THE RESPECTIVE IMPORTANCE OF PERSONALITY, CIGARETTE SMOKING AND INTERACTION EFFECTS FOR THE GENESIS OF CANCER AND CORONARY HEART DISEASE

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It is the privilege of the editor to note similarities of treatment, or relevance, or orientation in papers submitted to his Journal, and to bring together in one issue papers which appear to complement each other. The three papers here presented appear to complement each other, in the sense that one deals with the effects of smoking on cancer and other diseases, while the other papers deal with the role personality differences play in the genesis of cancer and coronary heart disease. In the latter papers smoking is also considered, as is the interaction between personality and smoking. Cancer is not the only disease considered in these papers, but it forms the major link between them; coronary heart disease is another link.

There is a large and somewhat acerbic literature suggesting that cigarette smoking is responsible in large part for death from many different diseases, particularly lung cancer, other cancers and coronary heart disease; details will be found in several reports from the Royal College of Physicians in England, and the Surgeon-General of the United States (Eysenck, 1980, 1985b).

Many claims are made regarding the number of people whose lives could be saved if they stopped smoking. The British Minister of Health has stated that 50,000 lives a year could be saved in Great Britain by smokers ceasing to smoke, and along similar lines his American counterpart asserted that 320,000 lives could thus be saved in his country. Even more surprising is the purported accuracy of claims made in *The Big Kill*, a fifteen-volume document launched by The Health Education Council jointly with the British Medical Association; one volume was issued for each Regional Health Authority in England and Wales. According to this publication, smoking annually kills 77,774 people (55,107 men and 22,667 women) in the whole of England and Wales from heart disease, lung cancer and bronchitis/emphysema. And because of their smoking, some 108,218 people are hospitalized each year with these diseases. The absurdity of such precision has rightly been pointed out in detail by Burch (1986) who has drawn attention to the many unproven assumptions underlying the argument.

What is not contested by anyone is that even though cigarette smoking may play a part in the genesis of cancer, coronary heart disease, and other disorders, it is neither a *necessary* nor a *sufficient* cause. As regards lung cancer, which has probably been linked most closely with cigarette smoking, of ten heavy smokers only one dies of lung cancer; hence smoking cannot be a *sufficient* cause. Of ten people who die of lung cancer, one is a non-smoker; hence smoking cannot be a *necessary* cause. These figures suggest that cigarette smoking is unlikely to be a cause of lung cancer and the other diseases mentioned, by itself, but rather that it is one of a whole set of causal determinants, including air pollution, drinking, psychosocial factors etc., which together are instrumental in causing the disease in question. In any case, it must always be remembered that a *statistical* association, however strong, does not necessarily imply a *causal* determination, and that there are many difficulties in demonstrating a causal link between cigarette smoking and disease (Eysenck, 1985b). Many eminent statisticians have voiced their disquiet about the cavalier way in which statistical and epidemiological methods have been used in relation to this problem (Berkson, 1958; Burch, 1976, 1978; Fisher, 1959; Katz, 1969; Sterling, 1973), and many other critics could be named. Clearly it would be premature to consider the controversy closed, as is often

suggested by those who consider cigarette smoking almost solely responsible for the majority of deaths from cancer, heart disease, and many other disorders.

As Wakefield points out in his contribution to this symposium, in the paper following this introduction, data on the statistical relationship between disease and cigarette smoking have usually been presented in the form of the relative risk of the disease for smokers and non-smokers. This is a quotient of the probability of the disease for smokers and the probability of the disease for non-smokers, and is interpreted as the number of times the risk for non-smokers is increased for smokers. As he points out, relative risk is a concept which does not measure the *degree of association* between smoking and disease, and it is very much preferable to state the relationship in terms of *correlations* which, particularly for psychologists, has a definite meaning first because of its statistical derivation, and second because of the long acquaintance of psychologist with statements of relationship made in those terms. When this is done, as he shows, the relationships observed in published studies are indeed minute; less than 3% of the total variance of the disease process (and in good studies perhaps more like 1%) is accounted for in terms of cigarette smoking! Wakefield has made an important contribution by thus translating the non-statistical estimates made by epidemiologists and medical people to terms having a precise statistical meaning.

An alternative or possibly a complementary theory to that which makes carcinogenic substances (tobacco, pollution, asbestos etc.) responsible for cancer, is one which makes psychosocial factors (stress, personality) responsible. Theories of this kind go back to Hippocrates and Galen, and have recently found much support in empirical studies. Stress in particular has received much attention, as shown by recent reviews by Fox (1978, 1983), Bammer and Newberry (1981), Cooper (1983) and Dobson (1982). There has also been much recent research on the relationship between stress, behaviour and the immune system (Borysenko and Borysenko, 1982). The causal hypothesis in this work has been that stress (particularly uncontrollable stress) leads to helplessness as defined by Seligman (1975), and suppresses the activity of the immune system, perhaps through the production of cortisol and other corticosteroids, which are known to reduce the efficacy of the immune system.

Weyer and Hodapp (1977) and Hodapp and Weyer (1982) have added the important point that neither objective environmental variables nor certain personality characteristics alone cause stress; a particular individual's evaluation of his environment is thought to be decisive in causing stress. "This means, of course, that when illness as a result of stress is to be investigated, it is not enough to search for isolated relationships between illness and personality or between illness and environment" (Weyer and Hodapp, 1977, p. 337). Their own investigations have resulted in a causal model which links various stress factors with extraversion and neuroticism (Hodapp and Weyer, 1982, p. 133). The point that stress cannot be objectively defined in terms of situations only, but always in relation to the reaction to the stress of the organism (strain), has been emphasised by Eysenck (1985b). In making this distinction between stress and strain, Eysenck (1975) used an analogy with Hooke's Law of elasticity: $Stress = k \times Strain$ where k is constant (the modulus of elasticity) that depends upon the nature of the material and the type of stress used to produce strain. This constant k, i.e. the Stress/Strain ratio, is called Young's modulus, and would seem to apply equally well to human emotions and stress, as it does to the elasticity of physical bodies. Stress is the objective situation which impinges on the individual; strain is the reaction produced in the individual by the externally imposed stress. The strain depends of course in part on the stress, but also on the specific type of stress used, and the personality of the individual reacting. This distinction is absolutely vital in understanding the importance of stress for physical disease, and unfortunately has been much neglected in the literature.

One indication of the importance of the *type* of stress used concerns the difference between *acute* and *chronic* stress. *Acute* stress apparently increases the likelihood of the development of carcinomas, whereas chronic stress may have the opposite effect (Sklar and Anisman, 1981). Eysenck (1983) has labelled this the "inoculation effect".

Turning next to personality factors, we find that two major sets of traits have been linked with cancer in the past. Already in the, 2nd Century after Christ, Galen considered that melancholic women suffer from cancer more frequently than sanguine women, and in the 19th Century several medical people have offered similar views based on their personal observations (Nunn, 1882; Walshe, 1846; Paget, 1870). This loss-depression-hopelessness syndrome (LeShan, 1959) and its relationship to cancer have formed the basis of much research (Eysenck, 1985b). It attributes the

development of cancer to the loss of significant objects in the life of the given person, such as career disappointments, loss of self-esteem, death of a loved person, etc. This conception is close to Seligman's (1975) well known concept of *learned helplessness*. It is often failure to cope with the stress-arousing situation that links it with the uncontrollable nature of cancer-producing stress in animal experiments, suggesting that possibly the teaching of the use of coping mechanisms through behaviour therapy might help to prevent this particular cause of disease (Eysenck, 1987a, b).

The second of the personality-related syndromes in cancer is one emphasising lack of emotional reaction, or its suppression. It is postulated that the onset and development of malignant tumors may be associated with the excessive use of repressive and denying mechanisms (Bahnson and Bahnson, 1964) or a general inhibition of emotional reactions (Kissen and Eysenck, 1962; Kissen, 1963a, b). These two traditional theories have received a certain amount of anecdotal support over the centuries, and in recent years there have been empirical studies which have gone some way towards supporting them. A review is given elsewhere (Eysenck, 1985a). The major criticism of much of the published work is related to the fact that studies were carried out on patients already suffering from cancer; usually the investigations were carried out on patients prior to diagnosis, so that the effects of fear and anxiety are likely to be roughly equal in strength in those who later on were found to suffer from these disorders and those who were not. Nevertheless, prospective studies are obviously desirable and much more likely to be informative than studies carried out on patients already ill. There is also the problem that even though may be a correlation between cancer and personality, the long developmental period of sub-clinical carcinomas makes it possible that it is the cancer that causes changes in personality, rather than personality being instrumental in causing cancer.

Cancer is not the only major disease which has been linked with personality; coronary heart disease is another. Since the early work of Peete (1955) interest has mainly shifted to the so-called Type A-Type B behaviour patterns (Friedman and Rosenman, 1974; Jenkins, 1978; Price, 1982; Rosenman and Chesney, 1980; Spielberger, 1976; Steptoe, 1981; Weyss, Detre and Cooper, 1981). Eysenck and Fulker (1983) have shown how the concept of Type A behaviour is related to extraversion and neuroticism, and that it is strongly determined by genetic factors, and several recent authors (Barefoot, Dahlstrom and Williams, 1983; Innes, 1980; Kantor and Robertson, 1977; and Williams, Barefoot and Shekelle, 1984) have drawn attention to the great importance of hostile and aggressive behaviour in relation to coronary heart disease.

Some of the earlier empirical studies, such as those by Carver and Glass (1978), Diamond (1982), Matthews, Glass, Rosenman and Bortner (1977), Strube, Turner, Cerro, Stevens and Hinchey (1984) and Van Egeren (1979) support this view, but certain possible questions are not answered; Check and Dyck (1986) report data which appear to put the relevance and importance of hostile aggression in the Type A personality beyond doubt. (See also Spielberger and London, 1982).

Altogether the unitary nature of Type A behaviour has been heavily criticized, and the special importance of the rated interview behaviour, as opposed to the content of the interview answers, has been emphasised (Dembroski and McDougall, 1985).

There are other studies linking coronary heart disease with neuroticism and anxiety (Bendien and Groen, 1963; Blumenthal, Thompson, Williams and Kong, 1979); sociability (Van Dijl, 1979), and other personality traits (E. G. Brozek, Keys and Blackburn, 1966; Dembroski, Weiss and Shields, 1978; Ibrahim, Jenkins, Cassel, McDonough and Hames, 1963; Ostfeld, Lebovits, Shekelle and Paul, 1967; Rime and Bonami, 1979; Siltanen, Lauroma, Nirkko, Punsar, Pyorala, Tuominen and Vanhala, 1975; Storment, 1951; and Theorell, deFaire, Schalling, Adamson and Askevold, 1979), but there are also severe criticisms to be made of all these studies (e.g. Bass and Wade, 1982; Costa, 1985; Costa, Fleg, McCrae and Lakatta, 1982; Jemmote and Locke, 1984) which suggest that while there may be some truth in these studies, it is difficult to come to very firm conclusions.

The theory linking personality and cancer can usefully be extended to cover also duration of life after diagnosis, i.e. the ability of the organism and its immune system to combat the disease. There is considerable evidence for the existence of such a relation (Levy, 1983). Greer *et al.* (1979, 1985) found in a 10-year follow-up that women with breast cancer who were rated as having a fighting spirit had better outcomes than women who were rated as helpless or stoic. Rogentine *et al.* (1979) reported significantly greater relapse in melanoma patients showing a passive or stoic response style. Visintainer and Casey (1984) demonstrated a similar association between passivity and disease course in melanoma patients. Similar results are reported by Derogatis *et al.* (1979) in breast cancer patients, and Jensen and Muenz (1984); also in breast cancer patients.

Levy et al. (1985) reports a study linking personality and an immunological mediator (the natural killer cell, NK); NK activity was found to have prognostic significance. Patients who had higher levels of NK activity at the time of primary treatment had significantly fewer nodes positive. The crucial finding was that patients who were rated as "adjusted" by independent observers, i.e. who make no complaints and had no apparent psychological difficulties, and who responded with a listless, apathetic response style tended to have significantly lower levels of NK activities than patients who appeared more disturbed. These latter patients tended to be more negatively reactive at the time of interview. "On the basis of these three factors—observer ratings of adjustment, perceived social support and level of reported listlessness we could account for 51% of the NK variance in these patients". (Levy, 1985, p. 167.)

Animal studies have also given support to the hypothesis that "learned helplessness" is associated with cancer (Laudenslager *et al.*, 1983, 1984); this and other studies suggest the existence of a causal relationship between acute behavioural helplessness in rats and mice, suppression of lymphocyte functioning, and faster tumor growth. This relationship seems to be modulated by endogenous opioids, since the experimental effects were reversed when an opioid antagonist was injected. In most of this work, the yoked "helpless" paradigm has been used, in which one rat is given shocks which can be prevented by suitable actions; this rat is yoked to another who receives identical shocks but is unable to prevent them. It is the uncontrollable shocks which produced stress leading to cancer, depleted brain norepinephrine levels, and depleted dopamine and serotonin levels in some brain areas, the extent and duration of the effects depending on age, strain of animal, and social housing conditions (Sklar and Anisman, 1981).

If we were to disregard the criticisms, the many failures to replicate (possibly due in part to the use of different tests, questionnaires etc.) and the problem caused by the many different paradigms used, and the different groups chosen to represent cancer and coronary heart disease (Fox, 1978), one might conclude cautiously that there is some evidence for the relationship between stress and disease, that there is a cancer prone personality characterized by reactions of hopelessness-helplessness and the inhibition of emotional expression; and that there is a coronary heart disease prone personality, in many ways the obverse of the cancer prone personality, characterized by strong feelings of anxiety, and by hostility and aggressiveness. Clearly it would be most desirable if these tentative findings could be supported by a prospective study using a sufficiently large number of cases to be convincing. Such a study is available in the work of Grossarth-Maticek and his colleagues listed in the bibliography. These studies have also given rise to a causal theory (Eysenck, 1987a, 1987b; Kanazir, Djordjevic-Markovic and Grossarth-Maticek, 1984) which will not here be discussed in any detail.

The work in question refers to a completed 10-year follow-up study in Yugoslavia, the sample consisting of 1,353 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 59–65 years old. 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 were also collected, and further medical information was recorded periodically. Ten years after starting the study, a physician assessed the occurrence of different diseases in the sample, and also recorded diagnosis on the death certificate. 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% of females. This design is clearly superior to that used in most of the studies mentioned above, and avoids most of the criticisms made of work in this area by Morrison and Paffanbarger (1981).

The major results have been reported in a paper by Grossarth-Maticek, Frentzel-Beyme and Becker (1984). The questionnaire used in the study contained 109 questions concerning several personality complexes, including those already indicated in our discussion as relevant to cancer and coronary heart disease. 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. A third set of questions related to rationality and anti-emotionality, i.e. the obverse of neuroticism-anxiety. Other questionnaires



Fig. 1. Relationship between duration of hopelessness and depression, and of duration of anger and excitement to various diseases.

refer to the need for harmonious interpersonal relationships; ignoring signs of illness: lack of positive emotional relations; absence of self-reported psychopathological symptoms, especially anxiety; and finally acquiescence. Besides the fact of the reaction itself, the questionnaire contained information about the duration of the particular reaction, such as depression and hopelessness, or anger and excitement, and of the time in years passed since an important event leading to the typical chronic reaction occurred. The risk ratios associated with increasing numbers of years of the duration of an emotional state are shown in Fig. 1. The consistent increase in risk ratios for *cancer* is observed for the hopelessness–depression scale after a period of 3 years, and a similar increase for coronary heart disease for the anger and excitement scales. Thus we find in this prospective study a clear indication both of the relationship between personality reactions to stress, and disease, and also a clear-cut distinction between the cancer prone and the coronary prone personality, the former being characterized by hopelessness and depression, the latter by anger and excitement.

Schmidt (1984) has carried out large scale factor analytic and correlational studies of the results of the Yugoslav study, and discovered that the factors resulting from his analysis 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 major scales emerging from the factor analysis were rational/anti-emotional behaviour; anger/excitement; harmonization; hopelessness/helplessness; hypochondriasis; anxiety. The remaining factors were less clearly marked. The exploratory factor analysis was followed by a confirmatory analysis, but we are here more concerned with the correlations between the factors and illness, rather than with the statistical details of the factor analysis.

The correlation between 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 stemming from Galen, and emerging in more testable form in recent studies.

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 showed a *negative* correlation with cancer, to the extent of r = -0.39 (Eta = -0.41). Other factors had much lower correlations and will not be listed therefore. Obviously the various scales are not independent, and



Fig. 2. Variance in cancer incidence accounted for by 7 psychosocial variables.

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 and Vetter (1982, 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. 2. It will be seen that X_1 and X_3 retain their strong positive relationship with cancer incidence, and that X_2 retains a marked negative correlation. The other variables have quite low regression coefficients. The explained variance (\mathbb{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. 2 as e (e = R). Actually the \mathbb{R}^2 for the first three predictors is equal to 0.49, so that there is little gained by including X_4 , X_5 , X_6 and X_7 .

Schmidt (1984) has pointed out some weaknesses in the statistical treatment, particularly the assumption that the dependent variable (cancer) is dichotomous; in addition, the items of the psychosocial scales are also used in a dichotomous form. Using more appropriate statistics, Schmidt found throughout higher correlations with cancer incidence (except for the anger/excitement scale), suggesting that something like 60% of the total variance for cancer incidence could be accounted for in terms of the personality variables chosen.

It is interesting to note that the inclusion of other variables (blood cholesterol, incidence of fever, herpes, hepatitis, liver cirrhosis, Vitamin A and Vitamin C, and lymphocyte percentage) do not do much to increase the accuracy of the prediction of cancer incidence in this sample (Grossarth-Maticek, Kanazir, Vetter and Schmidt, 1983). Unfortunately no detailed analysis of the coronary heart disease data is available, comparable to the Schmidt analysis of cancer, but an unpublished study by Grossarth-Maticek, Kanazir, Schmidt and Vetter (1982b) suggested here also that personality variables are important predictors, although less so than in the case of cancer. (See also the paper by Grossarth-Maticek, Frentzel-Beyme and Becker, 1984.)

In the data so far discussed, sex and age have been controlled for, but not smoking. This is a topic of particular interest in this symposium because our main purpose is to compare the relative importance of smoking and psychosocial variables in the genesis of cancer and coronary heart disease. The multiple discriminant analysis described above gives values of 0.06 for smoking and cardiac infarct/apoplexy, and 0.24 for lung cancer (Grossarth-Maticek, Kanazir, Schmidt and Vetter, 1982). However, this information, while suggesting that smoking is considerably less important as a predictor of these diseases 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). In this study, a comparison was made between the most prominent psychosocial risk factors, and the most prominent medical risk factors. The results are contained in Table 1. In all the analyses conducted, interaction was significant and worked in the same sense, i.e. that psychosocial variables are not only important predictors of disease incidence in themselves, but that they also decisively modify the efficacy of physical risk factors. Quite consistently, the

Dependent variable	Physical risk factor	Psychosocial risk factor	Significance of interaction	Estimated stand. regression coefficient of disease on physical risk factor for psychosocial risk factor	
			term (%)	absent	present
Apoplexy	Systolic blood pressure	Rationality and anti-emotionally	<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	∢۱	0.081	0.514
Average Factor of ine	crease			0.044 6.6	0.291

Table	1.	Interaction	between	physical	and	psyc	hosocial	risk	factors
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efficacy of the physical risk factors depends decisively on the presence of some social risk factor constellation. Without this, they are, on the average, less than 1/6 as important. (The factor of increase is actually 6.6.)

The same point is brought out in another paper by Grossarth-Maticek, Kanazir, Vetter and Jankovic (1983). Table 2 shows the results of a similar analysis to that described above, but using this time two psychosocial variables. Results again point to the importance of the personality variables, and their interaction with smoking.

Of particular interest is Table 3 (Grossarth-Maticek, Bastiaans and Kanazir, 1985). This Table 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. (For women there was strong association between R/A and smoking, and in view of the smaller number of subjects dying of lung cancer no detailed analysis was possible.)

It will be clear from the Table that practically all 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. Thus there is no correlation whatever 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 having scores of 10 or 11 on the R/A scale.

Table 2. Smoking as a risk factor for cardiac infarct and lung cancer in its interactional dependence on psychosocial variables

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

Table 3. Lung cancer incidence (death/number at risk) by smoking and rationality/anti-emotionality, Males

		Smoking habits				
	Score	Never smoked	l-20 cigs/day	21 + cigs/day	Total	
Rationality and	0	0/77	0/42	0/38	0/157	
anti-emotionality	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	

This prospective study supports in most ways the cross-sectional studies summarized earlier, and suggests that psychosocial factors, like stress and personality, are of considerable value in the prediction of cancer and coronary heart disease. The data further suggest that smoking is statistically less important as a predictor than is personality, but that smoking and personality form a synergistic relationship. In these studies, personality traits have been treated independently, although it has been noted that they are not uncorrelated. This finding suggests the elaboration of a typology of cancer prone and coronary prone persons, bringing together the relevant personality descriptions in a general conceptual framework. Such a typology should predict cancer and coronary heart disease even better than the individual traits. An attempt to construct and use such a typology is reported in the last paper in this symposium.

A possible implication of the studies surveyed in this article is that the personality traits discussed do not only have a statistical but a causal relationship with cancer and coronary heart disease. Epidemiological studies, even prospective ones, can only establish correlation, but not causation, a point often neglected by those who advocate the causal role of cigarette smoking in cancer and coronary heart disease. There is now a causal theory attempting to explain the observed correlation between psychosocial factors and disease (Eysenck, 1985b), but only actual intervention studies can demonstrate the causal aspects of the relationship.

Two such studies have in fact been carried out, and reported in the literature (Eysenck, 1987a, b; Grossarth-Maticek, 1986; Grossarth-Maticek, Eysenck, Vetter and Frentzel-Beyme, 1986). In these studies, 91 cancer prone and 82 coronary prone individuals were selected, and in each case divided on a chance basis into an experimental and a control group. The experimental group received a special type of cognitive behaviour therapy attempting to change the behaviour of the individuals involved in a direction away from that characteristic of the cancer prone or coronary prone person. These efforts were largely successful. In a 13-year follow-up it was found that very significantly fewer of the experimental group, as compared with the control group, died of cancer or coronary heart disease, respectively (see Table 4). This would seem to suggest that the relationship between personality and cancer is not only statistical, but has a causal basis.

Similar to these prophylactic studies is one in which the authors look at the influence of behaviour therapy on duration of survival (Grossarth-Maticek *et al.*, 1987). One hundred women suffering from terminal cancer of the breast were divided into four groups of twenty-five each, one group receiving neither chemotherapy nor behaviour therapy, another receiving both, a third group receiving chemotherapy but no behaviour therapy, and a fourth receiving behaviour therapy but no chemotherapy. Table 5 shows the results of the study in terms of survival in months. It is clear from the table that the group receiving neither type of therapy did worst, but the group receiving both therapies did best, with those receiving only one or the other type of therapy showing roughly equal survival lengths. There is a clear synergistic effect. The mean survival time of all hundred patients was 15.7 months, with a standard deviation of 7.3 months, total survival time varying from 6 to 38 months. Chemotherapy may increase survival time by 2.80 months, while behaviour therapy alone increased survival by 3.64 months. If the two effects were additive, one would expect a survival time of 11.28 = 2.80 = 3.64 = 17.2 months for the group with combined therapies.

	In Heidelberg stressed sample Deceased from						
Risk: cancer	Alive	other cause	cancer	Tota			
Control group	19	15	16	50			
Therapy group	45	5	0	50			
Total	64	20	16	100			
	In Heidelberg stressed sample Deceased from						
Risk: infarct/stroke	Alive	other cause	infarct/ stroke	Total			
Control group	17	13	16	46			
Therapy group	37	6	3	46			
Total	54	19	19	92			

Table 4. Effect of prophylactic behaviour therapy on cancer-prone and CHD prone probands

Chemotherapy					
	no	yes	Totals		
no	mean = 11.28	mean = 14.08	mean = 12.68		
Behaviour Therapy	N = 25	N = 25	-		
yes	mean = 14.92 N = 25	mean = 22.40 N = 25	mean = 18.66		
Totals	mean = 13.10	mean = 18.24	grand		
			mean = 15.67 N = 100		

Table 5. Survival in months of groups of women with cancer of the breast as a function of type of treatment

However, the mean survival time of the chemotherapy plus behaviour group was 22.40 months, exceeding the additive value by 4.68 (P > 0.005). This indicated a positive interaction between chemotherapy and behaviour therapy has taken place, and that they operate synergistically.

We have so far discussed personality traits in the terms used by the various authors in question. In terms of the system of personality description favoured by the writer (Eysenck and Eysenck, 1985) i.e. in terms of neuroticism-stability (N) extraversion-introversion (E) and psychoticism-tendermindedness (P), we could say that neuroticism and psychoticism both act in a direction to protect a person against cancer, but extraversion has the opposite effect. These relations may be mediated by hormones and peptides in the endocrine system, particularly ACTH and cortisol, and the secretion of both are related to stress. Furthermore, these hormones and peptides affect the immune system, and thus indirectly the incidence of cancer. Figure 3 (Eysenck, 1985a, b) gives a diagrammatic account of the relationships postulated, and the same paper reviews some of the evidence in favour of the causal system suggested.

Most of the discussion so far has dealt with neuroticism-anxiety and psychoticismaggressiveness, as negative indicators of cancer and a few words may be necessary to justify the inclusion of extraversion as a positive indicator of cancer. Some of the descriptive evidence has been reviewed elsewhere by the writer (Eysenck, 1985a), but it may be of interest to note some applications relating cancer proneness to the concept of CNS arousal. As Eysenck (1967) has suggested, low arousal/arousability is characteristic of extraverts, high arousal/arousability is characteristic of introverts. De La Pena (1983) has pointed out that "a number of investigations have shown that moderate-to-high dosages of CNS activating drugs inhibit most cancers, and that drugs producing CNS depression facilitate the development of most malignancy". (p. 67). Pena also reviews evidence to show that peptides like ACTH "have proved to be particularly effective in slowing down lymphatic cancers and certain leukemias". (p. 70.) He also points out that "there is a small body of evidence in support of the hypothesis which posits that activation of the



Fig. 3. Model of cancer-personality relationship.

sympathetic (ergotropic) system is associated with tumor regression, whereas activation of the parasympathetic (trophotropic) system is associated with tumor enhancement". (p. 72.) Altogether, Pena's theory concerning the psychobiology of cancer, relates it to low arousal, i.e. associates it with extraversion in the personality expression of states of low arousal.

This article constitutes a very sketchy introduction to the two papers that follow, but as psychologists have not on the whole been very much concerned with the cancer and coronary disease literature, and the burgeoning literature on the relationship between these diseases and personality, stress and other psychosocial variables, it seemed desirable to set the scene for the two papers which follow. Readers concerned with the general field are advised to read the papers and books referred to, as this sketchy account cannot in itself be sufficient to cover this very broad, hotly debated and often acrimonious field.

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