PERSONALITY, CANCER AND CARDIOVASCULAR DISEASE: A CAUSAL ANALYSIS*

H. J. EYSENCK

Department of Psychology, Institute of Psychiatry, De Crespigny Park, Denmark Hill, London SE5 8AF, England

(Received 31 May 1985)

Summary—This study attempts to investigate the relationship between personality, stress and disease, particularly cancer and cardiovascular disease, both from the *correlational* and the *causal* points of view. It is concluded that there is good evidence linking these diseases with different patterns of personality, and that these links suggest a causal relation. Hypotheses are suggested as to the nature of these causal relations, and it is also suggested that these hypotheses can be used to good advantage in constructing effective methods of behaviour therapy, both prophylactic and curative.

1. COMPLEXITY OF THE DISEASE CONCEPT

Following Pasteur, physicians during the past 100 years or so have tended to view diseases as specific entities, with specific causes and specific remedies. Those who maintain that the 'cause' of a particular disease, such as a virus, might only be one element in a very complex causal chain, which included environmental factors (nutrition, alcohol intake, smoking, bad air etc.) and behavioural and personality factors, received short shrift until relatively recently. It is only now that the incredible complexity of the whole process, involving specific causes, immune reactions, environmental factors, personality and behaviour is being realized, and even now simplistic notions, such as the widespread belief that smoking causes cancer and cardiovascular disease, predominate. In this paper I wish to review some of the evidence regarding the relationship between personality, on the one hand, and cancer and cardiovascular disease, on the other, and cite some investigations which make it clear that the connection is a causal, and not merely a statistical one.

The notion of *psychosomatic disease* is certainly much older than the type of Freudian speculation that is now so popular, and which is sometimes credited with the origin of this notion. Wilkinson (1981) has recently discussed psychiatric aspects of diabetes mellitus, and quotes Willis (1684) to the effect that sadness, long sorrow and other depressions are responsible for this disease (p. 74), and later on Maudsley (1899) observed that "diabetes is sometimes caused in man by mental anxiety" (p. 113). (It is still unknown whether these observations of Willis and Maudsley represent anything other than speculation.) Galen as early as the second century A.D. proposed that personality played an important role in the formation of neoplasms. Fourteen anecdotal studies, linking severe emotional stress or loss and subsequent development of cancer appeared between 1701 and 1893 (Le Shan and Worthington, 1956). Other early studies are reviewed by Le Shan (1959).

Indicative of the complexity of the whole disease process is a study of Seltzer and Jablon (1977) in which they studied mortality rates among veterans. They found that although the mortality of privates was very close to expectation based on population rates, non-commissioned officers (NCOs) had a 23% advantage and commissioned officers about a 40% advantage. Standardized mortality rates for malignant neoplasms were 0.97, 0.86 and 0.70 for privates, NCOs and officers, respectively, while for cardiovascular-renal disease they were 0.88, 0.76 and 0.46. For ischaemic heart disease the ratios were 0.98, 0.88 and 0.50, and for all causes 1.00, 0.77 and 0.59. (On the

^{*}This is the text of the Presidential Address to ISSID, presented 21 June 1985 at the 2nd ISSID Conference, St Feliu Catalonia, Spain.

The author is indebted to R. J. Reynolds Tobacco Co. for a grant which made possible the preparation of the manuscript.

average these ratios are well below 1.00, which is the average for the whole population; the reason is that soldiers on the whole tend to have lower mortality rates.)

In part, but only part, the differences between privates and officers were correlated with education and socioeconomic status (SES); after correction for education, standardized mortality ratios were 0.91 for privates, 0.77 for NCOs and 0.73 for commissioned officers. Similar results have been found by Keehn (1974), and the relationship with education has been observed among others by Moriyama, Krueger and Stamler (1971) and Kitagawa and Hauser (1973). Everson and Fraumeni (1975) found very low mortality ratios for physicians and lawyers, and the Metropoliton Life Insurance Co. (1973) reported mortality ratios of about 0.80 for U.S. congressmen and New York state legislators.

Much smaller differences are reported from England and Wales (Registrar General, 1958), with an increase from 0.98 in Social Class 1 to 1.18 in Class IV. Indeed, for some particular causes of death the trend for social class was opposite in direction to that found by Selzer and Jablon; thus for ischaemic heart disease the largest mortality ratios in the English data are for Social Class I and the smallest for Classes IV and V.

As Sclzer and Jablon point out: beyond the probable role of socioeconomic factors, it may be speculated that the selection process for advancement in rank (and advanced appointment) is also affected by factors which may have some underlying biological basis. "Those biological factors which contribute to higher levels of performance and leadership may also be associated with greater longevity." (p. 565)

Neuroticism, for example, is one personality dimension which has been strongly linked statistically with physical illness (Barquero, Munoz and Jauregui, 1981), but as we will see other personality dimensions are also implicated. Explanations for observed relationships are not always easy to find. Thus for instance Totman, Kiff, Reed and Craig (1980) studied 52 volunteers who were given experimental colds by nasal inoculation with rhinoviruses during the course of a 10-day residential stay at a common-cold research unit. Daily examinations were carried out by the clinicians, making ratings of 14 signs and symptoms of upper respiratory tract infection, and 5 general signs and symptoms. Objective records were also obtained of virus infection.

Of the many measures used, only two were predictive of colds and severity of colds, namely introversion, and an index of recent life stress. These two indices were not correlated with each other and both were significant at the 0.001 level. It is not easy to see why introverts should develop colds much more readily than extraverts, and the obvious interpretation may be that extraversion is connected with a particularly well-functioning immune system.

The importance of personality factors in the causation of disease has been disguised in recent years by rather simple-minded assertions regarding the importance of factors such as smoking and diet. The notion that faulty diet was at the root of cardiovascular disease has finally been laid to rest (Mann, 1977; McMichael, 1979) and as the latter points out: "All well-controlled trials of cholesterol-reducing diets and drugs have failed to reduce coronary mortality and morbidity" (p. 173). Burch (1976) and Eysenck (1980) have similarly pointed out that the evidence relating to the effects of smoking on cancer and cardiovascular disease was insufficient to arrive at any final conclusions.

Of particular interest in this connection is the Multiple Risk Factor Intervention Trial (Multiple Risk Factor Intervention Trial Research Group, 1982). This was a randomized primary prevention trial to test the effect of a multifactor intervention programme on mortality from coronary heart disease (CHD) in 12,866 high-risk men aged 35 to 57 yr. Men were randomly assigned either to a special intervention (SI) programme consisting of treatment for hypertension, counselling for cigarette smoking and dietary advice for lowering blood cholesterol levels, or to the usual sources of health care in the community (UC). Over an average follow-up period of 7 yr, risk factor levels declined in both groups, but to a greater degree for the SI men. Mortality from CHD was not statistically different for the two groups, and total mortality rates were 41.2 per 1000 in the experimental group, and 40.4 per 1000 in the control group. This difference showing a higher death rate in the group which received the intervention treatment and advice was not significant, but it certainly goes counter to the hypothesis originally formulated. Clearly there is no evidence here that giving up smoking, changing the diet to lower blood cholesterol and taking pills for reducing hypertension had any effect on the health of the Ss of this experiment. Similar failures in other

studies designed to show the effects of giving up smoking on lung cancer (e.g. Rose, Hamilton, Colwell and Shipley, 1982) have singularly failed to show the desired effects (Eysenck, 1985). It would clearly be premature to assign a causal role to smoking and diet in the causation of lung cancer (or cancer generally) and cardiovascular disease.

There are reports that giving up smoking apparently reduces mortality (e.g. Doll and Peto, 1976), but in these studies the design embodies a fundamental assumption which has been shown to be in error. In the Doll and Peto study, for instance, doctors who gave up smoking were contrasted with controls who continued to smoke, the assumption being that prior to giving up smoking the group of quitters was equal to the group of non-quitters with respect to personality, health and other relevant variables. Friedman, Siegelaub, Dales and Seltzer (1979) have shown this assumption to be completely unjustified. In a large follow-up study they showed that even before giving up smoking, quitters were more like non-smokers than continuing smokers from the point of view of various health checks related to cardiovascular disease. Similarly Eysenck (1980) showed that from the point of view of personality structure, quitters were differentiated from non-quitters and resembled non-smokers. Clearly designs relying entirely on self-selection are fundamentally faulty, and cannot generate trustworthy results.

2. STRESS, PERSONALITY AND DISEASE

Stress is one environmental factor which has been linked with cancer, cardiovascular disease and many other medical disorders (e.g. Bammer and Newberry, 1981; Cooper, 1983; Dobson, 1982) and there has been much research on the relationship between stress, behaviour and the immune system (Borysenko and Borysenko, 1982). Weyer and Hodapp (1979) and Hodapp and Weyer (1982) make the important point that neither objective environmental variables nor certain personality characteristics alone cause stress; rather, a particular individual's evaluation of his environment is thought to be much more decisive in causing strain. "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, 1979, 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, but only in relation to the reaction to the stress of the organism cannot be emphasized too strongly; identical environmental factors can be stressful for one person, rewarding for another.

Psychological investigations have mainly centred on the question of predictability (Miller, 1981). Predictability is preferred to unpredictability under conditions of no distraction, but with distraction the preference reverses, and Ss clearly prefer unpredictability. With regard to anticipatory arousal, the existing data are highly consistent in that predictable aversive events usually increase anticipatory arousal. However, such investigations must always be qualified by the investigation of individual differences and personality. Duckitt and Broll (1982) found that extraverts appear to be significantly more tolerant of recent life changes than introverts, and Denney and Frisch (1981) found that neuroticism was related to the occurrence of disease as strongly as was life stress, but that neuroticism did not act as a moderator variable, so that the interaction term in the analysis of variance was insignificant.

Chan (1977) and Jenkins (1979) have published reviews of the literature, but most of the papers are of rather poor quality and deal with odd and unusual personality traits. Studies by Johnson and Sarason (1979), Myers, Lindenthal and Pepper (1975), Smith, Johnson and Sarason (1978) and Tyson (1981) do suggest that both introversion and neuroticism are correlated with life stress, as experienced, but they do not establish that they act as moderator variables. The quality of research design in this area needs to be much improved if any meaningful results are to be derived from the data.

One of the reasons for the unsatisfactory state of much of this research is the fact that the effects of stress can be contradictory and biphasic (Eysenck, 1983). Sklar and Anisman (1981) have shown in a review of the literature, that it is necessary to distinguish between acute and chronic stress, with the former *reducing* the effectiveness of the immune system, and promoting disease, while the

H. J. EYSENCK

latter may have the opposite effects. Eysenck (1983) labels this protective effect of chronic stress the 'inoculation effect', and discusses its relevance to personality and disease research in some detail. In a later contribution, Eysenck (1984a) has attempted to use the inoculation effect as a means for explaining the observed correlations between personality and lung cancer; we will return to this theory presently.

We must now turn to the study of the relationship between personality, on the one hand, and cancer and cardiovascular disease, on the other. Empirical studies have used one of three designs. In the first of these, patients suffering from a particular disorder are tested, and contrasted with patients suffering from different disorders, or a random sample of the population, usually the standardization group of the test in question. This design is clearly faulty in that the possibility is not ruled out that the disease itself, and the patient's knowledge that he has contracted this particular disease, may affect his mood and the way he fills in the personality inventory. If patients turn out to be more anxious and depressed than non-patients, this will hardly cause surprise, and the result cannot be interpreted as giving support to the hypothesis that the anxiety and the depression existed prior to the disease.

The second design is a little more sophisticated. As an example, consider the design used by Kissen and Eysenck (1962) in their study of male lung cancer patients. In this study, 116 male lung cancer patients and 123 non-cancer controls, both groups being patients at surgical and medical chest units, were tested *before* diagnosis. Thus the fears and anxieties of the patients would be equalized, all of them presumably being equally afraid of the diagnosis of lung cancer. The possibility still exists, of course, that the disease itself may directly influence personality, e.g. through hormonal or other secretions, but in most cases this is not a serious possibility.

The third design, and much the most preferable, is of course that of a *prospective* study, in which personality investigations are carried out on a healthy population. Follow-up over 10 and more years is then conducted to discover who dies, and of what illness. This method is of course much to be preferred to the others, but as we shall see rather similar results are obtained from all three methods.

Results obtained by any or all of these methods are of course correlational and cannot directly be presumed to indicate a causal connection. In order to prove a causal connection we have two alternatives. One is to discover the causal chain that leads from personality to disease, or resistance to the disease; attempts to do that in the case of the relationship between cancer and personality have been made by Eysenck (1985). The other method is to use behaviour therapy to alter the relevant behaviour of the person at risk, in the direction opposite to that manifested at an earlier point in time. Thus if type A behaviour is causally related to myocardial infarction, then therapy designed to alter the behaviour of the patient in the direction of type B behaviour might be expected to result in a reduction of cardiac recurrences in myocardial infarction patients, as compared with patients not subjected to this type of therapy (Friedman, Thorsen, Gill, Powell, Ulmer, Thompson, Price, Rabin, Breall, Dixon, Levy and Bourg, 1984). Similarly, if hopelessness and non-emotional behaviour are characteristic of the cancer patient, then using a special method of behaviour therapy to alter the patient's attitudes and behaviours in a direction opposite to that related to cancer should have a prophylactic effect, and in terminally ill patients should act in such a way as to prolong life (Grossarth-Maticek, Kanazir, Vetter and Jankovic, 1983a; Grossarth-Maticek, Schmidt, Vetter and Arndt, 1983b). Studies such as these, which will be reviewed in more detail later on, give powerful support to the view that personality and the behaviour associated with it are indeed causal factors in the disease process.

It will be useful, before now turning to a brief review of the literature, to state the hypothesis which has guided my own work. This hypothesis states, first of all, that the personality traits associated with cancer, on the one hand, and cardiovascular disease, on the other, are at opposite ends of certain personality dimensions, so that a person at risk for cancer would not be at risk for cardiovascular disease and *vice versa*. In the second place, the person at risk for cardiovascular disease is hypothesized to be *high* on the personality factors of neuroticism and psychoticism. These dimensions of personality are of course aggregates of traits which are empirically found to be correlated (Eysenck and Eysenck, 1985); the constituent traits for neuroticism and psychoticism would of course be implicated equally with the super-factors. Thus impulsivity, which is a

constituent trait of psychoticism, would be hypothesized to be positively correlated with cardiovascular disease, and negatively with cancer.

In the third place, it is hypothesized that extraversion would be positively correlated with cancer. and negatively with cardiovascular disease. However, this prediction, to even a greater extent than the previous ones, has to be qualified by the fact that both cancer and cardiovascular disease cannot be assumed to be unitary disorders, but are groups of disorders not all of which may correlate with personality in the same direction. Thus Floderus (1974) has hypothesized that angina pectoris and hypertension are correlated with neuroticism and introversion, but myocardial infarction and hyperlipidemia are correlated with neuroticism and extraversion. In the case of cancer, cancer of the cervix has been associated with promiscuous behaviour, characteristic of extraverts (Eysenck, 1976); this may be quite specific to cancer of the cervix appearing in promiscuous women (Booth, 1969). Gagnon (1950) found that the incidence of carcinoma of the cervix was almost negligible in nuns, and Lombard and Potter (1950), in a large control study, found strong correlations between cervical cancer and such factors as marriage before the age of 20, divorce and separation, unrepaired cervical lacerations and syphilis. The question is an empirical one, and needs detailed investigation. It should always be remembered that generalization from one specific disorder to cancer in general may not be justified, and that angina pectoris is not identical with myocardial infarction.

The hypotheses in question cannot pretend to be based on very firm ground. They originated from observations made over thousands of years by outstanding physicians, starting with the Greek physician Galen (A.D. 131–201), and continuing until modern times. As an example, consider W. H. Walshe, who in 1846 published a book entitled *Nature and Treatment of Cancer*, in which he claimed that there seemed to be general agreement that "women of high colour and sanguinous temperament were more subject to mammary cancers than those of different constitutions". The description of the ancient 'temperament' of the sanguine personality is very similar to that of the stable extarvert (Eysenck and Eysenck, 1985), and would be in accord with the hypothesis that cancer was related to lack of neuroticism and to an extraverted temperament. It should of course be remembered that Walshe was speaking of cancer of the breast only, and of women; even if he were correct in his assumption, it would not necessarily follow that other types of cancer, either in males or females, would be related to the same personality structure. This is not the place to go into a detailed analysis of the historical origin of these ideas; we are here concerned more with the empirical evidence now available to support the hypothesis stated above.

3. CANCER AND PERSONALITY

Let us begin with cancer. A good starting point may be the study by Kissen and Eysenck (1962), which was specifically designed to test the hypothesis that neuroticism was negatively correlated with cancer. The design has already been presented; it should be added that patients in the control group were subdivided into age groups before a comparison of their scores was made, and that patients were also subdivided into those with and without psychosomatic disorder. As regards extraversion, there were no differences between cancer and control patients without psychosomatic disorders, but, in comparing the groups with psychosomatic disorder, it was found that the cancer group was considerably more extraverted than the control group. As regards neuroticism, the control group had much higher N scores than the cancer groups (cancer and control) had somewhat higher N scores than did the non-psychosomatic groups. Smoking did not account for these differences.

Kissen followed up our early findings in a whole series of studies (Kissen, 1963a, b, 1964a-c, 1966, 1967, 1969; Kissen, Brown and Kissen, 1969), and Kissen and Rao (1969) also looked at biochemical factors in lung cancer patients during the prediagnostic period. The main feature of his work subsequent to our joint study was that lung cancer patients had very significantly lower N scores than did other patients, prior to diagnosis. He calculated lung cancer mortality rates per 100,000 men aged 25 and over by levels of N scores, and found that people with very low scores had a mortality rate of 296, those with intermediate scores had a mortality rate of 108, and those

with very high scores had a mortality rate of only 56! Thus very low scorers on N have about a six-fold probability of developing lung cancer as compared with very high scorers.

While there seems to be little doubt that the hypothesis of a negative correlation between lung cancer and neuroticism is borne out, the interpretation is by no means obvious. Kissen made the assumption that cancer susceptibility was related to *repression* of emotion (poor outlets for emotional discharge), while my own interpretation was rather the *absence* of strong emotional reactions. There is no evidence in the literature for the psychoanalytic interpretation made by Kissen of our findings, and I will return in some more detail to this in the next section.

A more recent study by Berndt, Gunther and Rohte (1980; see also Eysenck, 1981) compared control groups of patients with patients who after completion of the questionnaire were found to suffer from breast cancer or bronchial carcinoma. The size of the female control group was 953; that of the breast cancer group was 231. The male control group numbered 195, and the male bronchial carcinoma group 123. The female bronchial carcinoma group was very small, numbering only 20, which makes it almost impossible for this group to give significant differences from the controls. In all three groups the cancer patients had N scores *lower* than the controls, with the differences reaching a 1% level of significance for the breast cancer group, and the male bronchial carcinoma group; for the female bronchial carcinoma group, because of the small number of patients, the result, although in the same direction, was not statistically significant. We may conclude that the Berndt investigation gives results essentially identical with those of the Kissen and Eysenck study, as far as neuroticism is concerned; there were no significant differences for extraversion in this study.

In an earlier prospective study, Hagnell (1966) reported on the results of an epidemiological survey of the 2515 habitants of two adjacent rural parishes in the south of Sweden, using a 10-yr follow-up and relating the subsequent history of each S with his personality scores on an interview carried out at the beginning of the study. Hagnell used the Sjöbring personality scales, which have been found to be significantly related to the three major dimensions of personality recognized by Eysenck. Interpreting Hagnell's interview ratings, it seems that a significantly high proportion of women who had developed cancer had been originally rated as extraverted (characterized by warmth, heartiness, concreteness, being interested in people and sociable).

Similar results were obtained by Coppen and Metcalfe (1963); diagnoses of patients were obtained after they had filled in the questionnaires. Forty-seven patients had a malignant tumour; 32 had cancer of the breast, 4 cancer of the uterus and 11 had cancer in other parts of the body. Two control groups were used, and care was taken that these should fall into the same age group as the patients with cancer. The cancer group had significantly higher E scores in both control groups, but the N scores did not differ significantly. The subgroups of cancer patients all had very similar means. Greer and Morris (1975) reported another interesting replication of the Kissen and Eysenck study, but used breast cancer cases instead of lung cancer cases. A consecutive series of 160 women at a hospital for breast tumour biopsy was studied by means of detailed structured interviews and standard tests, both interviews and tests being conducted on the day before exploratory operation without provisional knowledge of the diagnosis. The principal finding was a significant association between the diagnosis of breast cancer and the behaviour pattern, persisting throughout adult life, of abnormal relief of emotion. "This abnormality was, in most cases, extreme suppression of other feelings. Extreme expression of emotions, though much less common, also occurred in a higher proportion of cancer patients than controls." Greer and Morris found no correlation with extraversion. In a later paper, Morris, Greer, Pettingale and Watson (1981) again found the mean N scores were significantly lower for cancer patients as compared with controls. Greer, Morris and Pettingale (1979) found that survival 5 yr after the diagnosis of breast cancer was significantly related to psychological traits assessed at 3 months. Women considered on the basis of a structure interview to show a fighting spirit had better prognosis than those displaying stoic acceptance or helplessness and hopelessness. Similarly, Derogatis, Abeloff and Melisaratos (1979) found that women with breast cancers who survived more than 1 yr had higher ratings on measures of hostility and anger than those who died within the first year; these findings suggest a negative correlation between cancer and psychoticism.

A recent study by Dattore, Shantz and Coyne (1980) found the cancer group, as compared with the control group, showing more repression and less report of deep depression, as well as less hysterical reaction. Watson and Schuld (1977) reported results not in accord with the hypothesis, but the population used by them consisted of Ss all of whom had psychiatric diagnosis at the time of testing, and hence would not be comparable with the normal populations studied by all other researchers.

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. Those dying in less than 2 yr, as compared with those dying after more than 6 yr, had higher depression scores and lower neurotic outlet scores, as well as very low acting out scores at the time of the first assessment.

Blohmke, Engelhardt and Stelzer (1984) have reported another study comparing lung cancer patients with non-cancer controls, on a large scale. The most important difference between the groups was 'lack of nervousness', characterizing cancer patients, followed by 'positive social conformity' and 'no external control'; these are all in the direction predicted from the Kissen and Eysenck study. Two further differences were in the direction of greater extraversion for cancer patients; they showed more changes in the conditions of life, and had more subjective complaints.

In another study, Butler, Regelson, Lawlis and Bristow (1982) used the Cattell 16PF scales, and concluded that: "The research study supports the general hypothesis that cancer patients are homogeneous in measurable personality patterning, and show different personality correlates from normal populations. The data suggests that the samples were not similar to other mean profiles, such as cardiac patients or psychosomatic sample." (p. 20) It is difficult to see whether in detail the study agrees with the Kissen and Eysenck results as the subscales of the 16PF are difficult to interpret psychologically (Eysenck and Eysenck, 1985). In an interesting study of psychosocial risk factors for lung cancer, Horne and Picard (1979) found that psychosocial factors were 1-2 times as important as smoking history in predicting diagnosis of cancer; their Psychosocial scale, including items relevant to personality and to stress, correctly predicted the diagnosis of 80% of the 66 individuals with benign disease and 61% of the 44 with malignant disease, resulting in a probability level of less than 0.0001. An interesting study was reported by Stavrakay (1968), studying 204 cancer patients including 83 patients with breast cancer; he related their subsequent duration of life to the initial MMPI scores, and found that the group with the most favourable prognosis had been evaluated on projective tests as displaying strong hostile drives without loss of emotional control. This emphasis on hostile drives relates to psychoticism, of course, although the reliability and validity of projective devices is such as to render the conclusion rather doubtful.

There are many other studies, reviewed by Eysenck (1980, 1985), Crisp (1970) and others which are marginally relevant to our discussion, but which would unduly extend our survey without adding anything of significance. One curious feature which recurs throughout the set of studies reviewed is the presence of depression and hopelessness in future or actual victims of cancer, usually related to stressful life events. At first sight it may seem odd that anxiety and neuroticism in general seems to *protect* the individual against the onset of cancer, and to prolong life once cancer is diagnosed, while depression has the opposite effect. Normally depression and anxiety are found correlated, but of course the correlation is not perfect, and we will return to this point presently.

Psychoticism as such has not been investigated in relation to cancer, but there is a large literature, surveyed by Eysenck (1980, 1985) demonstrating that, in the words of Bahnson and Bahnson (1964a) we might consider that "Cancer is an alternative to psychosis", although elsewhere (Bahnson and Bahnson, 1964b) they also find some support for the theory that denial and repression of primitive impulses and of disturbing emotions is found most frequently in patients with malignant neoplasms. Roughly speaking, the ratio of deaths by cancer in schizophrenic patients as compared with non-patients is in the neighbourhood of 1:3, a disproportion not to be accounted for in terms of smoking. It would seem urgently desirable to carry out a study involving the P scale of the EPQ in order to discover to what extent the relationship with cancer is mediated by personality alone, and to what extent such factors as hospitalization, psychiatric disease processes and iatrogenic factors may play a part in the observed relationships.

4. PROSPECTIVE AND CURATIVE STUDIES

Perhaps the most impressive and important study to demonstrate the relevance of personality

factors to the incidence of cancer is the work of Grossarth-Maticek (1980; Grossarth-Maticek. Sigrist and Vetter, 1982b; Grossarth-Maticek, Kanazir, Schmidt and Vetter, 1982a, 1985a; Grossarth-Maticek, Frentzel-Beyme and Becker, 1984; Grossarth-Maticek, Bastiaans and Kanazir, 1985b). These papers report a completed 10-vr follow-up study in Yugoslavia, and two still ongoing follow-up studies in Heidelberg, Germany. The sample in the Yugoslav prospective study consisted of 1353 Ss; they were recruited by selecting the oldest person in every second house in a small Yugoslav town with a population of 14,000 people. Most of the Ss were between 50- and 65-yr old. Psychosocial data were recorded using a questionnaire and an observation catalogue. Height, weight, blood pressure and data on cigarette smoking were also collected, and further medical information was recorded periodically between 1969 and 1976. Ten years after starting the study, a physician assessed the occurrence of different diseases in the sample, and also recorded diagnoses on the death certificates. A total of 117 men and 87 women had developed cancer over this period; cancer of the lung, stomach, rectum and prostrate predominated amongst males, while breast, uterine and cervical cancer occurred in 69% of females. The Heidelberg replication of this study used a cross-sectional analysis of a random sample of 1026 Ss. This design is clearly superior to those discussed above, and avoids most of the criticism made of work in this area by Morrison and Paffanbarger (1981).

The major outcome of the Grossarth-Maticek study is shown in Table 1, indicating the determination of cancer and cardiovascular incidence in the form of observed and expected deaths. Figure 1 shows that the highest correlations relate to 'rational and anti-emotional behaviour', i.e. the obverse of neuroticism (0.41), and the number of traumatic life events evoking chronic hopelessness (0.43). Thus these data are in agreement with a majority of studies summarized in this paper, and powerfully reinforce the message that *neuroticism is negatively correlated with cancer*. Note also the positive correlation with cancer incidence of the absence of psychopathological symptoms such as anxiety, and the lack of positive emotional contact. (Actually the figures in Fig. 1 are standardized, partialled regression coefficients, while the error term represents the square root of the residual variance of cancer incidence. All the predictors included in Fig. 1 are statistically significant at the 0.01 level.)

Raw correlations are given by Schmidt (1984), who has calculated both linear correlations and η -coefficients. For chronic helplessness, these are 0.59 and 0.60; for rational and anti-emotional behaviour, 0.51 and 0.60.

	Low score		High score	
	Observed	Expected	Observed	Expected
Lung cancer	0	26	38	12
Other cancer	8	84	120	44
Ischaemic heart disease	38	78	77	37

Table 1. Observed and expected occurrence of disease in low and high scorers respectively on the Grossarth-Maticek rational-anti-emotional scale

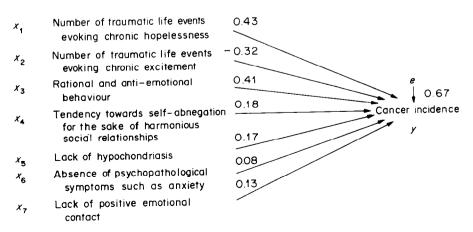


Fig. 1. Determinants of cancer incidence (Grossarth-Maticek et al., 1984).

Similar figures are found in the Heidelberg prospective studies, but as these are not completed it may be best not to give provisional results. It is however interesting to note that Grossarth-Maticek calculated χ^2 -values comparing the predictive effectiveness of smoking and his personality inventory. For lung cancer, the respective figures are 68.8 for smoking, 84.1 for personality. For ischaemic heart disease, they are 3.4 and 70.4. Clearly personality is a more important variable than smoking as far as lung cancer and cardiovascular disease are concerned; for all cancers the discrepancy is much larger than for lung cancer. Grossarth-Maticek (1980) has also presented evidence to show that these are *synergistic* effects, in the sense that the effects of smoking are disproportionally greater in those people whose personality predisposes them to cancer.

This conclusion may be vitiated by the well-known fact that lung cancer is much more readily diagnosed in smokers than in non-smokers (Feinstein and Wells, 1974). These authors looked at 654 patients who were diagnosed after necropsy as having died of lung cancer. In the series, they studied the relationship between the rate of non-diagnosis during life and the amount of antecedent cigarette smoking. In patients whose history of cigarette smoking was unknown, or who had not smoked, the rate of non-diagnosis was 38%; this dropped to 20% among the light smokers, 14% in the moderate smokers and 10% in heavy smokers! They also showed that this pre-mortem detection gradient was related to the intensity of diagnostic examinations received during life by their patients. For instance, the Papanicolaou cytologic examination, or Pap smear (of the sputum) was asked for far more frequently in smokers than in non-smokers (over 80% as compared with 56%). Thus a correlation with smoking might very easily have been a function of artifacts of this kind, as well as others discussed in greater detail elsewhere (Eysenck, 1985).

At a lecture at which I described the significantly greater predictive importance of personality as compared with smoking in the genesis of cancer and cardiovascular disease, one listener proclaimed that he would give up personality! This being difficult, the possibility remains that by changing a person's behaviour pattern in the direction opposite to that characteristic of the cancer-prone personality, behaviour therapy may be able to be useful in a prophylactic fashion, or to prolong life even after incurable cancer has been diagnosed. Both these possibilities have been investigated experimentally by Grossarth-Maticek (Grossarth-Maticek et al., 1983a,b). The first experiment to be considered is a life-preserving one. One hundred and seventy-nine women with breast cancer and visceral metastases were the subject of the experiment. Most of these accepted chemotherapy, while 56 declined it. Seventeen refused psychotherapy, and the rest accepted. Three different methods of psychotherapy were available. The first was behaviour therapy, where an effort was made to change undesirable behaviours into desirable through a process of imagery and desensitization. The second was psychotherapy of a Freudian kind, and the third was 'creative novation therapy', developed by Grossarth-Maticek as a form of behaviour therapy specially concerned with altering cancer-prone behaviours of the kind discovered in his follow-up studies. We thus have four groups, respectively with and without chemotherapy, and with and without psychotherapy. The criterion is the length of survival. Lowest was the group receiving neither type of therapy (mean survival = 11.28 months). Best was a group receiving both (22.40 months). Those receiving only one or the other showed a mean survival length of about 14.50 months. The two types of therapy seemed equally successful in prolonging life, but the three forms of psychotherapy were not equivalent in their effect (see Table 2).

For psychotherapy, mean survival length was lowest (12.83 months). Behaviour therapy was better (15.29 months), but creative novation therapy was far and away the best (23.54 months).

Chemotherapy		•	m		
Psychotherapy	No	Yes	Totals		
No	Mean = 11.28 $N = 25$	Mean = 14.08 $N = 25$	Mean = 12.68		
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 2. Survival (in months) of terminally ill cancer patients receiving or not receiving chemotherapy and psychotherapy

Thus procedures based on changing behaviour patterns of a cancer-prone personality type can apparently successfully prolong life in a highly significant manner.

In the second experiment, four groups of psychotherapy patients were compared, both with each other and with no psychotherapy controls. Three groups received creative novation therapy from therapists with different degrees of experience. The fourth group received psychotherapy of the traditional kind. The mean survival time in months, comparing each of the groups with their respective controls, was 9.39 for the creative novation therapy group treated by the most experienced therapist, 6.77 for therapists intermediate in experience and 4.84 for therapists with the least experience. The other psychotherapy group had a mean difference of -14.20 months; in other words Freudian psychotherapy actually has a *negative* effect on survival! (The differences between novation therapy groups due to experience of the therapist are not statistically significant, but are highly suggestive.)

Of possibly even greater importance and interest is a prophylactic study carried out by Grossarth-Maticek, involving 91 high cancer-prone Ss from his Heidelberg study; 45 of these were treated by means of novation behaviour therapy, while the other 46 received no therapy. To date, 12 patients in the control group have died of cancer; none in the experimental group. The difference is significant at the 0.001 level, and suggests strongly that behaviour therapy of this particular type can very successfully change behaviour patterns in such a way as to obviate the occurrence of cancer even in highly cancer-prone Ss. If this study could be replicated, then clearly we would have here a direct proof of the causal relationship between personality and cancer. (Also reported was a similar study of cardiovascular and control patients, with the former treated by novation behaviour therapy. The cardiovascular group was made up of 82 Ss, of whom there were 3 deaths from cardiac illness in the treatment group, and 14 in the control group. There were 5 and 6 deaths respectively from other causes, leaving 34 treatment patients and 20 no-treatment patients alive, a different significant at the 0.0090 level.)

5. PERSONALITY AND CARDIOVASCULAR DISEASE

This terminates our discussion of the cancer-personality relationship, and we must now turn to a discussion of the relationship between personality and cardiovascular disease. This will begin with a brief discussion of hypertension, then go on to a variety of studies using various personality factors, and finally turn to a discussion of the well-known 'type A-type B' theory of Rosenman and Friedman (Rosenman and Chesney, 1980; Steptoe, 1981). There is no doubt that hypertension is regarded by many as a psychosomatic disease (Alexander, 1950; Weiner, 1977), and being associated with cardiovascular disease it may deserve a brief discussion here. Weyer and Hodapp (1977), using the *Freiburger Persönlichkeitsinventar*, found no support for the 'suppressed hostility hypothesis' of hypertension; the data agree more with the hypothesis that unspecific dysthmyic personality traits are more often associated with psychosomatic complaints. While the corresponding associations were statistically significant, they were judged to be unimportant for the individual case.

In another study by Baer, Collins, Bourianoff and Ketchel (1979); a 16-item self-report instrument was used. They found evidence both for hostility and anxiety, and concluded that hypertensive individuals experienced intense arousal of negative emotions (Esler, Julius, Zweifler, Randall, Harburg, Gardiner and De Quattro, 1977).

Kopp (1984) and Kopp and Koranyi (1982) conclude that the majority of Ss in the first, mild stage of hypertension are characterized by tonic, sympathetic, ergotropic dominance, a low initial skin resistance level, high pulse rate and high integrated surface EMG values. Others, such as Baumann, Ziprian, Gödicke, Hartrodt, Neumann and Lauter (1973), De Champlain, Causineau, Van Ameringen, Marc-Aurel and Yamaquelin (1977) and Hodapp, Weyer and Becker (1975) report similar results.

While there appears to be a good deal of agreement, there are possible artifacts which have mostly been disregarded by these authors. Berglund, Ander, Lindström and Tibblin (1975) have indicated that the frequency of untreated hypertensives may result in the study of an unrepresentative group of complainers, and Costa, McCrae, Andres and Tobin (1980) have argued that personality may not be predictive of hypertension, but that anxiety and the other symptoms may be the product of hypertension. In one of the few follow-up studies in this area, they have given much support to this view. Thus in spite of the fairly unanimous agreement on the importance of dysthymic factors in hypertension (Kidson, 1971; Lewinsohn, 1956; Sainsbury, 1960), we must conclude that artifacts of the kind indicated may be responsible for the observed relationships.

We must now turn to coronary heart disease (CHD) or coronary artery disease (CAD). As in the case of cancer, there is a long history of association with personality, but in modern times it is perhaps Osler (1910) who drew attention to this connection when he suggested that it was not the "delicate neurotic person who was prone to angina, but the robust, the vigorous in mind and body, the keen and ambitious man, the indicatory of whose engine was always at full speed ahead". This suggestion was later incorporated in the famous 'type A' coronary-prone behaviour pattern which has dominated research in this area (Rosenman and Chesney, 1980). Before turning to this concept, however, a number of other approaches may be mentioned. These studies are not unrelated to the notions embodied in the type A theory, but are often similar to it.

Dunbar (1943) describes the coronary patient as being an excessively strenuous worker who went many years without vacation under stress and strain; he was prone to stick to one job for many years, and was marked by compulsive or depressive neurotic tendencies. He was characterized by a tendency to completion of scholastic courses, tasks etc. Gildea (1949) also stressed the unremitting compulsive compensatory work habits of coronary patients, whose planned careers led frequently to executive and managerial positions. They also showed contrasted traits of insecurity and inferiority, results not confirmed by Storment (1951). Pecte (1955) noted impatience, anxiety, conscientiousness and driving qualities which led to long hours, little recreation, poor sleep and immoderate dietary habits; he also noted paradoxical traits of aggression and mildness. Miles, Waldvogel, Barrabee and Cobb (1954) found patients less introspective and more capable of the free expression of aggression. In a follow-up study, Thomas (1976) found a group of coronary occlusion patients who had higher scores for depression and nervous tension, as well as anxiety and anger, than patients with malignant tumours.

Van Dijl (1979) tested the hypothesis that myocardial infarction (MI) patients are more sociable than controls, and found evidence for this proposition in three different samples. This would seem to link MI with extraversion; this finding replicates the conclusion by Bendien and Groen (1963) who found that patients with MI were significantly more extraverted than control patients. Floderus (1974) suggested the hypothesis that while both the two major clinical manifestations of CAD, MI and angina pectoris (AP) are characterized by emotional instability (neuroticism), MI develops in extraverted Ss, AP and hypertension in introverted Ss.

Blumenthal, Thompson, Williams and Kong (1979) found that patients with a history of MI were significantly *less* anxious than patients without such a history. They suggested that high anxiety levels may serve a protective function, and that anxiety in patients without a history of MI may be an important factor in the decision to have them referred for diagnostic coronary angiography.

Rime and Bonami (1979) found some evidence that CHD patients scored higher than control Ss for passivity, dependence and impulsiveness, as well as being more ego-defensive and self-assertive. Siltanen, Lauroma, Nirkko, Punsar, Pyorala, Tuominen and Vanhala (1975) in a partly prospective study found CHD to be correlated with anxiety, aggressivity, seclusion, defensiveness and carefulness.

In a prospective study, Pleszewski (1977) studied 61 patients with MI and 20 patients with CHD without MI. The former group was characterized, prior to infarction, by strong tendencies to success, chronic emotional tension, hard-driving (activity, haste, impatience, tension) and high tolerance for negative affect. It was also suggested that high emotional excitability co-existed with strong self-control. "Patients with efficacious self-control had sudden myocardial infarction; on the other hand patients with ineffective self-control had angina pectoris before infarction."

In a follow-up study of 255 medical students who had completed the MMPI while in medical school, Bareford, Dahlstrom and Williams (1983) found high levels of hostility associated with increased levels of arteriographically documented coronary atherosclerosis. A survey by Williams, Bareford and Shekelle (1984) suggests strongly that high scores on the Hostility scale of the MMPI are associated cross-sectionally with prevalence of CAD and prospectively with risk of CHD and with total mortality. They suggest that the psychological construct which is assessed by the Hostility scale can be described by the term cynicism, an attitudional set that stems from an

inadequately developed sense of basic trust and centres around beliefs that other persons are generally mean, selfish and undependable. They suggest, rather speculatively, that the biological pathways whereby the psychological characteristic of hostility/cynicism may be translated into disease processes could involve excessive secretion of testosterone during vigilant observation of others and excessive secretion of catecholamines and cortisol during the experience of anger, both of which would be expected to be more intensive and frequent in hostile/cynical people. The similarity of these traits to psychoticism is obvious. Also indicative of the importance of hostility and anger-in for both AP and MI is the work of Dembroski, MacDougall, Williams, Haney and Blumenthal (1985), analysing in detail the type A structured interview to determine what elements of the multidimensional type A pattern are related to CHD severity in a selected group of patients with minimal or severe CAD.

The works just quoted deal essentially with Hostility scores for young men; of older men, Ibrahim, Jenkins, Cassel, McDonough and Hames (1963) have suggested a causal relation between *repressed* hostility and CHD. Shekelle, Ostfeld, Lebovitz and Paul (1970) fail to find *any* relationship in the application of Ibrahim's methods to the longitudinal data. Kantor and Robertson (1977), using data from a study by McDonough, Hames, Stulb and Garrison (1963), attempted to reanalyse various published data in terms of a modification of the MMPI scales, and found some positive support for the hypothesis. The difficulty here, of course, is the same as that attending Kissen's attempt to link cancer with *repressed* anxiety; repression is inferred on the basis of an inadequate methodology.

The notion that anxiety/neuroticism/emotional instability precedes AP and MI has been strongly criticized by Costa (1985), Costa, Fleg, McCrae and Lakatta (1982), Medalie, Snyder, Groen, Nuefeld, Goldbourt and Riss (1973) and Ostfeld, Lebovits, Shekelle and Paul (1967). As Costa points out, MI is objective in a way that AP, being characterized mainly by subjective chest pain symptoms, is not. Failure to distinguish between subjective indicators (e.g. chest pain symptoms) and objective indicators (e.g sudden death, transmural MI, complete stenosis or occlusion of coronary arteries) is responsible for conflicting and confusing research results. This suggestion is strongly confirmed by the work of Ostfeld and his colleagues (1967) in a prospective study of 1885 males. They contrasted the personality scores of men who had and those who had not developed CAD, and between those who developed only AP and those who had only MI. N scores on various scales were elevated in men before the development of AP, but men who were to develop MI were not different from those without CHD on any of the scales.

In a similar prospective study by Medalie and his colleagues (1973), the incidence of AP was twice as great among men scoring in the top half of the scale on a 3-item index of anxiety, compared with those in the bottom half. The anxiety score was not predictive of MI, however. In the study by Costa and his colleagues, the individuals in the CAD-with-angina group, who most clearly suffered from heart disease, did not differ from disease-free controls in antecedent levels of neuroticism.

Keehn, Goldberg and Beebe (1974) followed-up 9000 psychoneurotics and 9000 controls over a 24-yr period, and reported that there were no differences in CAD-related mortality between the two groups. Equally clear-cut is a finding from a study by Elias, Robbins, Rice and Edgecomb (1982), who asked a group of 136 men and women scheduled for arteriography to complete several psychological measures of anxiety, depression and traumatic complaints. When degree of maximum stenosis was correlated with the psychological measures, a significant *negative* correlation was found; the more anxious, depressed or concerned with somatic complaints the individual was, the healthier his or her coronary arteries! In a similar way, Blumenthal *et al.* (1979) failed to find a significant association between neuroticism and degree of stenosis, as have Zyzanski, Jenkins, Ryan, Flessas and Everist (1976). All these data seem to contradict the consideration of neuroticism as a risk factor for CAD (Jenkins, 1978).

Bass and Wade (1982) studied 99 patients who underwent coronary arteriography for the investigation of chest pain, and were interviewed by the use of the Bortner Type A Questionnaire. The 26 men with normal and minimally diseased arteries had significantly higher mean type A scores than the 41 men with important coronary occlusions. There was no significant association between type A scores and the extent of coronary disease. The suggestion that type A behaviour is more indicative of hypochondriasis than of CHD is also borne out by a study reported by Ahnve,

De Faire, Orth-Gomes and Theorell (1979) who found that among men admitted to a coronary care unit because of chest pain those who were subsequently shown to have no evidence of ischaemic heart disease had significantly higher type A scores than did those who had genuine infarction in the control group.

Spielberger (1976) also failed to find differences in anxiety state or trait between 84 cardiac patients and 320 other patients; 111 neuropsychiatric patients, however, had very much elevated state and trait anxiety scores. The fact that coronary-prone behaviour breaks up into different parts when related to MI and AP cases is indicated in a paper by Jenkins, Zyzanski and Rosenman (1978), who followed-up a cohort of 2750 healthy men in a prospective study, after 4 yr, 67 of these sustained acute MIs, 30 were discovered by ECG to have had MIs, which had gone clinically unrecognized, and 23 had developed classical AP without ECG changes indicative of infarction. Thus the term 'cardiac heart disease' is too inclusive for proper scientific study. Ostfeld *et al.* (1967) in a prospective study, found that prospective AP patients were differentiated from normal control or infarct patients by having high scores on the Hysteria and Hypochondria scales of the MMPI, i.e. scales characteristic of extraversion. Brozek, Keys and Blackburn (1966), in another prospective study, also found that future AP patients were characterized by high Hysteria and Hypochondria scale scores; angina patients differed from infarct patients by having higher N scores.

Ideally questionnaire and rating studies of emotionality-neuroticism should be supplemented by psychophysiological investigations of laboratory stress. Krantz and Manuck (1984) have reviewed the partial and contradictory evidence on the relationship of acute psychophysiological reactivity to risk of cardiovascular disease, and conclude that "reactivity to stress is a construct with multiple dimensions: Different tasks and situations appear to elicit different patterns of physiological responses" (p. 435). When it is realized that different personality types react differently in identical situations and tasks, the complexity of the situation will become clear. "Reactivity *per se* should currently not be regarded as a proven risk factor", Krantz and Manuck (1984, p. 435) point out; using a similarly high level of proof, one can only conclude that in this respect reactivity does not differ from the usual epidemiological factors like smoking, where also a causal connection is far from proven (Eysenck, 1980, 1985).

6. TYPE A-TYPE B PERSONALITY

The concept of type A behaviour (Friedman and Rosenman, 1974) takes up many of the themes dealt with in the last few pages. The literature on this concept is now immense (Steptoe, 1981; Price, 1982; Weiss, Detre and Cooper, 1981); Dembroski, Schmidt and Blümchen (1983) and Dembroski, Weiss, Shields, Haynes and Feinleib (1978) review much of the literature. Here we can only give a very abbreviated account of this typology.

The notion of a coronary-prone type A personality includes a number of traits such as sustained aggression, ambition, competitiveness and a chronic sense of time urgency, as well as impatience, an intense commitment to occupational goals and behavioural alertness. Contrasting with this are the more relaxed people who do not display these features and who are labelled type B. Intermediate types, identified by subscripts, have also been used. A person's type is assessed from global impressions of performance in a structured interview (SI), designed to evoke type A responses, and a number of questionnaires have also been developed in an attempt to pin down the rather elusive personality traits in question.

There is a great deal of evidence that type A behaviour is characteristic of patients with CHD, and that such behaviour is predictive of future CHD. In addition, Friedman *et al.* (1984) have shown that type A behaviour can be changed by behaviour therapy, and that in patients so counselled the cumulative cardiac recurrence rate was 7.2% over a 3-yr period, as compared with a 13% recurrence rate in participants who only received cardiologic counselling. It is difficult to avoid the conclusion that contained in the rather vague notion of 'type A behaviour' there is buried an important biosocial concept intimately linked with CHD.

Nevertheless, there are a number of criticisms that have to be made of the concepts (Eysenck and Fulker, 1983). In the first place, the notion revives the idea, long rejected by psychologists, of a non-continuous typology, or at least of a bimodal distribution; there is no evidence for such a typology. A better representation of reality is a continuum with a normal distribution of scores from one extreme to the other.

In the second place, the typological way of conceiving and speaking of this continuum draws attention away from the question of dimensionality; it seems clear from the factor analytic and correlational studies that have been carried out that we are not dealing with a single dimension, but with several. It is important to identify these different dimensions, and to decide which are causally related to CHD. Rim (1981), using the Bortner (1969) Type A Questionnaire, found type A to correlate with N and E on the Eysenck Personality Questionnaire. A study by Pichot, de Bonis, Somogyi, Degre-Coustry, Kittel-Bossuit, Rustin-Vandenhende, Dramaix and Bernet (1977) also shows a positive correlation of the Bortner scale with Eysenck's N and E factors, as did Eysenck and Fulker (1983), who found 'type A' to be correlated with N and E.

In the third place, the literature suggests very strongly that questionnaire studies do not correlate highly with the SI, and are much less predictive of cardiovascular disease, if they are predictive at all. Dembroski and McDougall (1985) have suggested that only certain components of the global type A construct are related to coronary risk, and that these are more closely related to interview behaviour than to questionnaire responses. The most important variables may be: (1) non-verbal paralinguistic stylistics of loud, explosive, rapid and accelerated speech and latency of answers; (2) factor analytically derived self-report measures of time pressure, hard-driving behaviour, speed of activity, competitiveness and impatience; and (3) the attitudinal and behavioural dimensions of potential for hostility, anger-in and verbal competitiveness.

The difference between interview and questionnaire measurement of type A behaviour extends into the field of heritability. Rahe, Hervig and Rosenman (1978) found no evidence of heritability for interview ratings, but did find such evidence for questionnaire measurement. Theorell, de Faire, Schalling, Adamson and Askevold (1979) also found evidence for genetic determination in questionnaire and physiological measures, and Eysenck and Fulker (1983) found questionnaire measures of type A significantly determined by genetic factors. There is much room for further work in this field; it seems highly unlikely that the traits measured by the interview procedure are in fact not determined genetically in any way. The fact is that the number of twin pairs used was small, so that while MZ twins were always more similar than DZ twins, these differences did not achieve significance as they might have done with larger numbers.

The rather nebulous compound of qualities constituting the traditional type A behaviour indicates the fourth criticism to be made, namely that there has been little effort to define type A behaviour in terms of traditional concepts in the field of personality study, and relate it to these dimensions. Eysenck and Fulker (1983) have shown that questionnaire studies of type A behaviour are strongly related to neuroticism and extraversion, but the more important SI behaviour has not been so related to traditional personality dimensions.

The last comment and criticism of the type A behaviour pattern is that it seems to apply, particularly in its predictive aspects, only to middle-class white Americans; it does not seem to be predictive for blue-collar workers, or blacks, and there have been difficulties in using it with female Ss also. It is possible that changes in the measurement procedures may alleviate these difficulties, but at the moment they are very real. Of the many studies in this field, only a few can be mentioned here, for a variety of reasons. Others have been reviewed in the books and publications already cited; most of these studies are subject to severe criticisms, and later in this paper an attempt will be made to detail these criticisms.

If we try to summarize the evidence available from these numerous studies in terms that are more meaningful to a psychologist than the type A nomenclature, we might perhaps say that the coronary-prone patient exhibits a high degree of psychoticism, especially as far as hostility/ aggression/cynicism are concerned, together with certain aspects of neuroticism, such as depression, rather than others, such as anxiety. Innes (1980) has shown some relationship between impulsivity, one aspect of psychoticism, and the coronary-prone behaviour pattern. In a much larger study by Chesney, Black, Chadwick and Rosenman (1981), psychological characteristics of 384 adult males were classified as type A or type B by the structured interviewer who examined them. Ss classified type A differed significantly from Ss classified type B on a number of psychological scales including measures of aggression, autonomy and impulsiveness, but not on measures of psychological distress. These are all aspects of psychoticism, which thus seems to be closely related to type A

as measured by the SI; pencil-and-paper questionnaire assessments of type A showed very low correlations with the SI, as already mentioned.

A study by Jenkins, Zyzanski, Ryan, Flessas and Tannenbaum (1977) suggested, in addition to the more usual features, that coronary-prone patients have a low threshold for becoming tense or depressed. A possible relationship between type A behaviour and extraversion is shown in a study by Frankenhaeuser, Lundberg and Forsman (1980) who compared type A and type B persons in terms of psychophysiological arousal during periods of inactivity and strenuous mental work. Type As felt more distressed than type Bs during inactivity, suggesting extraversion. In another study, Lundberg and Forsman (1979) found differences between type A and type B persons with respect to various hormonal and other assays, including greater cortisol excretion in type A persons under stimulation; this may be important in view of the relationship between cortisol and depression.

7. CORRELATION OR CAUSE?

So far we have been discussing essentially correlational studies where the implications of causality are by no means clear. Cross-sectional studies have obvious difficulties, and cannot be taken too seriously. Where patients already know the nature of their disease, this knowledge may obviously affect their personality ratings and interview responses, particularly, as far as anxiety, depression and other emotional reactions are concerned. In cross-sectional studies where the personality assessment is carried out prior to diagnosis, it is still possible that patients suffering from cancer or cardiovascular disease may be differentially affect in their assessment behaviour, quite apart from the fact that the disease itself may have a differential effect on the person, even without his knowledge. Thus little by way of causal implication can be read into these results.

Prospective studies, such as those of Grossarth-Maticek and some of those relating to type A behaviour, are of course not subject to these objections, but they too have their problems. Disease processes may be present long before they are diagnosed, and affect behaviour and personality. The influence of personality may not be direct, but indirect, i.e. through life style and other factors. This notion is rather similar to that first put forward by Pearl (1928), who made differential 'rates of living' responsible for longevity or early death. Differential mortality tends to reduce the sample, particulary an elderly sample, in ways that are difficult to take into account. Prospective studies do give good reasons for assuming that personality factors have a causal basis, particularly, when, as in the Grossarth-Maticek studies, they can be shown to be significantly more predictive than such obvious and clear-cut factors as smoking. (For lung cancer, other cancers and also for cardiovascular disease, partialling out personality left smoking insignificant as a predictor variable.)

We are left with a third method for assessing the causal relevance of personality to disease which is drastically differentiated from cross-sectional or longitudinal (prospective) studies, and not subject to any of the difficulties mentioned. Personality is essentially defined as habitual patterns of behaviour, and behaviour, of course, can be modified. If a given pattern is cancer-predictive or coronary-prone, and if such behaviour can be significanly modified by some form of behaviour therapy, then on the causal hypothesis it should follow that either healthy persons so treated should be less liable to cancer or cardiovascular disease, or that people already ill should survive longer with such treatment. If this could be demonstrated successfully, then the causal hypothesis would be strengthened to a considerable extent. We have already mentioned a study by Friedman *et al.* (1984) showing that modification of type A behaviour can significantly lessen recurrence of cardiac infarcts, but the effect was not very large, and is difficult to allocate to a specific type of behaviour change. Much more impressive are three large-scale studies carried out by Grossarth-Maticek *et al.* (1983b). All of these are concerned with reversing the type of behaviour found by Grossarth-Maticek to be conducive to the development of cancer or cardiovascular disease, as outlined above; as already explained, all these gave positive results.

These data provide convincing evidence of a causal relation between personality, cancer and cardiovascular disease. It will be seen from the preceding summary of the evidence that there are good reasons to suppose that a fairly strong relationship exists between personality, on the one hand, and cancer and cardiovascular disease, on the other. It also seems likely, from the results of

prospective studies and treatment studies, that this relationship is a causal one. Some of the evidence, at least, is in agreement with the hypothesis stated at the beginning of this paper that the personality traits which make a person coronary-prone are different from and in some cases opposite to those which make a person cancer-prone. [Thomas and Greenstreet (1973) in a prospective study found coronary and tumour patients very significantly differentiated on two canonical variables derived from a whole battery of tests, only some of which were based on personality variables.] It is the stable, extraverted low P person who seems to be more likely to develop neoplasms, whereas it is the emotional, hostile-aggressive high P scorer who seems more at risk as far as cardiovascular disease is concerned. Hopelessness and depression, mediated by stress, resembling Seligman's notion of 'learned helplessness', appear to be equally unfavourable personality characteristics for both cancer and CHD. The literature is by no means unanimous in supporting these conclusions, and there are certain obvious difficulties which have not been resolved. One of the major problems is that of 'repression". Many data, such as the absence of emotion in cancer-prone patients, can be explained either in terms of genetically low predisposition to respond emotionally to disturbing stimuli, or, as by Kissen, by reference to represed or suppressed emotions and feelings. Such suggestions are freely made, but usually without any empirical test. Such tests are now available (Gudjonsson, 1981) but they have not been applied, and consequently little can be said about this alleged 'repression', particularly as the concept is lacking in empirical support even in clinical situations where it originated (Grünbaum, 1984). Speculative psychoanalytic concepts still haunt the field, in spite of the absence of any empirical support for their usefulness.

Many of the apparent contradictions can perhaps be explained by the different methodologies adopted. Cross-sectional as opposed to longitudinal studies constitute different methodologies which, as already explained, cannot be expected to give identical results. The difference between SI and qustionnaire data has also already been documented, and may be of great importance. Differences in criterion measures are also vital; death from cancer or cardiovascular disease must be contrasted with the appearance of symptoms, or biochemical and physiological measures supposed to be related to the disease. We must also bear in mind the important fact that diagnoses of specific types of cancer or cardiovascular disease have a validity of no more than 50% or so, when tested against autopsy (Eysenck, 1985); in addition, diagnoses are often affected by behaviour patterns, such as those associated with smoking, drinking etc.--diagnoses of lung cancer are much more readily made in patients who smoke than in those who do not, evidencing 'detection bias', as Feinstein and Wells (1974) call it. Iatrogenic factors need to be taken into account. Linn, Linn and Jensen (1981) showed that hospitalization alone could depress immunological response. Last but not least, the measures of personality used by some of the authors are suspect; thus the use of the Rorschach and other projective techniques having poor reliability and little validity can hardly be countenanced any longer. Equally, the interpretation of scores on the subscales of the MMPI or the 16PF, in view of the known unreliability and suspect validity of the scales, must be in doubt. These and other weaknesses make a simply tally of results impossible, and necessitate a more sophisticated type of evaluation.

In conclusion, a few words may be apposite regarding the import of the personality correlations with disease here considered, and their relevance to the debate about the causal influence of smoking on lung cancer and cardiovascular disease. It may also be apposite to discuss very briefly the causal links that may be responsible for the observed personality-disease correlations. Eysenck (1980, 1985) has suggested an alternative view to the traditional one, making smoking a causal factor for lung cancer and cardiovascular disease. Figure 2 illustrates the main points of this theory. We start with the observed correlation between smoking and lung cancer (r_{st}), the size of which is itself of course in doubt for reasons given in the study cited. What is suggested is that genetic factors are in part responsible for lung cancer (1) and also for smoking (2); these genetic factors (4) and lung cancer (5). Additionally, stress is linked to smoking (6) and lung cancer (7). These links may explain the observed correlation along the lines of the causal network suggested, without smoking itself having a direct causal influence on lung cancer. A similar diagram could be drawn for cardiovascular disease, where in any case the correlation is much lower, and altogether in doubt.

As regards the way in which personality may be causally related to lung cancer, Eysenck (1983,

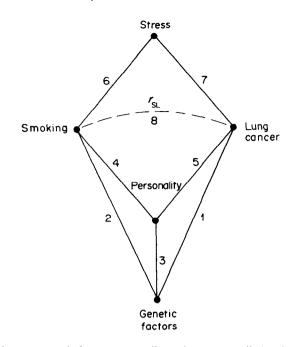


Fig. 2. Relationships between genetic factors, personality and stress as mediating the correlation between smoking and lung cancer.

1984a, b, 1985) has made a number of suggestions. The counter-intuitive negative relationship between lung cancer and neuroticism and psychoticism may be explained along the following lines. Stress has usually been found to increase liability to cancer, and one might have thought that high N and P scorers would suffer considerably more stress than low N and P scorers, by virtue of their constitutional reactivity. However, the literature makes it likely that it is *acute* stress which promotes cancer, while *chronic* stress may act to protect the organism against cancer, possibly through some 'inoculation' effect (Eysenck, 1983).

The negative relationship with introversion may be due to the fact that immune reactions have now been shown to be capable of being conditioned by means of Pavlovian mechanisms, and that introverts on the whole tend to condition more readily and more strongly than extraverts (Eysenck, 1967). This suggests the possibility that through conditioning introverts may increase the protection afforded by the immune system against cancer (Eysenck, 1985).

A more powerful causal factor may be the endocrine system, which is known to influence both personality and immunology to cancer. ACTH, for instance, is known to be produced by epinephrine, which is related to both introversion and neuroticism; it also is related to immune reaction protecting the individual against cancer. There are complex relationships between ACTH, the endogenous opiates, cortisol etc.; these are suggested in Fig. 3 (Eysenck, 1985). This is not the place to discuss these theories; they are mentioned only to indicate that the causal theory linking personality and disease leads to testable hypotheses which may give us better insight into the nature of these diseases than can be gained by simple-mined assertions that 'smoking causes cancer and cardiovascular disease'. Obviously such research is only at the beginning, but already a good deal of material is available to suggest that further work along these lines will be fruitful, and may lead to better prophylactic measures and improved treatment, as well as better understanding. However that may be, it will be clear that psychology, and the study of personality in particular, is relevant not only to psychiatry, but to the whole of medicine, where it is now commonplace to say that we must treat the person, not only the disease. For that purpose, clearly an understanding of the structure and dynamics of personality must be an absolute priority.

A recent review by Jemott and Locke (1984) makes it clear that "the bulk of evidence favours the view that psychosocial variables may play a role in modulating the human immune response" (p. 78). An annotated bibliography (Locke and Hornig-Rohan, 1984) lists over 1300 abstracts covering a period of 8 yr (1976–1982) relevant to this issue. Among the factors studied, personality

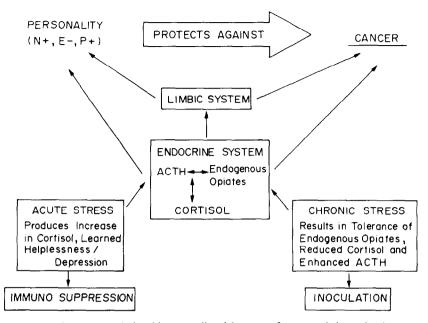


Fig. 3. Personality-cancer relationship as mediated by stress factors and the endocrine system.

is conspicuously lacking for the most part. It is clear that "stress is associated with an increased incidence of diseases against which the immune system defends and that it is associated with diminished immunocompetence as determined in a variety of *in vitro* assays" (Jemmott and Locke, 1984, p. 103). The evidence suggests that personality differences play an important part in this interaction; without a proper theory guiding research into this area. We are not likely to achieve replicable results. As Lewin used to say: "There is nothing as practical as a good theory".

REFERENCES

- Ahnve S., de Faire U., Orth-Gomes K. and Theorell T. (1979) Type A behaviour in patients with non-coronary chest pain admitted to a coronary care unit. J. psychosom. Res. 23, 219-223.
- Alexander F. (1950) Psychosomatic Medicine. Norton, New York.
- Baer P. E., Collins F. H., Bourianoff G. G. and Ketchel M. F. (1979) Assessing personality factors in essential hypertension with a brief self-report instrument. *Psychosom. Med.* **41**, 321–330.
- Bahnson C. B. and Bahnson M. B. (1964a) Cancer as an alternative to psychosis. In Psychosomatic Aspects of Neoplastic Disease (Edited by Kissen D. M. and Le Shan L.). Lippincott, Philadelphia, Pa.
- Bahnson C. B. and Bahnson M. B. (1964b) Denial and repression of primitive impulses and of disturbing emotions in patients with malignant neoplasms. *Ibid.*
- Bammer K. and Newberry B. (1981) Stress and Cancer. Hogrefe, Toronto.
- Bareford J. C., Dahlstrom W. G. and Williams R. B. (1983) Hostility, CHD incidence and total mortality: a 25-year follow-up study of 255 physicians. *Psychosom. Med.* 45, 59-63.
- Barquero J. L. V., Munoz P. E. and Jauregui V. M. (1981) The interaction between physical illness and neurotic morbidity in the community. Br. J. Psychiat. 139, 328-335.
- Baumann R., Ziprian H., Gödicke W., Hartrodt E., Neumann E. and Lauter J. (1973) The influence of acute psychic stress situations on biochemical and negative parameters of essential hypertensives at the early stage of the disease. *Psychother. Psychosom.* 22, 131–140.
- Bass C. and Wade C. (1982) Type A behaviour: not specifically pathogenic? Lancet Nov., 1147-1149.
- Bendien J. and Groen J. (1963) A psychological statistical study of neuroticism and extraversion in patients with myocardial infarction. J. psychosom. Res. 7, 11-14.
- Berglund G., Ander S., Lindström B. and Tibblin G. (1975) Personality and reporting of symptoms in normo- and hypertensive 50 year old males. J. psychosom. Res. 19, 139-145.
- Berndt H., Gunther H. and Rohte G. (1980) Persönlichkeitsstruktur nach Eysenck bei Kranken mit Brustdrüsen-und Bronchialkrebs und Diagnoseverzögerung durch den Patienten. Arch. Geschwülstforsch. 40, 359-368.
- Blohmke M., Engelhardt B. and Stelzer O. (1984) Psychosocial factors and smoking as risk factors in lung carcinoma. J. psychosom. Res. 28, 221-229.
- Blumberg E. M., West P. M. and Ellis F. W. (1954) A possible relation between psychological factors and human cancer. *Psychosom. Med.* 16, 277-286.
- Blumenthal J. A., Thompson L. W., Williams R. S. and Kong Y. (1979) Anxiety-proneness and coronary heart disease. J. psychosom. Res. 23, 17-21.
- Booth G. (1969) General and organic-specific object relationships in cancer. Ann. N.Y. Acad. Sci. 164, 568-577.

- Bortner R. W. (1969) A short rating scale as a potential measure of pattern A behaviour. J. chron. Dis. 22, 87-91.
- Borysenko M. and Borysenko J. (1982) Stress, behaviour and immunity: animal models and mediating mechanisms. Gen. Hosp. Psychiat. 4, 59-67.
- Brozek J., Keys A. and Blackburn H. (1966) Personality differences between potential coronary and non-coronary subjects. Ann. N.Y. Acad. Sci. 134, 1057–1063.
- Burch P. R. J. (1976) The Biology of Cancer. MT, Lancaster, Lancs.
- Butler J., Regelson W., Lawlis G. F. and Bristow O. V. (1982) Personality profile comparison between cancer patients and other disease groups. *Multivar. exp. clin. Res.* 6, 15-21.
- Chan K. B. (1977) Individual differences in reactions to stress and their personality and situational determinants: some implications for community mental health. Soc. Sci. Med. 11, 89-103.
- Chesney M. A., Black G. W., Chadwick J. H. and Rosenman R. H. (1981) Psychological correlates of the type A behaviour patterns. J. behav. Med. 4, 217-229.
- Cooper C. L. (1983) Stress Research. Wiley, New York.
- Coppen A. and Metcalfe M. (1963) Cancer and extraversion. Br. med. J. 11, 18-19.
- Costa P. T. (1985) Is neuroticism a risk factor for CAD? Is type A a measure of neuroticism? In *Biobehavioural Bases of* Coronary Heart Disease (Edited by Schmidt T., Dembroski T. and Blumchen G.). Springer, New York.
- Costa P. T., McCrae R., Andres R. and Tobin J. (1980) Hypertension, somatic complaints, and personality. In Hypertension and Cognitive Processes (Edited by Elias M. E. and Streeten D.), pp. 95-110. Beech-Hill, Mt. Desert.
- Costa P. T., Fleg J. L., McCrae R. R. and Lakatta E. G. (1982) Neuroticism, coronary artery disease, and chest pain complaints: cross-sectional and longitudinal studies. *Expl Aging Res.* 8, 37-44.
- Crisp A. H. (1970) Some psychosomatic aspects of neoplasis. Br. J. med. Psychol. 43, 313-331.
- Dattore P. J., Shantz F. C. and Coyne L. (1980) Premorbid personality differentiation of cancer and non-cancer groups: a test of a hypothesis of cancer proneness. J. counsel. clin. Psychol. 48, 388-394.
- De Champlain J., Causineau D., Van Ameringen M., Marc-Aurel J. and Yamaquelin N. (1977) The role of the sympathetic system in experimental and human hypertension. *Postgrad. med. J.* 53, 15-30.
- Dembroski T. M. and MacDougall J. M. (1985) Beyond global type A: relationships of paralinguistic attributes, hostility and anger-in to coronary heart disease. In *Stress and Coping* (Edited by Field T., McCabe P. and Schneiderman N.). Erlbaum, Hillsdale, N.J.
- Dembroski T. M., Weiss S. M., Shields J., Haynes S. and Feinleib M. (1978) Coronary-prone Behaviour. Springer, New York.
- Dembroski T. M., Schmidt T. H. and Blümchen G. (Eds) (1983) Biobehavioural Bases of Coronary Heart Disease. Karger, Basel.
- Dembroski T. M., MacDougall J. M., Williams R. B., Haney T. L. and Blumenthal J. A. (1985) Components of type A, hostility and anger-in: relationship to angiographic findings. *Psychosom. Med.* In press.
- Denney D. R. and Frisch M. B. (1981) The role of neuroticism in relation to life stress and illness. J. psychosom. Res. 25, 303-307.
- Derogatis L. R., Abeloff M. D. and Melisaratos M. (1979) Psychological coping mechanisms and survival time in metastatic breast cancer. J. Am. med. Ass. 242, 1504-1508.
- van Dijl H. (1979) Myocardial infarction patients and sociability. J. psychosom. Res. 23, 3-6.
- Dobson C. B. (1982) Stress: the Hidden Adversary. MT, Lancaster, Lancs.
- Doll R. and Peto R. (1976) Mortality in relation to smoking: 20 years' observation on male British doctors. Br. med. J. 2, 1525-1536.
- Duckitt J. and Broll T. (1982) Personality factors as moderators of the psychological impact of life stress. S. Afr. J. Psychol. 12, 76–80.
- Dunbar F. (1943) Psychosomatic Diagnosis. Hoeber, New York.
- Elias M. F., Robbins M. A., Rice A. and Edgecomb J. L. (1982) Symptom reporting, anxiety and depression in arteriographically classified middle-aged chest pain patients. Expl Aging Res. 8, 45-81.
- Esler M., Julius S., Zweifler A., Randall O., Harburg E., Gardiner H., and De Quattro V. (1977) Mild high-renin essential hypertension neurogenic human hypertension. *New Engl. J. Med.* 296, 405-411.
- Everson R. B. and Fraumeni J. F. (1975) Mortality among medical students and young physicians. J. med. Educ. 50, 809-811.
- Eysenck H. J. (1967) The Biological Basis of Personality. Thomas, Springfield, Ill.
- Eysenck H. J. (1976) Sex and Personality. Open Books, London.
- Eysenck H. J. (1980) The Causes and Effects of Smoking. Sage, New York.
- Eysenck H. J. (1981) Personality and cancer: some comments on a paper by H. Berndt. Arch. Geschwuhtforsch. 51, 442-443.
- Eysenck H. J. (1983) Stress, disease and personality: the "inoculation effect". In Stress Research (Edited by Cooper C. L.), pp. 121-146. Wiley, New York.
- Eysenck H. J. (1984a) Personality, stress and lung cancer. In Contributions to Medical Psychology, Vol. 3 (Edited by Rachman S.), pp. 151-171. Pergamon Press, Oxford.
- Eysenck H. J. (1984b) Lung cancer and the stress-personality inventory. In *Psychosocial Stress and Cancer* (Edited by Cooper C. L.), pp. 49-71. Wiley, New York.
- Eysenck H. J. (1985) Smoking and health. In Smoking and Society (Edited by Tollison R.). In press.
- Eysenck H. J. and Eysenck M. W. (1985) Personality and Individual Differences. Plenum Press, New York.
- Eysenck H. J. and Fulker D. (1983) The components of type A behaviour and its genetic determinants. Person. individ. Diff. 4, 499-505.
- Feinstein A. R. and Wells C. K. (1974) Cigarette smoking and lung cancer: the problem of "detection bias" in epidemiologic rates of disease. Trans. Ass. Am. Physn 87, 180-185.
- Floderus B. (1974) Psycho-social factors in relation to coronary heart disease and associated risk factors. Nord. Hyg. Tidskr., Suppl. 6.
- Frankenhaeuser M., Lundberg U. and Forsman L. (1980) Note on arousing type A persons by depriving them of work. J. psychosom. Res. 24, 45-47.
- Friedman G. D., Siegelaub A. B., Dales L. D. and Seltzer C. C. (1979) Characteristics predictive of coronary heart disease

in ex-smokers before they stopped smoking; comparison with persistent smokers and non-smokers. J. chron. Dis. 32. 175 - 190

- Friedman M. and Rosenman R. (1974) Type A Behaviour and Your Heart. Wildwood, London.
- Friedman M., Thoresen C. E., Gill J. J., Powell L. H., Ulmer D., Thompson L., Price V. A., Rabin D. D., Breall W. S., Dixon T., Levy R. and Bourg E. (1984) Alteration of type A behaviour and reduction in cardiac recurrences in postmyocardial infarction patients. Am. Heart J. 108, 237-248.
- Gagnon F. (1950) Contribution to the study of the etiology and prevention of cancer of the cervix of the uterus. Am. J. Obstet. Gynec. 60, 516-527.
- Gildea E. F. (1949) Special features of personality which are common to certain psychosomatic disorders. Psychosom. Med. 11, 273-278.
- Greer S. and Morris T. (1975) Psychological attributes of women who develop breast cancer: a controlled study. J. psychosom. Med. 19, 147-153.
- Greer S., Morris T., and Pettingale K. W. (1979) Psychological response to breast cancer: effect on outcome. Lancet 11, 785-789.
- Grossarth-Maticek R. (1980) Synergic effects of cigarette smoking, systolic blood pressure, and psychosocial risk factors for lung cancer, cardiac infarct and apoplexi cerebri.
- Grossarth-Maticek R., Kanazir D., Schimidt P. and Vetter H. (1982a) Psychosomatic factors in the process of cancerogenesis. Psychother. Psychosom. 28, 284-302.
- Grossarth-Maticek R., Sigrist J. and Vetter N. (1982b) Interpersonal repression as a predictor of cancer. Soc. Sci. Med. 16, 493-498.
- Grossarth-Maticek R., Kanazir D. T., Vetter H. and Jankovic M. (1983a) Psychother. Psychosom. 39, 94-105.
- Grossarth-Maticek R., Schmidt P., Vetter H. and Arndt S. (1983b) Psychotherapy reserach in oncology. In Health Care and Human Behaviour (Edited by Steptoe A. and Mathews A.). Academic Press, London.
- Grossarth-Maticek R., Frentzel-Beyme R. and Becker N. (1984) Cancer risks associated with life events and conflict solutions. Cancer Detect. Prevent. 7, 201-209.
- Grossarth-Maticek R., Kanazir D. T., Schmidt P. and Vetter H. (1985a) Psychosocial and organic variables as predictors for lung cancer, cardiac infarct and apoplexy: some differential predictors. Person. individ. Diff. 6, 313-323
- Grossarth-Maticek R., Bastiaans J. and Kanazir D. T. (1985b) Psychosocial factors as strong predictors of mortality from cancer, ischemic heart disease and stroke: the Yugoslav Prospective Study. J. psychosom. Med. In press.
- Grünbaum A. (1984) The Foundations of Psychoanalysis. Univ. of California Press, Berkeley, Calif.
- Gudjonsson G. H. (1981) Self-reported emotional disturbance and its relation to electrodermal reactivity, defensiveness and trait anxiety. Person. individ. Diff. 2, 47-52.
- Hagnell O. (1966) The premorbid personality of persons who develop cancer in a total population investigated in 1947 and 1957. Ann. N.Y. Acad. Sci. 125, 846-855.
- Hodapp V. and Weyer G. (1982) Zur Stress-Hypothese der Essentiellen Hypertonie. In Essentielle Hypertonie: Psychologisch-medizinische Aspekte (Edited by Vaitl D.). Springer, Berlin.
- Hodapp V., Weyer G. and Becker J. (1975) Situational stereotypy in essential hypertension patients. J. psychosom. Res. 19, 113-121.
- Horne R. L. and Picard R. S. (1979) Psychosocial risk factors for lung cancer. Psychosom. Med. 41, 503-514.
- Ibrahim M. A., Jenkins C. D., Cassel J., McDonough J. R. and Hames C. G. (1966) Personality traits and coronary heart disease. J. chron. Dis. 19, 255-262.
- Innes J. M. (1980) Impulsivity and the coronary-prone behaviour pattern. Psychol. Rep. 47, 976-978.
- Jemmott J. B. and Locke S. E. (1984) Psychosocial factors, immunologic mediation, and human susceptibility to infectious diseases. How much do we know? Psychol. Bull. 95, 78-108.
- Jenkins C. D. (1978) Behavioural risk factors in coronary artery disease. A. Rev. Med. 29, 543-562.
- Jenkins C. D. (1979) Psychosocial modifiers of responses to stress. In Stress and Mental Disorder (Edited by Barrett J, E, Rose R. M. and Klennan G. L.). Raven Press, New York.
- Jenkins C. D., Zyzanski S. J., Ryan T. J., Flessas A. and Tannenbaum S. (1977) Social insecurity and coronary-prone type A responses as identifiers of severe atherosclerosis. J. consult. clin. Psychol. 45, 1060-1067.
- Jenkins C. D., Zyzanski S. J. and Rosenman R. N. (1978) Coronary-prone behaviour: one pattern or several? Psychosom. Med. 40, 25-43.
- Johnson J. H. and Sarason I. G. (1979) Recent developments in research on life stress. In Human Stress and Cognition (Edited by Hamilton V. and Warburton D. M.). Wiley, Chichester, Sussex.
- Kantor S. and Robertson A. J. (1977) Repressed hostility and coronary heart disease: reappraisal of a relationship in terms of a meaning-focussed approach to psychological measurement. Soc. Sci. Med. 11, 625-634.
- Keehn R. J. (1974) Probability of death related to previous army rank. Lancet 2, 170.
- Keehn R. J., Goldberg I. P. and Beebe G. W. (1974) Twenty-four year mortality follow-up of army veterans with disability separation for psychoneurosis in 1944. *Psychosom. Med.* 36, 27-76.
- Kidson M. A. (1971) Personality factors in hypertension. Aust. N.Z. Jl Psychiat. 5, 139-145.
- Kissen D. M. (1963a) Personality characteristics in males conducive to lung cancer. Br. J. med. Psychol. 36, 27-36.
- Kissen D. M. (1963b) Aspects of personality of men with lung cancer. Acta psychother. 11, 200-210.
- Kissen D. M. (1964a) Relationship between lung cancer, cigarette smoking, inhalation and personality. Br. J. med. Psychol. 37. 203-216.
- Kissen D. M. (1964b) Lung cancer, inhalation and personality. In Psychosomatic Aspects of Neoplastic Disease (Edited by Kissen D. M. and Le Shan L.). Lippincott, Philadelphia, Pa.
- Kissen D. M. (1964c) The influence of some environmental factors on personality inventory scores in psychosomatic research. J. psychosom. Res. 8, 145-149. Kissen D. M. (1966) The significance of personality in lung cancer in men. Ann. N.Y. Acad. Sci. 125, 820-826.
- Kissen D. M. (1967) Psychological factors, personality and lung cancer in men aged 55-64. Br. J. med. Psychol. 40, 29-34. Kissen D. M. (1969) Present status of psychosomatic cancer research. Geriatrics 24, 129-137.
- Kissen D. M. and Eysenck H. J. (1962) Personality in male lung cancer patients. J. psychosom. Res. 6, 123-137.
- Kissen D. M. and Rao L. G. S. (1969) Steroid secretion patterns and personality in lung cancer. Ann. N.Y. Acad. Sci. 164, 476-482.

- Kissen D. M., Brown R. and Kissen M. (1969) A further report on personality and psychosocial factors in lung cancer. Ann. N.Y. Acad. Sci. 164, 535-545.
- Kitagawa E. M. and Hauser P. M. (1973) Differential Mortality in the United States: a Study in Socioeconomic Epidemiology. Harvard Univ. Press, Cambridge, Mass.
- Kopp M. S. (1984) Electrodermal characteristics in psychosomatic patient groups. Int. J. Psychophysiol, 2, 73-85.
- Kopp M. S. and Koranyi L. (1982) Autonomic and psychologic correlates in hypertension and duodenal ulcer. Pavlov. J. biol. Sci. 17, 178-187.
- Le Shan L. (1959) Psychological states as factors in the development of malignant disease: a critical review. J. natn Cancer Inst. 22, 1-18.
- Le Shan L. and Worthington R. E. (1956) Personality as factor in the pathogenics of cancer: review the literature. Br. J. med. Psychol. 29, 49-56.
- Lewinsohn P. M. (1956) Personality correlates of duodenal ulcer and other psychosomatic reactions. J. clin. Psychol. 12. 296-298.
- Locke S. E. and Hornig-Rohan R. (1984) Mind and Immunity: Behavioral Immunology (1976-82). Institute for the Advancement of Health, New York.
- Lombard H. L. and Potter E. A. (1950) Epidemiological aspects of cervical cancer-II. Hereditary and environmental factors. Lancet 3, 960-968.
- Lundberg U. and Forsman L. (1979) Adrenal-medullary and adrenal-cortical responses to understimulation and overstimulation: comparison between type A and type B persons. Biol. Psychol. 9, 79-89.
- Mann G. V. (1977) Diet-heart: end of an era. New Engl. J. Med. 296, 644-649.
- Maudsley H. (1899) The Pathology of Mind, 3rd edn. Appleton, New York.
- McDonough J. R., Hames C. G., Stulb S. and Garrison G. E. (1963) Cardiovascular disease field study in Evans County: characteristics of the study population. Publ. Hlth Rep. 78, 1051-1058.
- McMichael Sir John (1979) Fats and atheroma: an inquest. Br. med. J. 5, 173-175.
- Medalie J. H., Synder M., Groen J. J., Nuefeld H. N., Goldbourt V. and Riss E. (1973) Angina pectoris among 10,000 men: 5 year incidence and univariate analysis. Am. J. Med. 55, 583-594.
- Mctropolitan Life Insurance Company (1973) Longevity of legislators. State Bull. Metropolitan Life Insurance Co. 54, 3-4.
- Miles H. H., Waldvogel S., Barrabee E. L. and Cobb S. (1954) Psychosomatic study of 46 young men with coronary artery disease. Psychosom. Med. 16, 455.
- Miller S. M. (1981) Predictability and human stress: toward a clarification of evidence and theory. Adv. exp. soc. Psychol. 14, 203-253.
- Morivama I, M., Krueger D. E. and Stamler J. (1971) Cardiovascular Diseases in the United States. Harvard Univ. Press, Cambridge, Mass
- Morris T., Greer S., Pettingale K. W. and Watson M. (1981) Patterns of expression of anger and their psychological correlates in women with breast cancer. J. psychosom. Dis. 25, 111-117.
- Morrison F. R. and Paffanbarger R. A. (1981) Epidemiological aspects of biobehaviour in the aetiology of cancer: a critical review. In Perspectives on Behavioural Medicine (Edited by Weiss S. M., Herd J. A. and Fox B. N.), pp. 135-162. Academic Press, New York.
- Multiple Risk Factor Intervention Trial Research Group (1982) Multiple Risk Factor Intervention Trial. J. Am. med. Ass. 248, 1465-1477.
- Musante L., MacDougall J. M., Dembroski T. M. and van Horn A. E. (1985) Component analysis of the type A coronary-prone behaviour pattern in male and female college students. J. Person. soc. Psychol. In press.
- Myers J., Lindenthal J. and Pepper M. (1975) Life events, social interpretation and psychiatric symptomatology. J. Hlth soc. Behav. 16, 421-429.
- Osler W. (1910) The Lumleian lectures on angina pectoris. Lancet 1, 839-845.
- Ostfeld A. M., Lebovits B. Z., Shekelle R. S. and Paul O. A. (1967) Prospective study of the relationship between personality and coronary heart disease. J. chron. Dis. 17, 265-276.
- Pearl R. (1928) The Rate of Living. Knopp, New York.
- Pecte D. C. (1955) Psychosomatic Genesis of Coronary Artery Disease. Thomas, Springfield, Ill. Pichot P., de Bonis M., Somogyi M., Degre-Coustry C., Kittel-Bossuit F., Rustin-Vandenhende R. M., Dramaix M. and Bernet, A. (1977) Etude métrologique d'une batterie de tests destinée à l'étude des facteurs psychologiques en epdemiologie cardio-vasculaire. Int. Rev. appl. Psychol. 26, 11-19.
- Pleszewski Z. (1977) Funkcjonowanie Emocjonalne Pacjentow przed i po Zawale Serca. Seria Psychologia I Pedagogika No. 39.
- Price V. A. (1982) Type A Behaviour Patterns. Academic Press, New York.
- Rahe R. H., Hervig L. and Rosenman R. H. (1978) The heritability of type A behaviour. Psychosom. Med. 40, 478-486. Registrar General for England & Wales (1958) Decennial Supplement, England and Wales, 1951. Occupational Mortality, Part II, Vol. 1. HMSO, London.
- Rim Y. (1981) Pattern-a behaviour and its personality correlates in students of both sexes. Scientia paed. exp. 18, 98-102.
- Rimé B. and Bonami M. (1979) Overt and covert personality triats associated with coronary heart disease. Br. J. med. Psychol. 52, 77-84.
- Rose G., Hamilton P., Colwell L. and Shipley M. (1982) A randomized controlled trial of anti-smoking advice: 10 year results. J. Epid. Community HIth 36, 102-108.
- Rosenman R. H. and Chesney M. A. (1980) The relationship of type A behaviour pattern to coronary heart disease. Activitas nerv. Sup. 22, 1-45.
- Sainsbury D. (1960) Neuroticism and hypertension in an outpatient population. J. psychosom. Res. 12, 261-273.
- Schmidt P. (1984) Autoritarismus, Entfremdung und Psychosomatische Krebsforschung: Explikation der drei Forschungsprogramme durch eine allgemeine Theorie und empirische Tests mittels Strukturvergleichung. Unpublished Habilitationsschrift, Univ. of Heidelberg.
- Seltzer C. C. and Jablon S. (1977) Army rank and subsequent mortality by cause: 23-year follow-up. Am. J. Epid. 105, 559-566.
- Shekelle R. B., Ostfeld A. M., Lebovitz B. Z. and Paul O. (1970) Personality triats and CHD; a re-examination of Ibrahim's hypothesis using longitudinal data. J. chron. Dis. 23, 33-38.

- Siltanen P., Lauroma M., Nirkko O., Punsar S., Pyorala K., Tuominen H. and Vanhala K. (1975) Psychological characteristics related to coronary heart disease. J. psychosom. Res. 19, 183-195.
- Sklar L. S. and Anisman N. (1981) Stress and cancer. Psychol. Bull. 89, 369-406.
- Smith R. E., Johnson J. H. and Sarason I. G. (1978) Life change, the sensation seeking motive, and psychological distress. J. consult. clin. Psychol. 46, 348-349.
- Spielberger C. D. (1976) Stress, anxiety and cardiovascular disease. Jl S. Carol. med. Ass., Suppl. Feb., 15-22. Stavrakay K. M. (1968) Psychological factors in the outcome of human cancer. J. psychosom. Res. 12, 251-259.
- Steptoe A. (1981) Psychological Factors in Cardiovascular Disorders. Academic Press, New York.
- Storment C. T. (1951) Personality and heart disease. Psychosom. Med. 13, 304-712.
- Theorell T., de Faire V., Schalling D., Adamson V. and Askevold F. (1979) Personality traits and psychological reactions to a stressful interview in twins with varying degrees of coronary heart disease. J. psychosom. Res. 23, 89-99.
- Thomas C. B. (1976) Precursors of premature disease and deaths. Ann. intern. Med. 85, 653-658.
- Thomas C. B. and Greenstreet R. L. (1973) Psychological characteristics in youth as predictors of five disease states: suicide, mental illness, hypertension, coronary heart disease and tumour. John Hopkins med. J. 132, 16-43.
- Totman R., Kiff J., Reed S. E. and Craig J. W. (1980) Predicting experimental colds in volunteers from different measures of recent life stress. J. psychosom. Res. 24, 155-163.
- Tyson G. A. (1981) Locus of central and stressful life events. S. Afr. J. Psychol. 11, 116-117.
- Watson C. G. and Schuld D. (1977) Psychosomatic factors in the etiology of neoplasms. J. counsel. clin. Psychol. 45, 455-461.
- Weiner H. (1977) Personality factors and the importance of emotional stress in hypertension. In Hypertension (Edited by Genst J., Koiw E. and Kuchel O.). McGraw-Hill, New York.
- Weiss S. M., Detre T. and Cooper T. (1981) Coronary-prone behaviour and coronary heart disease: a critical review. Circulation 63, 1199-1215.
- Weyer G. and Hodapp V. (1977) Persönlichkeitseigenschaften bei essentiellen Hypertonikern. Z. klin. Psychol. 6, 70-78. Weyer G. and Hodapp V. (1979) Job stress and essential hypertension. In Stress and Anxiety, Vol. 6 (Edited by Sarason I. G. and Spielberger C. D.), pp. 337-349. Hemisphere, Washington, D.C.
- Wilkinson D. G. (1981) Psychiatric aspects of diabetes mellitus. Br. J. Psychiat. 138, 1-9.
- Williams R. B., Bareford J. C. and Shekelle R. B. (1984) The health consequences of hostility. In Anger, Hostility and Behavioural Medicine (Edited by Chesney M. A., Goldstone S. E. and Rosenman R. H.). Hemisphere (McGraw-Hill), Washington, D.C.
- Willis T. (1684) Pharmacentive rationalis. In The Works of Thomas Willis. Dring Harper & Leigh, London.
- Zyzanski S. J., Jenkins C. D., Ryan T., Flessas A. and Everist M. (1976) Psychologic correlates of coronary angiographic findings. Archs intern. Med. 136, 1234-1237.