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Personality, stress and cancer: Prediction and prophylaxis

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This paper reports results from three prospective studies, in which probands were followed over periods of 10 years, before inquiring about death and cause of death. Personality inventories were administered at the beginning of the 10 year period, as were questions concerning smoking, drinking, medical diseases, etc. It was found that personality variables were much more predictive of death from cancer or cardiovascular disease than was smoking, and that different personality types were susceptible to either of these two diseases. Personality type was defined in terms of differential ways of dealing with interpersonal stress, and it was found that stress was a very potent cause of death, in the sense that stressed probands had a 40 per cent higher death rate than non-stressed probands.

It has long been suspected that stress exerts some kind of causal role as far as the genesis of cancer is concerned, but the evidence has often been unsatisfactory (Cooper, C. L., 1983, 1984; Dobson, 1982; Kaplan, 1983). Stress has also been related to cardiovascular disease (Price, 1982; Steptoe, 1981), and aging (Cooper, E. L. 1984). The reasons why much of the work reported in these books must be regarded as unsatisfactory are, first of all, that many of the studies were retrospective. It is doubtful whether memory of past events can be uncontaminated by present-day illness, and vague hypotheses in the minds of those afflicted, and in any case memories of past events are notoriously unreliable, particularly when these are of a strongly emotional nature. Only prospective studies can give truly acceptable evidence of the importance of stress.

The second reason, as Eysenck (1975) has pointed out, is the confusion that exists in much of the literature between the concepts of 'stress' and 'strain'. In physics, a clear distinction is made between these two concepts, as for instance in Hooke's law of elasticity: stress $= k \times$ strain, where k is a constant (the modulus of elasticity) that depends upon the nature of the material and the type of stress used to produce the strain. This constant k, i.e. the stress/strain ratio, is called Young's modulus. In other words, stress is what is imposed upon the material in question by the outer world; strain is the reaction of that material to the stress. To translate this into psychological terms, we might say that the loss of a wife is a *stress*; the psychological, hormonal, physiological and other consequences are the strain.

Why is it important to discriminate between stress and strain? The answer, of course, lies in the fact that what may in the outer world appear identical stresses may set up quite different strains, depending on the individual. The loss of a wife may be

a devastating blow to a young husband who loves his wife, and is inconsolable about her loss. It may be a relief to the husband of a wife who has been ill with a painful malady for many years, and who regards her death as a deliverance from pain. It may be a joyous occasion for a philandering husband who inherits his wife's money, and is now free to spend it on his girlfriends. Questionnaires which only look at *events*, without looking at an individual's reactions, can produce data which are quite valueless because they leave out of account these important factors.

The usual way of discussing stress in the psychiatric literature also leaves out of account the importance of personality. Physical pain imposes less of a strain on extraverts than on introverts, while sensory discrimination imposes less of a strain on introverts than on extraverts (Eysenck, 1967; Eysenck & Eysenck, 1985). Many types of stimuli, experiences and encounters impose more of a strain on emotionally unstable than emotionally stable people. These differences are the equivalent of the term k in Hooke's formula, and they cannot be omitted from any scientific study of the influence of stress on bodily disease.

Schroeder & Costa (1984) have given voice to such doubts in a paper critical of the traditional literature. As they say:

> There is good reason to suspect that the link between environmental stress and illness has been exaggerated in both the public mind and the psychological literature. Individuals are eager to find explanations for events that occurred to them, including reasons they experience . . . and they may seize upon the stress hypothesis to account for what would otherwise have to be considered ill fortune. Unfortunately, many researchers have also come to suppose that illness is closely linked to life-stress, encouraged by retrospective self-report studies, which have often shown sizeable associations between recalled stress and recalled illness. But it could be argued that memory, perception and response tendencies figure so prominently in these studies that these factors alone could account for the findings [p. 833].

Maddi, Bartone & Puccetti (1987) have put forward a reasonable reply to the Schroeder & Costa argument, but there is no doubt that many of the published studies are subject to the criticisms they have voiced, and that reliance should only be placed on prospective studies showing evidence that they recognize the distinction between stress and strain, as well as the importance of personality variables. The most satisfactory evidence has been produced with respect to the influence of psychological predictors on heart disease (Booth-Kewley & Friedman, 1987; Chesney & Rosenman, 1985); evidence as far as cancer is concerned is less impressive. In this paper I shall be concerned with a series of three prospective studies carried out in Yugoslavia and Heidelberg (West Germany) respectively which give strong evidence for the validity of the general theory with respect to cancer also.

The studies to be discussed in some detail have been described from various points of view by Eysenck (1987), Grossarth-Maticek and his colleagues, references to whose work will be found in the bibliography, Kanazir, Djordjevic-Markovic & Grossarth-Maticek (1984) and Schmidt (1984). Most of these are relatively short communications, but those by Eysenck (1987), Grossarth-Maticek, Eysenck & Vetter (1987) and Schmidt (1984) are on a rather larger and more detailed scale. Evidence concerning the main hypotheses linking personality with cancer has been reviewed by Eysenck (1985); this extends the hypothesis of a 'disease-prone personality' (Friedman & Booth-Kewley, 1987) from the five diseases reviewed by them to

cancer. A survey by Temoshok (in press), which arrives at conclusions similar to those reported by Eysenck, is also of interest in this connection. The two major personality traits which appear to characterize the cancer-prone personality are (1) a helpless/hopeless reaction to stress, involving a failure to cope with a stressful situation, and (2) a rational, non-emotional, repressed reaction to life-events which would normally produce strong emotions, whether of fear or anger.

The three prospective studies to be discussed here were all initiated and organized by Dr Ronald Grossarth-Maticek, who graduated and carried out his first study in Yugoslavia, but then went to Heidelberg to carry out the other two studies. All are 10-year follow-up investigations, in which personality variables, smoking habits, drinking habits, medical variables, etc., were ascertained at the beginning of the study, and death and cause of death after 10 years. The two Heidelberg studies have been extended for a further three-year follow-up, but only some of the results are available at present. The first study to be described is that carried out in Yugoslavia, on a sample consisting of 1353 subjects. These were recruited by selecting the oldest person in every second household in a small Yugoslav town with a population of 14 000 people; most of the subjects were between 59 and 65 years old. In a number of cases, additional subjects were included who had been nominated as suffering from severe psychological stress. Separate analyses of these samples suggest that the inclusion of this additional group makes the results less impressive than they would otherwise have been, but in order not to exclude any subjects on what might seem an arbitrary basis the data here given are based on the total sample. Psychosocial data were recorded using a questionnaire and an observational catalogue, and employing an interview procedure. Height, weight and blood pressure, and data on cigarette smoking and drinking were also collected, and further medical information was recorded periodically. Ten years after starting the study, a physician assessed the occurrences of different diseases in the sample and also recorded diagnosis on the death certificate. As death certificate data are notoriously unreliable, Dr M. Jankovic undertook to obtain a more correct diagnosis of cause of death by an extensive procedure of looking at the medical histories of the deceased subjects, talking to relatives and doctors, and generally attempting to obtain the best possible assessment of the cause of death.

In those who died of cancer, cancer of the lung, rectum and prostate predominated amongst males, but breast, uterine and cervical cancer occurred in 69 per cent of the females.

The attempts Grossarth-Maticek and his colleagues made of linking personality, stress and cancer made use of two rather different methods, both of which were used on the three samples. In the first of these, a questionnaire containing 109 questions concerning several personality traits was used, each trait measurement being made up of a number of separate questions. Thus one group of questions related to adverse life-events or situations leading to long-lasting hopelessness/helplessness. Another group of questions related to adverse life-events or situations leading to anger and/or hostility (this set of questions was hypothesized to be related more closely to coronary heart disease, as later shown by Chesney & Rosenman, 1985, and Booth-Kewley & Friedman, 1987, than cancer; indeed, as Eysenck (1985) has pointed out, the personality correlates of cancer are in many ways opposite to those characteristic of coronary heart disease).

A third set of questions related to rationality and anti-emotionality, i.e. the obverse of neuroticism–anxiety. Other questionnaires referred to the need for harmonious interpersonal relationships; ignoring signs of illness; lack of positive emotional relations; absence of self-report of psychopathological symptoms, especially anxiety; and finally acquiescence.

Table 1 shows the number dying of lung cancer or other types of cancer having respectively a low score and a high score on the R–A (rationality–anti-emotionality) scale. According to the theory, those with a high score would be expected to die more frequently of cancer than those with the low score, and the results show, at a very high level of statistical significance, that this expectation is indeed borne out. Both lung cancer and other cancers tend to have high scores (10 or 11 on a 11-point scale), while low scorers (between 0 and 9 points) seem to be protected against any type of cancer.

Table 1. Cancer and scores on the rational–anti-emotional scale of Grossarth-Maticek (Eysenck, 1987)

	Low score		High score	
	Obs.	Exp.	Obs.	Exp.
Lung cancer	0	26	38	12
Other cancer	8	84	120	44

Schmidt (1984) has carried out large-scale factor analytic and correlational studies of the results of the Yugoslav investigation, and found that the factors resulting from his analyses coincided to a large extent with the *a priori* scales constructed by Grossarth-Maticek, most of which had reasonably high reliabilities ranging from 0.79 to 0.95. The correlation between the scale for hopelessness/helplessness and cancer was 0.59 (*eta* = 0.60). The correlation between cancer and rationality–anti-emotionality was 0.51 (*eta* = 0.60). These two correlations thus confirm very clearly the general hypothesis of the cancer-prone personality, as outlined above, and lend weight to the largely retrospective studies which have found evidence in favour of it.

A third factor, entitled 'harmonization', i.e. a tendency to shun quarrels and try to bring about harmony among and with people split on some issue, also correlated quite highly with the occurrence of cancer, namely to the extent of 0.49 (*eta* = 0.57). Hypochondriasis, as expected, showed a *negative* correlation with cancer, to the extent of −0.39 (*eta* = −0.41). Obviously the various scales mentioned, as well as the others used, are not independent, and it is of some interest to discover to what extent they can be used jointly to arrive at a multiple correlation.

Grossarth-Maticek, Kanazir, Schmidt & Vetter (1982*a*, p. 297) have constructed a path model with cancer as the dependent variable and seven psychosocial scales as the independent variables. The results, in the form of standardized partial regression coefficients are given in Fig. 1, where it will be seen that the hopeless and rational

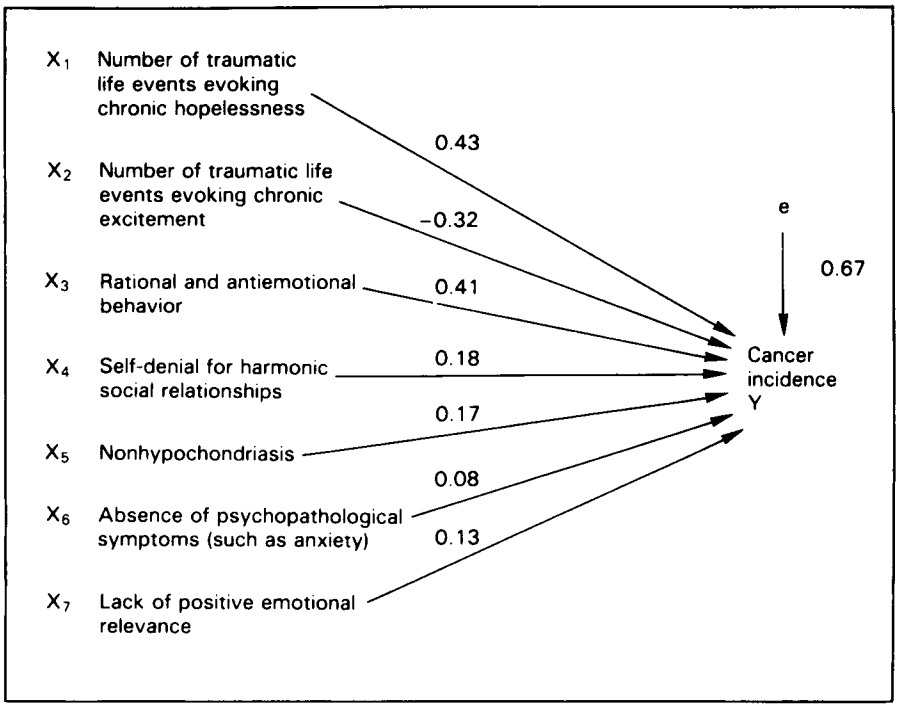

Figure 1. Standardized partial regression coefficients of path moded linking personality traits and cancer (Grossarth-Maticek *et al.*, 1982).

behaviour variables retain their strong positive relationship with cancer incidence, while chronic excitement has the expected negative correlation. The explained variance (R^2) for these seven variables is 0.55 with an error term denoting the unexplained variance of 0.45; this is indicated in Fig. 1 as e ($e = R$). Actually the R^2 for the first three predictors is equal to 0.49, so that there is little gain by including the other variables.

Schmidt (1984) has pointed out some weaknesses in the statistical treatment used, particularly the assumption that the dependent variable is dichotomous. Using more appropriate statistics, Schmidt found throughout higher correlations with cancer incidence, suggesting that something like 60 per cent of the total variance for cancer incidence could be accounted for in terms of the personality variables chosen.

In these data, sex and age have been controlled for, but not smoking. In view of the controversy regarding the causal role of smoking in cancer, particularly lung cancer, this topic demands a separate analysis (Eysenck, 1980, 1986). The multiple discriminant analysis described above gives values of 0.24 for smoking with respect to lung cancer (Grossarth-Maticek, Kanazir, Schmidt & Vetter, 1982*b*). This information, while suggesting that smoking is considerably less important as a predictor of cancer than is personality, is quite insufficient to tell us much about the complex relationships between these variables. A more detailed analysis is given by Grossarth-Maticek (1980), where comparison was made between the most prominent

Table 2. Interaction between physical and psychosocial risk factors (Grossarth-Maticek, 1980)

Dependent variable	Physical risk factor	Psychosocial risk factor	Significance of interaction term (%)	Estimated stand, regression coefficient of disease on physical risk factor for psychosocial risk factor	
				Absent	Present
Apoplexy	Systolic blood pressure	Rationality and anti-emotionality	< 1	0.061	0.239
Infarct	Systolic blood pressure	Rationality and anti-emotionality	< 5	0.025	0.150
Infarct	Smoking	Rationality and anti-emotionality	< 1	0.005	0.170
Lung cancer	Smoking	Rationality and anti-emotionality	< 1	0.046	0.384
Lung cancer	Smoking	Life events that caused lasting depression and hopelessness	< 1	0.081	0.514
Average	—	—	—	0.044	0.291
Factor of increase		—		6.6	—

Table 3. Multiple correlations (corrected for bias) between disease and physical and psychosocial risk factors (Grossarth-Maticek *et al.*, 1983).

Disease	Physical predictors	Psychosocial predictors	Both
Infarct	0.20	0.36	0.40
Apoplexy	0.23	0.32	0.35
Lung cancer	0.27	0.36	0.42

psychosocial risk factors, and the most prominent medical risk factors. The results are given in Table 2. In all the analyses conducted, interaction was significant and worked in the same way; i.e. psychosocial variables were not only important predictors of cancer incidence in themselves, but they also decisively modified the efficacy of physical risk factors. Quite consistently, the efficacy of the physical risk factors depended decisively on the presence of some social risk factor constellation. Without this, they were, on the average, less than 1/6 as important, the factor of increase actually turning up as 6.6!

Another type of analysis brings out very much the same point (Grossarth-Maticek, Kanazir, Vetter & Jankovic, 1983). Table 3 shows the result, using this time only two psychosocial variables. Results again point out the importance of the personality variables, and their interaction with smoking.

More detailed still is Table 4 (Grossarth-Maticek, Bastiaans & Kanazir, 1985), which illustrates the synergistic relationship of smoking and rational–anti-emotional behaviour with lung cancer mortality. It shows that for the males in this study lung cancer risk was minimal except for those who *both* smoked heavily *and* had a high R/A score. There were too few women dying of lung cancer to make an analysis possible for them also.

The table shows clearly that the great majority of those who died of lung cancer had both high scores on the R/A scale and smoked more than 21 cigarettes per day. It would thus appear that smoking shows a statistical relationship with lung cancer *only* in people having the appropriate cancer-prone type of personality. There is practically no correlation between smoking and lung cancer in the 654 males with scores of 9 or less on the R/A scale, but quite a strong correlation in the 310 males scoring 10 or 11 on the R/A scale.

Table 4. Lung cancer incidence (deaths/number at risk) by smoking and rationality–anti-emotionality in males (Grossarth-Maticek *et al.*, 1985)

Rationality and anti-emotionality	Smoking habits			
	Never smoked	1–20 cigs/day	21+ cigs/day	Total
0	0/77	0/42	0/38	0/157
1–9	0/214	0/142	0/141	0/497
10–11	1/117	0/54	31/139	32/310
Total	1/408	0/238	31/318	32/964

The treatment of the psychosocial personality variables so far has been along the traditional lines of trait psychology. Grossarth-Maticek also attempted a more typological approach, in which subjects were classified into one of four personality 'types', namely a cancer-prone type (Type 1), a coronary heart disease-prone type (Type 2), an intermediate type (Type 3), and a healthy type (Type 4). Description of these types was essentially based on their reactions to stress, and the results are therefore particularly relevant to the theory that stress (or rather strain!) is relevant to cancer incidence. According to this theory, it is the occurrence of the stress, and the particular reaction of the different types to this stress, which are important for cancer. A detailed description of the types is given by Grossarth-Maticek, Eysenck & Vetter (1987), from which the following descriptions are taken. In addition to these brief descriptions, the paper contains typical case histories, as well as the questionnaires on which allocation of a person to one of the four types was based.

Type 1: Understimulation

Persons of this type show a permanent tendency to regard an emotionally highly valued object as the most important condition for their own well-being and happiness. The stress produced by the continued withdrawal or absence of this object is experienced as an emotionally traumatic event. Type 1 individuals fail to distance themselves from the object and remain dependent upon it. Thus individuals of this type do not achieve success in reaching the object, and remain distant and isolated from this highly valued and emotionally important object. Great stress is produced by this failure to achieve nearness to the highly valued person, success in the highly valued occupation, or whatever. This type shows a lack of ***autonomy***.

Type 2: Overarousal

Persons of this type show a continued tendency to regard an emotionally highly important object as the most important cause for their particular distress and unhappiness. Rejection by the object (if a person), or failure to reach it (as in the case of occupational success) is experienced as an emotional trauma, but persons of this type fail to achieve disengagement from the object; rather, they feel more and more helplessly dependent on the object. Thus persons of this type remain in constant contact with these negatively valued and emotionally disturbing people and situations, and fail to distance themselves and free themselves from dependence on the disturbing object. Where persons of Type 1 keep on seeking nearness to the object of their desires, and experience their failure in terms of hopelessness and helplessness, persons of Type 2 fail to disengage themselves from the object, and experience a reaction of anger, aggression and arousal.

Type 3: Ambivalence

Persons of this type show a tendency to shift from the typical reaction of Type 1 to the typical reaction of Type 2, and back again. As Grossarth-Maticek (1986) put it: 'This type shows a permanent tendency to regard an emotionally highly valued object alternately as the most important condition for his own well-being, and as the main cause for his own unhappiness' (p. 27). Thus in individuals of this type, we have an alternation of feelings of hopelessness/helplessness and of anger/arousal.

Type 4: Personal autonomy

The typical reactions of Types 1, 2 and 3 indicate a dependence on the highly valued object and their reactions are characterized by constant contradiction between expected consequences and the actual consequences of their actions. For persons of Type 4 there is a strong tendency to regard their own autonomy, and the autonomy of the persons with whom they wish to be in contact, as the most important condition for their own well-being and happiness. This enables persons of Type 4 to experience realistically the approach or avoidance behaviour of the object of their desires, and thus enables them to accept the autonomy of the object. In other words, persons of

Types 1 and 2 show a dependence on important objects which engage their emotions, but cannot remain autonomous when these emotional objects withdraw or remain unattainable; it is this that constitutes the stress which according to the theory leads to cancer or coronary heart disease. Persons of Type 4 are able to deal with this situation by virtue of their autonomy-preserving ability, and thus avoid the stress reaction.

The data to be presented now were taken from the Yugoslav study already described, as well as from two samples collected in Heidelberg and followed up between the years 1972 to 1982. The first of the two Heidelberg samples was a random sample, with certain age and sex controls specified, but otherwise with subjects selected on a random basis. The sample consisted of 1026 persons, 54 per cent of whom were male with 90 per cent being between the ages of 40 and 60. This sample is thus considerably younger than the Yugoslav one, and hence would be expected to have many fewer deaths at follow-up.

The third sample was selected by members of the normal Heidelberg sample, who nominated friends and relatives who were 'highly stressed'; this sample contained 1537 persons, 50 per cent of whom were male with ages ranging from 42 to 63 in 90 per cent of the sample. From both the Heidelberg samples there were losses due to the ascertainment of chronic disease in some members, and there were also losses at follow-up due to leaving the town, leaving 872 for the normal sample, and 1273 for the highly stressed sample. In addition, 231 cases in the highly stressed sample were used for an intervention study to be discussed presently, some acting as controls, others being included in the experimental group, and these must be subtracted from the sample, leaving a total of 1042 persons.

We thus have three samples differing in sex composition, age and amount of stress experienced; we would expect on theoretical grounds that a higher proportion of the Yugoslav and the highly stressed Heidelberg study would die of cancer and coronary heart disease, the former because of their higher age, the latter because of the stress experienced, than that of the normal Heidelberg sample. Our interest is primarily in the effect of stress on morbidity, i.e. a comparison of the two Heidelberg samples, and secondly on the specific relationship between typology within each of these three groups, and the death rates from cancer and coronary heart disease respectively, the expectation being that persons of Type 1 would die more frequently of cancer, persons of Type 2 more frequently of coronary heart disease, with persons of Type 3 and 4 relatively protected against both. The age and sex composition of the three groups is given in the paper by Grossarth-Maticek, Eysenck & Vetter (1987).

The results of the Yugoslav study are given in Table 5, and shown in diagrammatic form in Fig. 2. The most crucial figures are those which show that of Type 1, 46.2 per cent died of cancer, but only 5.6 per cent of coronary heart disease, whereas of those with Type 2, 8.3 per cent died of cancer, and 29.2 per cent of coronary heart disease. Negligible numbers of individuals of Types 3 and 4 died of either cancer or coronary heart disease, and individuals of Type 4 show a 90.7 per cent survival rate, as compared with 56.7 per cent of Type 3, 28.3 per cent of Type 2, and 23.8 per cent of Type 1. The numbers of individuals in the 4 type categories are given in the final column, and it will be seen that in this relatively normal and unselected sample, the great majority were of Type 4.

Table 5. Death by cancer and coronary heart disease in various personality type groups, Yugoslavia sample (Grossarth-Maticek, Eysenck & Vetter, 1987)

	Living	Cancer	Coronary heart disease	Other causes of deaths	Total *n*
Type 1	72 = 23.8%	140 = 46.2%	25 = 8.3%	66 = 21.8%	303
Type 2	96 = 28.3%	19 = 5.6%	99 = 29.2%	125 = 36.9%	339
Type 3	123 = 56.7%	4 = 1.8%	20 = 9.2%	70 = 32.3%	217
Type 4	437 = 90.7%	3 = 0.6%	8 = 1.7%	34 = 7.1%	482
Impossible to allocate to type	6	0	4	2	12
Total	734 = 54.2%	166 = 12.3%	156 = 11.5%	297 = 27.0%	1353

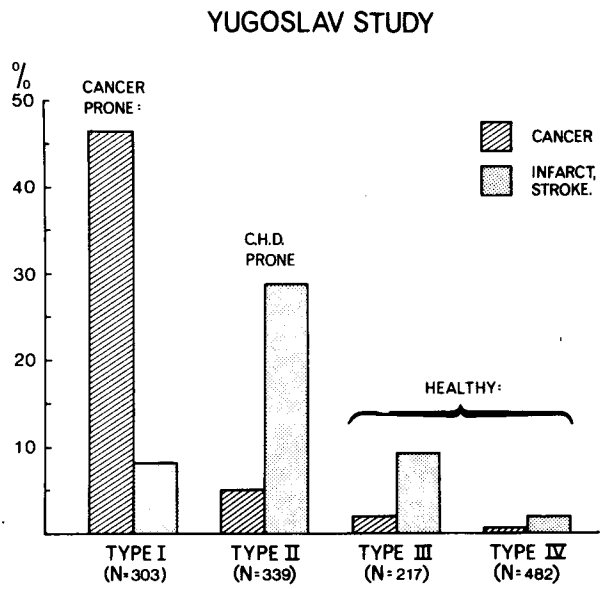

Figure 2. Main causes of death for four personality types in Yugoslav study (Grossarth-Maticek, *et al.*, 1982).

Results for the normal Heidelberg sample are given in Table 6, and in diagrammatic form in Fig. 3. The survival rate is of course much higher in this sample, because it is younger than the Yugoslav one. The crucial figures again are the ones showing that of Type 1, 17.4 per cent died of cancer, whereas of those of Type 2, only 5.9 per cent died of cancer. Conversely, only 1.8 per cent of Type 1 died of coronary heart disease, whereas 13.5 per cent of Type 2 did so. Individuals of Type 4 again showed the highest survival rate, followed by those of Type 3. In this sample there were almost as many persons of Type 4 as there were of Types 1, 2 and 3 together.

Table 6. Death by cancer and coronary heart disease in various personality type groups, Heidelberg normal sample (Grossarth-Maticek, Eysenck, & Vetter, 1987)

	Living	Cancer	Coronary heart disease	Other causes of deaths	Total *n*
Type 1	78 = 71.6%	19 = 17.4%	2 = 1.8%	10 = 9.2%	109
Type 2	109 = 64.1%	10 = 5.9%	23 = 13.5%	28 = 16.5%	170
Type 3	185 = 98.4%	0	1 = 0.5%	2 = 1.1%	188
Type 4	387 = 99.0%	0	1 = 0.3%	3 = 0.8%	391
Impossible to allocate to type	14	0	0	0	14
Total	773 = 88.6%	29 = 3.3%	27 = 3.1%	43 = 4.9%	872

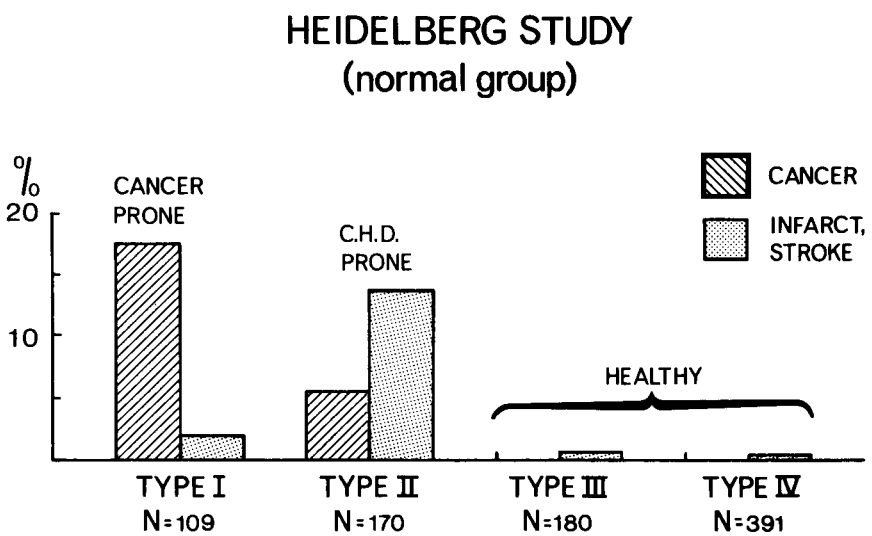

Figure 3. Main causes of deaths for four personality types in Heidelberg normal study (Grossarth-Maticek, *et al.*, 1982).

The results of the Heidelberg stressed sample are given in Table 7, and in Fig. 4. The results are rather similar to those of the Yugoslav study. Of those persons who were of Type 1, 38.4 per cent died of cancer, whereas only 2.3 per cent of Type 2 did so. Conversely, 27.8 per cent of those of Type 2 died of coronary heart disease, but only 7.0 per cent of Type 1. As in the other two samples, persons of Type 4 had the lowest death rate, followed by those of Type 3. In this group, as expected, only a small minority were of Type 4.

All the results given in these tables and figures are of course highly significant statistically. Chi^2 statistics were calculated, correcting for age and sex differences in each case, using the Mantel-Haenszel (1959) formula; this left all the differences mentioned beyond the 0.01 level.

Table 7. Death by cancer and coronary heart disease in various personality type groups, Heidelberg stressed sample (Grossarth-Maticek, Eysenck & Vetter, 1987)

	Living	Cancer	Coronary heart disease	Other causes of death	Total *n*
Type 1	188 = 38.4%	188 = 38.4%	34 = 7.0%	79 = 16.2%	489
Type 2	148 = 47.9%	7 = 2.3%	86 = 27.8%	68 = 22.0%	309
Type 3	153 = 92.7%	4 = 2.4%	0	8 = 4.8%	165
Type 4	71 = 97.3%	0	0	2 = 2.7%	73
Impossible to allocate to type	6	0	0	0	6
Total	566 = 54.3%	199 = 19.1%	120 = 11.5%	157 = 15.1%	1042

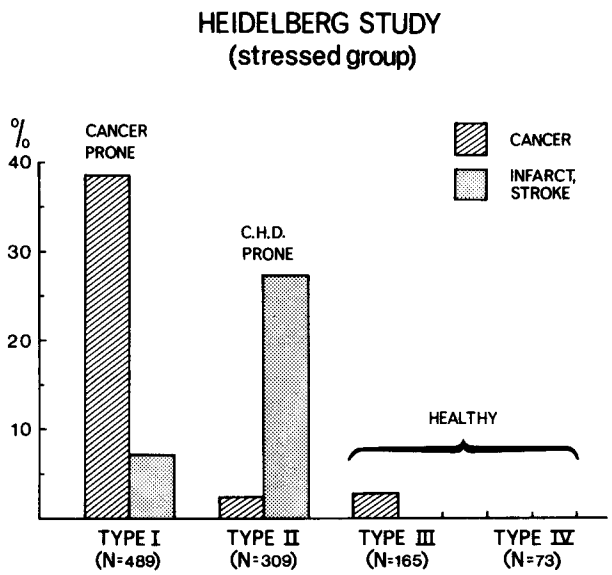

Figure 4. Main causes of deaths for four personality types in Heidelberg stressed sample (Grossarth-Maticek, *et al.*, 1982).

We must now turn to a consideration of the importance of smoking for the type-cancer relationship. Table 8 gives the number of cancer deaths, other deaths and total deaths for non-smokers and smokers of Type 1, as compared with individuals of the other three types, for the Yugoslav and the Heidelberg stressed sample. (There were not enough deaths to carry out a meaningful analysis in the normal Heidelberg sample.) Among non-smokers, as expected, there are very few deaths from lung cancer, but of the 13 that occur in total, 10 occur in persons of Type 1. For smokers, there are 74 deaths, only six of which occur in persons other than Type 1. These results give rise to an association between Type 1 and lung cancer of $P = 0.0001$ for

Table 8. Number of cancer and other deaths for smokers and non-smokers of Type 1 (Grossarth-Maticek, Eysenck & Vetter, 1987)

	Yugoslavia			Heidelberg		
	Lung cancer deaths	Other deaths	Total	Lung cancer deaths	Other deaths	Total
Non-smokers						
Type 1	1 = 0.8%	118	119	9 = 3.8%	227	236
Others	0	550	550	3 = 1.0%	297	300
Smokers						
Type 1	31 = 16.9%	153	184	37 = 14.6%	216	253
Others	6 = 1.2%	428	488	0	247	247

both the samples considered when a correction has been made for differences in smoking habits according to the Mantel-Haenszel (1959) formula. It is clear that quite independently of smoking, individuals of Type 1 are cancer-prone, as compared to individuals of Types 2, 3 and 4.

The table also makes clear the synergistic interaction between smoking and typology. The only group which has a high proportion of deaths from lung cancer is that of smokers of Type 1. Smokers not of Type 1, and non-smokers either of Type 1 or of the other types have negligible rates of cancer deaths. Of the two factors, smoking and personality, personality would seem to be the stronger. Of 735 smokers not of Type 1, only six were found to have died of lung cancer; this figure is not very different from three non-smokers not of Type 1 who died of the 850 individuals who were non-smokers. Smoking would appear to present a danger to health as far as lung cancer is concerned only for individuals of Type 1. This finding strongly supports the stress laid by Eysenck (1980, 1985) on the importance of personality factors in this field, and the highly specific nature of the relationship between smoking and lung cancer. We might like to consider the possibility of isolating a particular group at risk for lung cancer from smoking, while for the other, much larger groups of personality types there is no such relationship.

We have looked at the relationship between psychosocial variables, smoking and cancer from two different points of view, namely those of trait and type personality measurement. We may now ask about the degree of relationship between these two sets of variables. Table 9 gives the means within type groups of the trait variables considered in the previous section, and the *eta* correlations for the three samples respectively. Note that the rationality–anti-emotionality scale was not administered to the representative Heidelberg sample. The data clearly show a reasonably close relationship between the original questionnaire studied, and the typology.

A final question to be asked relates to the similarity of the typology here developed to, and its relationship with other typologies widely used in English-speaking countries. A recent survey by Temoshok (in press) suggests close ties between the two sides. There appears to be a similarity between the 'Type A' personality and our

Table 9. Relationship between traits and types in three samples (Grossarth-Maticek, Eysenck & Vetter, 1987)

	Yugoslavia			Heidelberg			
	Type		*eta*	Representative	*eta*	Stressed	*eta*
Number of life-events leading to hopelessness	1	2.74	—	6.11	—	6.11	—
	2	1.01	—	3.44	—	3.90	—
	3	0.53	—	1.81	—	3.50	—
	4	0.39	—	0.80	—	2.39	—
Signif. of differences	—	0.0000	0.38	0.0000	0.62	0.0000	0.54
Number of life-events leading to anger	1	1.49	—	1.72	—	2.43	—
	2	1.81	—	3.56	—	4.94	—
	3	0.51	—	1.04	—	2.36	—
	4	0.33	—	0.40	—	1.37	—
Signif. of differences	—	0.0000	0.29	0.0000	0.53	0.0000	0.47
Rationality/anti-emotionality	1	8.19	—	—	—	6.76	—
	2	7.52	—	—	—	5.69	—
	3	3.62	—	—	—	4.35	—
	4	2.24	—	—	—	3.71	—
Signif. of differences	—	0.0000	0.59	—	—	0.0000	0.38
Need for harmonious interpersonal relationships	1	2.34	—	3.97	—	2.83	—
	2	1.33	—	3.20	—	2.73	—
	3	1.23	—	3.39	—	3.27	—
	4	1.25	—	3.21	—	3.31	—
Signif. of differences	—	0.0000	0.30	0.0000	0.19	0.0000	0.18
Ignoring signs of illness	1	1.93	—	1.67	—	1.71	—
	2	1.30	—	1.53	—	1.61	—
	3	1.14	—	1.33	—	1.33	—
	4	1.02	—	1.22	—	1.43	—
Signif. of differences	—	0.0000	0.27	0.0000	0.15	0.0000	0.14

Note. The table reports means within type groups.
Significances are from analysis of variance F tests. The etas are analogous to correlation coefficients.

'Type 2' – at least as long as we concentrate on the relevant aspects of Type A. Type B would correspond to our 'Type 4', to a reasonable approximation. Temoshok (1987) has suggested, on the basis of her own work (1985) and of a review of cancer–psychosocial factors associations, that there existed a 'Type C', different and indeed contrasting with Type A and characteristic of the cancer-prone personality, see also Morris & Greer, 1980. This 'Type C' corresponds quite closely to our 'Type 1', although it should be remembered that 'Type C' is derived from different studies of patients already diagnosed as suffering from cancer, while our 'Type 1' is derived from three prognostic studies in which personality diagnosis was made of healthy

individuals, and preceded by 10 years the establishment of death and cause of death. This 'Type 1' or 'Type C' is related not only to *proneness* but also to the development of cancer, once diagnosed, the activity of NK cells (natural killer cells), and the level of corticosteroids (particularly cortisol). These similarities emerging from widely different research paradigms, different measuring instruments, in different countries, must be regarded as encouraging for the recognition of psychosocial and personality factors as causal aspects in cancer.

One further point may be made, in connection with our primary purpose of establishing a relationship between stress and disease. In the 10-year follow-up of the two Heidelberg samples, we noted that approximately 40 per cent more died in the stressed than in the normal sample. A further follow-up of these two samples has just been completed, extending the total period by another three years. The 40 per cent greater number of deaths in the stressed group was maintained in this additional follow-up, demonstrating once again the importance of stress–strain for disease and mortality. Being prospective, these studies do not suffer from the usual problems of retrospective studies.

The theory underlying the series of studies reported here postulated that personality (i.e. organized and regular patterns of behaviour characteristic of a person) was a causal factor in the genesis of cancer. However, all the evidence so far presented deals with *correlations*, and the postulation of a causal relationship may still be doubtful. Thus the possibility cannot be gainsaid that cancer may produce changes in personality, and that the long developmental period of cancers prior to their being diagnosed may have been effective in mediating this relationship. The only way in which a causal as opposed to a *correlational* relationship may be indicated is by an intervention study, i.e. the use of the experimental method in *changing* one of the supposedly causal, independent variables, and studying the effect of this on the dependent variable, in this case cancer.

A study of this kind was in fact done on a subsample of the Heidelberg stressed group. One hundred subjects were taken from this group, and randomly divided into a control and a therapy group (Grossarth-Maticek, Eysenck, Vetter & Frentzel-Beyme, 1986). The cognitive behaviour therapy used here was essentially based on the hypothesis that a relationship existed between personality, as outlined in previous sections, and cancer; the aim of the therapy then was to *change* the behaviour of the person from that characteristic of Type 1 to that characteristic of Type 4. In other words, socially acceptable expressions of emotion were encouraged, and the person was taught coping behaviours appropriate to his particular experience of stress–strain. Therapy was individual, and attempted to teach coping behaviours appropriate to the individual's particular situation.

Previous accounts of this work have made use of the 10-year follow-up, but of the total group of 100 only 91 could be traced, the others having moved away from Heidelberg. In an additional three-year follow-up, all the missing subjects were traced, so that we are now dealing with the whole sample of 100 subjects, 50 in the control group and 50 in the therapy group.

Table 10 shows the results of the study. It will be seen that of those in the control group, 19 are still living, although in the therapy group 45 are still living. In the control group, 16 died of cancer; in the therapy group none. These data, as far as

Table 10. Deaths in therapy and control groups

	Living	Died (cancer)	Died (other causes)	Total
Control groups	19	16	15	50
Therapy groups	45	0	5	50
				100

cause of death is concerned, are still preliminary, a careful re-checking being under way at present to make certain of the correct diagnosis, but there can be no doubt from the figures that these cancer-prone probands (all of Type 1) died predominantly of cancer, that those who received therapy survived much more frequently than those who did not receive therapy, and that the number of deaths in the control group was much higher than would have been expected in a non-stressed sample of the population.

These data are important, both from the theoretical and the practical point of view. They suggest that the relationship between personality and cancer is indeed a causal one, although of course they do not definitively prove such a causal relationship. This would require a general theory from which the relationship could be deduced, as well as detailed proof for the various links in that theory, connecting personality with cancer. An attempt to present an early version of such a theory will be made presently, but many of the important links are still missing, and it would be irresponsible to claim too much for the data existing at the moment.

The data in Table 10 may also be looked at from a more practical point of view; they suggest emphatically that prophylactic intervention is possible and indeed strongly indicated in the case of individuals of Type 1. If the study could be replicated, it would suggest that death from cancer can be avoided, or at least postponed, by the use of cognitive behaviour therapy. This is an important conclusion, particularly because of the relatively poor evidence often cited in an attempt to show that giving up smoking enables subjects to avoid or postpone cancer (Eysenck, 1980; 1986).

The theory in question (Eysenck, 1986) is outlined in diagrammatic form in Fig. 5. It maintains essentially that certain hormones and peptides influence *both* personality and the immune system, thus producing the observed correlation between personality and cancer. The endocrine system, in turn, can be influenced by external stress–strain, and by changes in personality behaviour, such as that produced by attempts to alter Type 1 to Type 4 behaviour. Stress is divided into acute and chronic stress, because there is some evidence that while acute stress may lead to immunosuppression and cancer, chronic stress may lead to inoculation and protection against cancer (Sklar & Anisman, 1981; Eysenck, 1983).

As an example of the complex interaction between peptides and hormones we may consider cortisol. Cortisol is known to be related both to depressive feelings of hopelessness/helplessness, and to immunosuppression, and there is evidence that

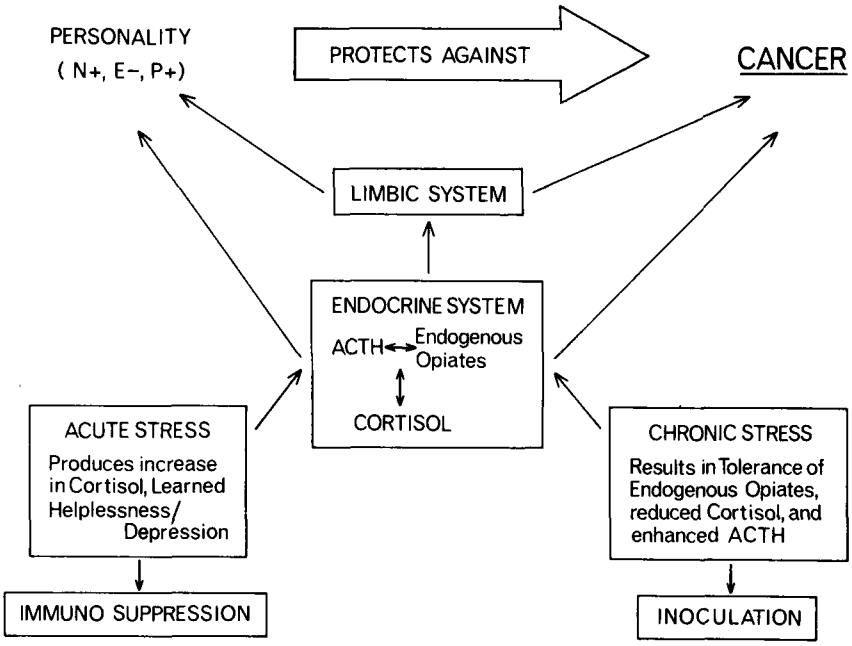

Figure 5. Causal theory linking personality and cancer (Eysenck, 1986).

when stress leads to these characteristic strain reactions, there is a decline in the activity of natural killer cells (e.g. Goodkin, Antoni & Blaney, 1986; Levy, Herberman, Lippman & d'Angelo, 1987; Levy, Herberman, Maluish, Schlien & Lippman, 1985). Thus it seems possible that people who are genetically predisposed to react to stress and strain with feelings of helplessness/hopelessness and depression, increased their cortisol level and thereby produced immunosuppression, lowering of natural killer cell activity, etc. In this way, personality, stress–strain and cancer might be related along a perfectly intelligible chain of reactions.

A fuller account of the theory will be found in Eysenck (1986), but it should be noted that cortisol here only stands as a representative of a very complex and interacting chain of peptides and hormones, including among others ACTH, the endogenous opiates, etc. It cannot be the purpose of this chapter to go into details concerning the theory; it is only mentioned to demonstrate that the relationship between personality and cancer is not as unlikely and outside the realm of natural science investigation as migh seem at first sight.

Direct evidence that cognitive behaviour therapy can not only affect morbidity, but can also affect the cortisol level is given by Rodin (1984, 1986) who gave such a therapy to one group of elderly females, while giving placebo or no treatment to control groups. She succeeded in showing that the therapy group did much better as far as survival was concerned, but also was the only group to show a permanent reduction in cortisol level.

It is not suggested that the positions with respect to the interaction between

personality, stress–strain and cancer have been worked out in sufficient detail, or that the theory outlined very briefly above is more than a guidepost to future research. There is now, however, too much empirical material to doubt that stress–strain, interacting with personality, plays a causal role in the genesis of cancer, probably in combination with such factors as smoking, drinking, etc. Details about this synergistic interplay still remain to be worked out, but it is clear that simplistic formulations like 'Smoking causes cancer' have no part to play in the scientific study of this disease.

References

Booth-Kewley, S. & Friedman, H. S. (1987). Psychological predictors of heart disease: A quantitative review. *Psychological Bulletin*, **101**, 343–362.

Chesney, M. A. & Roseman, R. H. (eds) (1985). *Anger and Hostility in Cardiovascular and Behavioural Disorders*. Washington DC: Hemisphere.

Cooper, C. L. (1983). *Stress Research*. New York: Wiley.

Cooper, C. L. (1984). *Psychosocial Stress and Cancer*. New York: Wiley.

Cooper, E. L. (Ed.) (1984). *Stress, Immunity, and Ageing*. New York: Marcel Dekker.

Dobson, C. B. (1982). *Stress: The Hidden Adversary*. Lancaster Medical and Technical Publishers.

Eysenck, H. J. (1967). *The Biological Basis of Personality*. Springfield, IL: Thomas.

Eysenck, H. J. (1975). The measurement of emotions: Psychological parameters and methods. In L. Levi (Ed.), *Emotions – Their Parameters and Measurement*, pp. 439–467. New York: Raven Press.

Eysenck, H. J. (1980). *The Causes and Effects of Smoking*. London: Maurice Temple Smith.

Eysenck, H. J. (1983). Stress, disease and personality: The 'inoculation effect'. In C. L. Cooper (Ed.), *Stress Research*, pp. 121–146. New York: Wiley.

Eysenck, H. J. (1985). Personality, cancer and cardiovascular disease: A causal analysis. *Personality and Individual Differences*, **5**, 535–557.

Eysenck, H. J. (1986). Smoking and health. In R. D. Tollison (Ed.), *Smoking and Society*, pp. 17–88. Lexington: Lexington Books.

Eysenck, H. J. (1987). The respective importance of personality, cigarette smoking and intervention effects for the genesis of cancer and coronary heart disease. *Personality and Individual Differences*, (in press).

Eysenck, H. J. & Eysenck, M. W. (1985). *Personality and Individual Differences*. New York: Plenum Press.

Friedman, H. S. & Booth-Kewles, S. (1987). The 'disease-prone personality'. *American Psychologist*, **42**, 534–555.

Goodkin, K., Antoni, M. H. & Blaney, P. H. (1986). Stress and hopelessness in the promotion of cervical intraepithelial neoplasma to an invasive squamous cell carcinoma of the cervix. *Journal of Psychosomatic Research*, **30**, 67–76.

Grossarth-Maticek, R. (1980). Synergistic effects of cigarette smoking, systolic blood pressure and psychosocial risk factors for lung cancer, cardiac infarct and apoplexy cerebri. *Psychotherapy and Psychosomatics*, **34**, 267–272.

Grossarth-Maticek, R., Bastiaans, J. & Kanazir, D. T. (1985). Psychosocial factors as strong predictors of mortality from cancer, ischaemic heart disease and stroke: The Yugoslav prospective study. *Journal of Psychosomatic Research*, **29**, 167–176.

Grossarth-Maticek, R., Eysenck, H. J. & Vetter, H. (1987). Personality type, smoking habit and their interaction as predictors of cancer and coronary heart disease. *Personality and Individual Differences*, (in press).

Grossarth-Maticek, R. Eysenck, H. J., Vetter, H., & Schmidt, P. (1986). Results of the Heidelberg prospective psychosomatic intervention study. Paper presented at the International Conference on Health Psychology, Tilburg University, 3–5 July.

Grossarth-Maticek, R., Eysenck, H. J., Vetter, H. & Frentzel-Beyme, R. (1986). The Heidelberg prospective intervention study. Paper presented at the First International Symposium of Primary Prevention of Cancer: Antwerp, Belgium, 19–22 March. Raven Press.

Grossarth-Maticek, R., Frentzel-Beyme, R. & Becker, N. (1984). Cancer risks associated with life events and conflict solutions. *Cancer Detection and Prevention*, **7**, 201–209.

Grossarth-Maticek, R., Kanazir, D., Schmidt, P. & Vetter, H. (1982*a*). Psychosomatic factors in the process of cancerogenesis. *Psychotherapy and Psychosomatics*, **38**, 284–302.

Grossarth-Maticek, R., Kanazir, D. T., Schmidt, P. & Vetter, H. (1982*b*). Psychosocial and organic variables for lung cancer, cardiac infarct and apoplexy. Paper presented at Plenary Session of XIV European Conference on Psychosomatic Research, Noordwijkerhout, 19–25 September.

Grossarth-Maticek, R., Kanazir, D. T., Vetter, H. & Jankovic, M. (1983). Smoking as a risk factor for lung cancer and cardiac infarct as mediated by psychosocial variables. *Psychotherapy and Psychosomatics*, **39**, 94–105.

Grossarth-Maticek, R., Kanazir, D. T., Vetter, H. & Schmidt, P. (1983). Psychosomatic factors involved in the process of cancer genesis. *Psychotherapy and Psychosomatics*, **40**, 191–210.

Grossarth-Maticek, R., Schmidt, P., Vetter, H. & Arndt, S. (1984). Psychotherapy research in oncology. In A. Steptoe & A. M. Matthews (Eds), *Healthcare and Human Behaviour*, pp. 325–340. London: Academic Press.

Grossarth-Maticek, R., Siegrist, J. & Vetter, H. (1982). Interpersonal regression as a predictor of cancer. *Social and Scientific Medicine*, **16**, 493–498.

Kanazir, D., Djordjevic-Markovic, R. & Grossarth-Maticek, R. (1984). Psychosocial (emotional), stress, steroid hormones and carcinogenesis: Molecular aspects, facts and speculations. In Y. A. Ouchinnikov (Ed.), *Progress in Bioorganic Chemistry and Molecular Biology*, pp. 509–520. Amsterdam: Elsevier.

Kaplan, H. B. (Ed.) (1983). *Psychosocial Stress: Trends in Theory and Research*. New York: Academic Press.

Levy, S., Herberman, R., Lippman, M. & d'Angelo, T. (1987). Correlation of stress factors with sustained depression of natural killer cell activity and predicted prognosis in patients with breast cancer. *Journal of Clinical Oncology*, 348–353.

Levy, S., Herberman, R. B., Maluish, A. M., Schlien, B. & Lippman, M. (1985). Prognostic risk assessment in primary breast cancer behavioural and immunological parameters. *Health Psychology*, **4**, 99–113.

Maddi, S. R., Bartone, D. T. & Puccetti, M. (1987). Stressful events are indeed a factor in physical illness: Reply to Schroeder and Costa (1984). *Journal of Personality and Social Psychology*, **52**, 833–843.

Mantel, N. & Haenszel, W. (1959). Statistical aspects of the analysis of data from retrospective studies of disease. *Journal of the National Cancer Institute*, **22**, 719–748.

Morris, T. & Greer, S. (1980). A 'Type C' for cancer? *Cancer Detection and Prevention*, **3**, Abstract No. 102.

Price, V. A. (1982). *Type A Behaviour Pattern*. New York: Academic Press.

Rodin, J. (1984). Managing the stress of ageing: The role of control and coping. In S. Levine & H. Norin (Eds), *Coping and Health*, pp. 171–202. New York: Plenum Press.

Rodin, J. (1986). Health, control and ageing. In M. Baltes & P. B. Baltes (Eds), *Ageing and the Psychology of Control*. Hillsdale, NJ: Erlbaum.

Schmidt, P. (1984). Antoritarismus, Entfremdung und psychosomatische Krebsforschung: Explikation der drei Forschungs-programme durch eine allgemeine Theorie und empirische Tests mittels Strukturvergleichung. Habilitationsschrift: University of Giessen.

Schroeder, D. H. & Costa, P. T. (1984). Influence of life events stress in physical illness: Substantive effects or methodological flaws? *Journal of Personality and Social Psychology*, **46**, 853–863.

Sklar, L. S. & Anisman, H. (1981). Stress and cancer. *Psychological Bulletin*, **89**, 396–406.

Steptoe, A. (1981). *Psychological Factors in Cardiovascular Disorders*. New York: Academic Press.

Temoshok, L. (1985). Biopsychosocial studies in cutaneous malignant melanoma: Psychosocial factors associated with prognostic indicators, progression, psychophysiology and tumor–host response. *Social Science and Medicine*, **20**, 833–840.

Temoshok, L. Personality, coping style, emotion, and cancer: Toward an integrative model. *Cancer Surveys* (in press).