 Probands Followed up over a Ten-year Period. Lung Cancer as a Function of Smoking and Stress (Eysenck, Grossarth-Maticek & Everitt, 1991)

	No stress	Stress	Stress effect
No smoking	0.35%	2.89%	(2.89% – 0.35%) = 2.54%
Smoking	0.80%	15.56%	
Smoking effect	(0.80% – 0.35%) = 0.45%		
N = 2374	Real combined effect:		(15.56% – 0.35%) = 15.21%
	Additive effect:		(0.45% + 2.54%) = 2.99%
	Difference (synergistic effect)		= 12.22%

The mortality entry in the cell 'No stress' – 'no smoking' (0.35%) is usually referred to as a 'background effect', i.e. the effect of all other risk factors. Stress in the 'no smoking' group, shows a mortality of 2.89%, thus the *stress effect* is  $2.89\% - 0.35\% = 2.54\%$ . The *smoking effect*, similarly, is  $0.80\% - 0.35\% = 0.45\%$ . On this basis one might say that the stress effect in this group was 5 times as large as the smoking effect. The sum of these two effects is  $0.45\% + 2.54\% = 2.99\%$ , but the true mortality in the smoking–stress cell is 15.56%, from which we must subtract the background effect of 0.35% to reach an effect size of 15.21%. Thus the synergistic effect, over and above the additive effect, is 12.22%, many times higher than the stress or the smoking effect, singly or together. This is the traditional method used by epidemiologists to look at interactive effects, and it shows a very powerful *synergistic* influence.

This study was replicated (Eysenck *et al.*, 1991), in order to see whether it might have been a statistical accident. Such replications are particularly necessary because of the statistical problems mentioned. Table 5 shows the results of this second study, using a different population, but otherwise identical in looking at lung cancer as a function of smoking and stress. Results are very similar. Here the smoking effect is actually negative, but again so small that this is not significant. The main finding is that again the synergistic effect is very much greater than the individual effects of stress and smoking.

Table 6 shows figures for mammary and other carcinomas in women. There are 179 women in each cell, with psychosocial stress being defined in terms of a questionnaire dealing with *rejection* by parents in childhood and/or by lovers in adult life. The other variable is made up of physical risk factors for mammary carcinoma, which are well known. The figures in each cell again show very clearly the effects of synergistic interactions

Table 5. Replication of Study in Table 3. Lung Cancer as a Function of Smoking and Stress

	No stress	Stress	Stress effect
No smoking	0.69%	2.09%	$(2.09\% - 0.69\%) = 1.40\%$
Smoking	0.24%	10.59%	
Smoking effect	$(0.24\% - 0.69\% = 0.45\%)$		
N = 1914	Real combined effect:		$(10.59\% - 0.69\%) = 9.90\%$
	Additive effect:		$(1.40\% + 0.45\%) = 0.95\%$
	Difference (synergistic effect)		<u><u><math>= 8.95\%</math></u></u>

Table 6. Interaction of Physical and Psychological Risk Factors Leading to Mammary Carcinoma

	N: No psychosocial	Psychosocial
No physical:	1 (0.5%) 7	3 ( 1.6%) 35
Physical:	2 (1.1%) 8	25 (13.9%) 36

(N = 179 in each cell)

Table 7. Interaction of Stress and Genetic Predisposition in the Causation of Mammary Carcinoma

	No stress		Stress	
	n: Mammary cancer	%	n: Mammary cancer	%
No genetic predisposition	306	1 ( 0.3)	238	1 ( 0.4)
One relative	208	3 ( 1.4)	141	6 ( 4.1)
Two relatives	70	3 ( 4.2)	68	6 ( 8.8)
Three relatives	28	3 (10.7)	29	11 (37.9)

for mammary carcinoma. Thus results for lung cancer are similar to those found in connection with mammary carcinoma.

A final study deals with the interaction between psychosocial stress, as defined above, and genetic predisposition as the physical stress. Genetic predisposition was defined in terms of the number of close relatives who had died of mammary cancer, and Table 7 shows the results. Cancer is a function of *both* genetic predisposition, which gives a straight dose-response curve, and stress. Again, there is a clear-cut synergistic effect.

Results such as these are very important (Eysenck, 1991a), they demonstrate the extreme difficulty of using epidemiological evidence in this field. Univariate analyses are quite unequal to the demands of any rational model of risk factor interaction, yet most analyses of the effects of smoking have relied on univariate analyses, and used these to extrapolate to population estimates concerning the number of deaths 'caused' by smoking. Such extrapolations are totally inadmissible in the presence of large numbers of risk factors interacting synergistically. Smoking seems to have little effect in the absence of personality/stress, or other risk factors, and cannot therefore be said to 'cause' cancer or CHD. The term 'cause' has a very definite meaning in science, and is clearly inappropriate here (Eysenck, 1991a). To say this is not to deny that smoking is an

important risk factor in cancer, and particularly lung cancer; it assumes particular importance in conjunction with other risk factors, particularly psychosocial ones.

### THERAPEUTIC INTERVENTION STUDIES

Is it possible to interpret the studies so far discussed in a *causal* manner? They would seem to establish a *correlational* relation between the cancer-prone personality and death from cancer, but that connection could be via causally effective intermediaries (even if that is not a likely possibility). More convincing would be an *intervention*, perhaps along therapeutic lines, which would lead to measurable *changes* in personality, and a *reduction* in the cancer mortality of cancer-prone individuals. Alternatively, such intervention might be used to prolong life expectancy of people with inoperable cancers. I shall discuss the latter type of research first.

There are many early studies, not very rigorously controlled, that have given a positive answer. Le Shan (1977), Simonton, Matthew-Simonton and Creighton (1978), Achterberg, Lawlis, Simonton and Simonton (1977) are typical of this group. In the Simonton studies, for instance, 159 'incurable' cancer patients were treated by psychological methods. Two years later, 63 were alive, 22% had "no evidence of disease", and 19% had tumours that were shrinking.

Particularly impressive are some more rigorous recent studies. Grossarth-Maticek (1980) reports on 24 pairs of terminally-ill cancer patients matched for type of cancer, and then allocated randomly to treatment or control, using his own autonomy-training method of psychological treatment (Grossarth-Maticek & Eysenck, 1991b). Mean survival time was 3.09 years for the control group, 5.07 years for the therapy group. Spiegel, Bloom, Kaemer and Gottheil (1989) reported in a study of terminally-ill women that those receiving psychological treatment survived about twice as long as those who did not. Women with terminal cancer of the breast were also subjects of another study (Eysenck & Grossarth-Maticek, 1991) in which half had agreed to chemotherapy, half had refused; half of each group received autonomy training, half did not. Both chemotherapy and behaviour therapy did significantly better than no therapy in prolonging life; both together did specifically better than the *sum* of their individual effects, i.e. there was a synergistic effect. These various studies definitely suggest that psychological intervention does have a significant effect on survival.

Levy and her colleagues (Levy *et al.*, 1985) have produced evidence that psychological interventions can increase cancer patients' optimism and reduce feelings of hopelessness; in addition an increase in the number of natural killer cells was reported; these are an important part of the

immune defence system against cancer (Seligman, 1991). Similarly, Fawzy and his colleagues (1990, 1993) have reported that melanoma patients who received group treatment improved psychologically and also had higher amounts of natural killer cells.

In this study 68 patients with malignant melanoma were divided into 2 groups of 34 each, the experimental group receiving a 6-week structured psychiatric group intervention, while the control group received only the routine medical care also received by the experimental group. For the entire sample of 68 patients, a measure of distress taken at baseline was found to be a *positive* factor for survival — this is in good agreement with the ‘fighting spirit’ hypothesis, and the relation between suppression and emotion and cancer. Premorbid coping behaviour and the effectiveness of coping in dealing with life-threatening situations were also found to be critical factors. Patients with active-behavioural coping abilities, whether or not they participated in the intervention, had the best health outcomes. “Those who minimize the importance and threat of cancer to their well-being appears to be the greatest risk” (Fawzey *et al.*, 1993, p. 687). As regards intervention, 3 of the treatment patients died, but 10 of the controls did. Seven of the former, but 13 of the latter suffered recurrence. These results of a 5-year follow-up are in good agreement with the other studies mentioned.

As far as prophylactic use of psychological therapy is concerned, we have the important work of Grossarth-Maticek. The treatment itself and its rationale are described in a paper by Grossarth-Maticek and Eysenck, (1991b), and a summary of the results is given in a companion paper by Eysenck and Grossarth-Maticek (1991). In the first of their studies 100 Type 1 healthy probands were randomly assigned to a therapy or a control group; they were then followed up (Grossarth-Maticek, Schmidt, Vetter & Arndt, 1984), and mortality and cause of death noted. After 13 years, 16 members of the control group had died of cancer, none of the therapy group. Cancer incidence was 21 in the control group, 13 in the therapy group. Similar results are reported when group therapy was used, and even when bibliotherapy plus 3 hours of individual therapy was employed (Eysenck & Grossarth-Maticek, 1991). These results are very promising, and they indicate clearly the causal nature of the personality cancer connection. They are in urgent need of replication because it is very difficult to sort out the influence of the method of treatment, and the influence of the therapist; in these studies Grossarth-Maticek, who is a charismatic type of personality, carried out the major part of the treatment. Other successful therapists (e.g. Le Shan; Simonton; Spiegel) used rather different methods, and it is obviously important to know to what extent different methods may combine identical effective elements — a universal problem in psychotherapy (Giles, 1993). However,

this question is irrelevant to the clear demonstration of psychological effectiveness on cancer-prevention and prolongation of life.

### HOW PERSONALITY CAN INFLUENCE CANCER

Psychological therapies are often considered 'placebo' treatments by orthodox physicians, but the term 'placebo' hides many conceptual problems and difficulties (Gruenbaum, 1993). The power of such placebo effects is undoubted (e.g. Beecher, 1955; Benson & Epstein, 1975; Benson & McCullie, 1979; Shapiro & Morris, 1978), with over 1,000 articles and books attesting to its influence (Turner, Gallimore & Fox-Henning, 1980; White *et al.*, 1985). Roberts (1983) has estimated that so-called placebos produce one-third excellent results, one-third good results, one-third poor results in *physical illness* under conditions of heightened expectations, i.e. when both physician and patient believe in the value of the treatment, and Roberts *et al.* (1993) have offered support for this estimate in an important study.

Five medical and surgical treatments, once considered to be efficacious by their proponents, but no longer considered effective based upon later controlled trials, were selected according to strict inclusive criteria. A search of the English literature was conducted for all studies published for each treatment area. The results of these studies were categorized, where possible, into excellent, good, and poor outcomes. For these five treatments combined, 40% excellent, 30% good, and 30% poor results were reported by proponents. It was concluded that the *power of non-specific effects far exceeds that commonly reported in the literature.*

Obviously the alleged placebo effects must use *biological* pathways to influence physical outcome, and hence must be 'specific' to that extent; perhaps 'unintended' rather than 'non-specific' would be the better description to use. But when psychological treatment is targeted on certain types of behaviour, such as emotional expression, which are considered carcinogenic, or at least carcinosupportive, they are both targeted and specific. What is important at that stage is the discovery of the biological pathways indicating the physical effects of psychological factors.

The possibility that personality can influence the development of cancerous growth seems counter-intuitive to many people still under the spell of Cartesian dualism. There is now sufficient evidence to demonstrate the existence of causal connections between personality and stress, on the one hand, and cancer on the other, through the intermediary of the immune system. The general theory linking the two has been discussed in some detail by Eysenck (1991a), together with a review of the evidence. We may begin with a statement of Solomon's (1987) postulates:



- (1) Enduring coping style and personality factors (trait characteristics) should influence the susceptibility of an individual's immune system to alteration by exogenous events, including reactions to events. (Thus, an 'immunosuppression-prone' behavioural pattern is hypothesized.)
- (2) Emotional upset and distress (state characteristics) should alter the incidence, severity, and/or course of diseases that are immunologically resisted (infections and neoplastic) or are associated with aberrant immunologic function (allergic and autoimmune).
- (3) Severe emotional disturbance and mental dysfunction should be accompanied by immunologic abnormalities.
- (4) Experimental behavioural manipulation (e.g. stress, conditioning) should have immunologic consequences.
- (5) Experimental manipulation of appropriate parts of the central nervous system (CNS) should have immunologic consequences.
- (6) Hormones and other substances regulated or elaborated by the CNS should influence immune mechanisms.
- (7) Biochemical and functional similarities might be expected between the substances modulating the function and reactivity of the CNS (neuropeptides) and the substances with comparable effects on the immune system (cytokines).
- (8) Behavioural interventions (such as psychotherapy, relaxation techniques, imagery, biofeedback, and hypnosis) should be able to enhance or optimize immune function.
- (9) Altered CNS neurotransmitter receptor-site sensitivities believed to be associated with mental illnesses should be reflected in lymphocyte receptors.
- (10) The 'functional' modes of expression of CNS and immune system should be similar.

Research since has in large measure replicated and extended the studies reviewed by Solomon and Eysenck; there is far too much material to give anything but a very brief review of it here. The most widely studied intermediary between *stress response* and *immunodepression* has been cortisol, but other intermediaries (ACTH; endogenous opiates) should also be taken into account. Vickers (1988) showed that for those individuals who demonstrate affective disruptions and low defensive reserve, there is a high correlation between repressive behaviour, personality and plasma cortisol secretion rate following a stressful event. It is of course well known that cortisol impairs several components of cell-mediated immunity (Gorman & Kertzner, 1991; Cupps & Fauci, 1982). Vickers provided cumulative evidence from five studies that over 16% of the variance in cortisol level could be predicted from (often

sub-optimal) emotionality scores. Thus the theory would suggest a causal pathway as follows:

Stress-strain (ineffectual personality response to stress) — cortisol secretion — immunodepression — cancer growth — death. Recent reviews document each of these links; good summaries are the following: O'Leary (1990), Stein (1989), Weisse (1992), Herbert and Cohen (1993), Antoni (1987), Zakowski, Hall and Baum (1992), Kiecolt-Glaser and Glaser (1992), Gorman and Kertzner (1991), Locke (1986), Pletnikoff, Faith, Murgo and Good (1986), and Kennedy, Kiecolt-Glaser and Glaser (1988). Of the more interesting recent studies not reviewed in these summaries, one may be worthy of special mention. Wiedefeld *et al.* (1990) were successful in showing that the development of strong perceived self-efficacy to control phobic stressors, in an intervention design, had an immunoenhancing effect.

Human reactions are of course of main interest, but animal work (e.g. Borysenko & Borysenko, 1982; Metzler, 1979; Metzler & Nitch, 1986) also supplies convincing evidence that carcinogenesis is intimately connected with events in the central nervous system. The demonstration by Ader and Cohen (1975) that immunosuppressors could be conditioned along Pavlovian lines points to the same conclusion. It would be difficult nowadays to deny the existence of a strong link between personality, reaction to stress, immunological reaction, and cancer, or of the possibility of psychological intervention altering the various parts of this system in the direction of increasing immunological efficacy.

This conclusion is particularly important in the context of this section. However compelling the studies linking personality with cancer, doubts must always remain as long as there is no evidence of a possible *causal link* between the two. To find that there is indeed good evidence for a strong link, susceptible to stress and also to intervention, makes the general argument much more compelling. There are of course other intermediaries, some of which are of interest in possibly mediating the opposition between cancer and CHD. Plasma cholesterol concentration is one example. There is a definite *positive* correlation between cholesterol and CHD, and an almost equally strong *negative* correlation between cholesterol and cancer (Isles *et al.*, 1989). Unfortunately little is known about the correlation between personality and cholesterol, but this should certainly be the subject of a determined research effort, testing the obvious suggestion of a *positive* correlation with Type A and a *negative* one with Type C.

What does seem to be established is that cholesterol performs a role in the genesis of CHD similar to that of cortisol in relation to cancer, and equally subject to psychosocial factors (Rosenman, 1993). Within-person levels show a 10–20% weekly variability under standardized conditions

(Mogadam, Ahmed, Marsh & Godwin, 1991), and the sympathetic nervous system appears to be involved (Dzan & Sacks, 1987; Howes, Krum & Louis, 1987). During emotional stress, increased levels of cholesterol have been found to occur (Friedman, Rosenman & Carroll, 1958; Hammarsten *et al.*, 1957). These early studies were soon confirmed, e.g. Dreyfuss and Czaczkes (1959), Grundy and Griffin (1959), Thomas and Murphy (1958), Wertlake, Wilcox, Haley and Paterson (1958), and Francis (1979). These studies of the effects of minor life stresses (e.g. examinations) produced 10–25% increases of total and LDL cholesterol, so did other life events of a stressful nature (e.g. Wolf *et al.*, 1962; Catley *et al.*, 1962; Groen *et al.*, 1962; Rahe, Ryman & Biarsner, 1976). Rosenman (1993) gives an extended discussion and literature survey which suggests strongly that plasma cholesterol may have an important intermediary role to play among the psychological factors affecting coronary heart disease.

#### FAILURE TO REPLICATE

Critics (e.g. Fox, 1978, 1981, 1983, 1989; Fox & Temoshok, 1988) have drawn attention to the fact that positive findings are often counterbalanced by negative findings; such failure to replicate is particularly obvious in relation to depression as a precursor of cancer. I have already mentioned the work of Shekelle *et al.* (1981) and Persky *et al.* (1987). Kaplan and Reynolds (1988), also already mentioned, obtained insignificant results. So did studies by Hahn and Petitti (1988), and Linkins and Comstock (1988). Finally, Zonderman, Costa and McCrae (1989) failed to find a connection between depression and cancer, while Allgulander and Lavori (1991) failed to find excessive mortality among anxious neurotics. Similarly, several studies failed to discover a link between neuroticism and cancer (e.g. Keehn, Goldberg & Beebe, 1974; Coryell, 1981), results which led Angell (1985) to say that: “belief in disease as a direct reflection of mental state is largely folklore” (p. 1572). What is the explanation of these contradictions?

The first explanation must be that many studies in this field are done by epidemiologists and oncologists without any training in psychology, with only the most rudimentary ideas of the theories underlying work in this area. 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. To simply administer routine questionnaires to thousands of subjects and expect to capture a very complex psychological phenomenon is not a reasonable proposition. If anything we would expect a *negative* correlation, a *denial* of real anxiety, anger, and perhaps also depression, but motivation being

completely uncontrolled in these studies no psychologist familiar with the field would make any confident prediction. These studies are of little interest, and irrelevant to the theory which has been elaborated and found successful over the years. If at least these studies had added a Lie Scale to the depression–emotion scales, something might have been saved from the wreck, but not one of these allegedly negative studies shows any awareness of the need for such a scale. Psychological studies need to be directed by psychologists, knowledgeable in this field and aware of the theories involved, and using the most appropriate measuring instruments; nothing else will do.

In the second place there is good evidence, already reviewed, that investigations carried out by interviewers, i.e. involving personal contact, the establishment of trust, and the possibility of explaining doubtful points in the questionnaire to the proband, give very significantly better results than studies relying solely on questionnaires dished out anonymously and without detailed explanation and motivation (Grossarth-Maticek *et al.*, 1993). Epidemiological studies are usually done on large numbers and with a disturbing disregard of the needs, wishes and problems of the participants; this is not likely to result in psychologically meaningful results, although it is undoubtedly cheaper than doing the job in a professional manner.

In the third place, researchers often neglect previous findings which would indicate the desirability of making finer distinctions between different aspects of concepts which are very complex in nature like 'stress'. What is important is not 'stress' as viewed from outside, but 'strain' as experienced by the individual. There is also the distinction between controllable and uncontrollable stress, only the latter being very relevant to cancer. Finally, there is the important distinction between chronic and acute stress, giving rise to the concept of an 'inoculation' effect under chronic stress conditions, reversing the deleterious effects of acute stress. When these distinctions are not made, different experiments simply looking at 'stress', and disregarding differences such as those mentioned may easily reach opposite conclusions. Worthwhile research must pay attention to distinctions of this kind (Eysenck, 1981).

Finally, we must ask: When is a replication not a replication? Often what are suggested to be replications are in fact so different from the original study that it is not possible to regard them as 'replications'. There are usually differences in measuring instruments, method of application, sex and age structure of sample, duration of follow-up, type of cancer and stage of development, as well as factors difficult to quantify, but important nonetheless, such as motivation, attitude to research purpose, instructions, etc. The very theories investigated are often different, as already explained in connection with 'depression'. The very terms used

are often multidimensional; 'depression' is like a fever, a symptom that may result from, and be correlated with many different causal factors. Theory links one particular type of depression with cancer; it does *not* predict any connection with other types of depression (Eysenck, 1991a).

This is not to say that there are no significant negative studies — there are. That by Cassileth *et al.* (1985) is perhaps the best known. The study is well done, but here too there is complete neglect of the theory linking *suppression* of emotion with cancer; furthermore, patients were suffering from advanced, high-risk malignant diseases. Perhaps at this advanced stage of cancer personality factors take a back seat; to assess how reasonable such an explanation may be would require a separate investigation. No single study can confirm or disconfirm such wide-ranging hypotheses as those discussed in this chapter, we have to rely on a consideration of all the available evidence, and on that score there seems little doubt that personality, as Osler (1906) already predicted, plays an important role, in interaction with other risk factors, in the development of cancer.

### SUMMARY AND CONCLUSIONS

The evidence surveyed suggests a number of conclusions:

- (1) Personality factors, concerned mainly with reaction to stress and coping mechanisms, play a powerful part in longevity (autonomous; self-regulating; hardy).
- (2) Specific personality traits play a part in predisposing certain people (cancer-prone, Type C) to cancer.
- (3) Specific personality traits, differing in many ways from those characteristic of the cancer-prone personality, predispose certain people (CHD-prone; Type A) to coronary heart disease.
- (4) Personality traits characteristic of the cancer-prone personality serve to shorten the life-span of people already suffering from cancer.
- (5) Prophylactic psychological therapy can cause cancer-prone people to avoid developing carcinomas, at least for a time.
- (6) Psychological therapy can help people suffering from inoperable cancer to live longer than controls.
- (7) There is much experimental support for a theory linking personality factors with immuno-suppressive agents, like cortisol, and through immunosuppression with cancer.
- (8) It has been shown that cortisol level and the state of the immune system can be improved by means of psychological treatment.
- (9) Physical risk factors for cancer have been found to act *synergistically*, not additively.

- (10) Psychosocial and physical treatments have been found to act *synergistically*, not additively.
- (11) Measurements of the cancer-prone personality are only relevant if they are geared to testing the major theories developed by the leading experts in the field.
- (12) Measurement using personal contact (interviewing methods) is significantly more likely to give positive results than simply handing out questionnaires.

In all, the evidence *supports* the underlying theories of a cancer–personality link, although of course much remains to be discovered concerning the specific nature of that link.

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