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Anxiety, Learned Helplessness, and Cancer:

A Causal Theory

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Abstract—It is argued that the ancient Cartesian body-mind separation still haunts psychology and medicine, and has led to a restriction of theoretical and applied psychological work to psychiatry. It is suggested that psychosocial factors, such as personality, stress, and coping behavior have relevance to many medical disorders normally considered to be of purely physical origin. It is also suggested that it is possible to postulate causal factors relating these psychosocial factors to the immune system, hormones and peptides, and the limbic system in general. A detailed discussion is given of work that has recently been done on the relationship between psychosocial factors and cancer, including the prophylactic use of behaviour therapy to alter some of the psychosocial factors to make possible the prevention of cancer, or the prolongation of life in patients who are incurably ill.

It has often been said that: "Disease is too serious a matter to be left to physicians," and the failure of physicians to take into account psychosocial factors in the causation and maintenance of disease tends to bear out this statement. Indicative of the complexity of the disease process is a study by Selzer 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 had about a 40% advantage. Standardised mortality rates for malignant neoplasms were 0.97, 0.86, and 0.70 for Privates, NCOs, and officers, respectively. 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. Thus, mortality rates for former Privates were almost twice as high as for former officers.

In part these differences are due to social class, education, and socioeconomic status, but even when these are taken into account, large dif-

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ferences still remain (Eysenck, 1985a). Figures published by the British Registrar General (1958) show only a small increase from 0.98 in Social Class 1 to 1.18 in Social Class 4. For some diseases, such as ischaemic heart disease, the largest mortality ratios in the English data were for Social Class 1, and the smallest for Classes 4 and 5. As Selzer and Jablon (1977) point out, beyond the probable role of socioeconomic factors, it may be speculated that the selection process for advancement in rank is also affected by factors which may have some underlying biological bases: "Those biological factors which contribute to higher levels of performance and leadership may also be associated with greater longevity" (p. 565).

There is a large body of evidence suggesting that stress, and ability to cope with stress, are relevant to many physical diseases, possibly through neurohumoral maintenance of immune homeostasis (Fox, 1983; Korneva, Klimenko, & Shkhinek, 1985). Immunity is influenced by neural determinants as well as by experience (conditioning), as Brittain and Wiener (1985) make clear, and immune functioning is to some extent controlled by stressors, and ability to cope with stressors, and possibly mediated by endogenous opiates and other peptides. In this complex interaction, cognitive factors also play an important part (Taylor, 1983).

The present paper will deal particularly with the relationship between psychosocial factors, such as stress, anxiety, "learned helplessness," and cancer. Such relationships between psychological variables and disease have been posited at least since the 18th century (Eysenck, 1986). Stress in particular has received much attention, as shown by recent reviews by Fox (1978, 1983), Bammer and Newberry (1981), Cooper (1983), and Dobson (1982). There has also been much recent research on the relationship between stress, behaviour, and the immune system (Borysenko & Borysenko, 1982). The causal hypothesis in this work has been that stress (particularly uncontrollable stress, leading to learned helplessness) suppresses the activity of the immune system, perhaps through the production of cortisol and other corticosteroids, which are known to reduce the efficacy of the immune system. Weyer and Hodapp (1979) and Hodapp and Weyer (1982) add 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 decisive in causing stress. "This means, of course, that when illness as a result of stress is to be investigated, it is not enough to search for isolated relationships between illness and personality or between illness and environment" (Weyer & Hodapp, 1979, p. 337). Their own investigations have resulted in a causal model which links various stress factors with extraversion and neuroticism (anxiety) (Hodapp & Weyer, 1982). The point that stress cannot be objectively defined in terms of situations only, but always in relation to the reaction to the stress of the organism (strain), has been emphasized by Eysenck (1985a). Identical environmental situations can be stressful for one person, rewarding for another.

Two major psychological syndromes have been linked theoretically with stress, leading to the development of malignant neoplasms. The first of these is the loss-depression-hopelessness syndrome (LeShan, 1959). According to this theory, the development of cancer is attributed to the loss of significant objects in the life of a given person, such as career disappointments, loss of self-esteem, death of a loved person, etc. This conception is not far removed from Seligman's well known concept of *learned helplessness* (Abramson, Seligman, & Teasdale, 1978; Maier & Seligman, 1976; Miller & Seligman, & Kurlander, 1975; Rosellini & Seligman, 1975). The work of this group also showed that the circle of learned helplessness and depression could be broken by suitable therapeutic procedures (Seligman, Rosellini, & Kozak, 1975).

The second of these syndromes is one emphasising lack of emotional reaction, or its suppression. It is postulated that the onset and development of malignant tumours may be associated with the excessive use of repressive and denying mechanisms (Bahnson & Bahnson, 1964b) or a general inhibition of emotional reactions (Kissen & Eysenck, 1962; Kissen, 1963a, 1963b). These two theories seem to be somewhat contradictory, as they assert that the cancer-prone subject is both likely to suffer depression and hopelessness, on the one hand, and lack of anxiety, on the other. However, it should be remembered that anxiety and depression, while usually correlated in the general population, are not identical; if it is true that "true grief is passionless," we may find deep depression without anxiety. In any case, a theory such as this requires empirical investigation rather than a priori rejection.

As Dattore, Shontz, and Coyne (1980) point out, "These various theoretical positions have been both supported and contradicted by research" (p. 389). Critical reviews by Brown (1966), Dattore (1978), Perrin and Pierce (1959), and Eysenck (1985a) indicate that the major criticism of much of the published work is that studies were carried out on patients already suffering from cancer; at best, the investigations were carried out on patients prior to diagnosis, so that the effects of fear and anxiety are likely to be roughly equal in strength in those who later on were found to suffer from these disorders and those who were not. Nevertheless, prospective studies are obviously desirable, and much more likely to be informative than studies carried out on patients already ill. Eysenck (1985a, 1985b) concluded his survey of a large number of retrospective studies by stating that there is evidence for a relationship between disease and personality. For cancer, it appears that both hypotheses outlined above are correct. The evidence strongly suggests that cancer-prone patients have *lower* scores than controls on questionnaires of neuroticism, anxiety, etc. Evidence from the work of Kissen and his associates (Kissen, 1963a, 1963b, 1964a, 1964b, 1964c, 1966, 1967, 1969; Kissen, Brown, & Kissen, 1969; Kissen & Eysenck, 1962) showed that subjects low on the Eysenck neuroticism scale had a six-fold likelihood of suffering from lung cancer as compared with subjects with high scores on the scale. Kissen 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.

The evidence for depression-learned helplessness-hopelessness is less clear-cut. It is related to the discovery that psychoticism, a personality trait related to aggressiveness, ego-centricity, tough-mindedness, coldness, and lack of empathy, act like neuroticism, as a protection against cancer. These traits are the obverse of "learned helplessness." As Bahnson and Bahnson (1964) pointed out, we may consider cancer as an alternative to psychosis. Roughly speaking, the ratio of deaths by cancer in schizophrenic patients as compared with nonpatients is in the neighbourhood of 1:3, a proportion not to be accounted for in terms of smoking.

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 (1982); Grossarth-Maticek, Kanazir, Schmidt, and Vetter (1982a, 1985a); Grossarth-Maticek, Frentzel-Beyme, and Becker (1984); and Grossarth-Maticek, Bastiaans, and Kanazir (1985b). These papers report on a completed 10-yr follow-up study in Yugoslavia, and two still ongoing follow-up studies in Heidelberg. The sample in the Yugoslavian prospective study consisted of 1353 subjects, who were recruited by selecting the oldest person in every second house in a small town with a population of 14,000 people. Most of the subjects were between 59 and 65 years old. Psychosocial data were recorded using a questionnaire and an observational catalogue. Height, weight, and 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 occurence of different diseases in the sample, and also recorded diagnoses on the death certificates. In those who died of cancer, cancer of the lung, stomach, rectum, and prostate predominated among 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 subjects. This design is clearly superior to those discussed above, and avoids most of the criticisms 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 incidence in the form of observed and expected deaths. The scale used was entitled "rational, anti-emotional behaviour" (R–A Scale), which is the obverse end of the continuum entitled "Neuroticism" in the Kissen and Eysenck (1962) study. This scale has 11 items, highly intercorrelated, relating to this concept of non-neurotic, non-emotional behaviour.

Expressed in terms of correlations, chronic helplessness correlated with cancer 0.59, and rational and anti-emotional behaviour 0.51. These are very high coefficients indeed in a predictive study. It is also interesting to note that Grossarth-Maticek calculated x^2-values comparing the predictive effectiveness of smoking and R–A personality. For lung

TABLE 1
EXPECTED AND OBSERVED DEATHS FROM LUNG CANCER AND OTHER CANCERS FOR LOW AND HIGH SCORES ON THE RATIONAL-ANTI-EMOTIONAL (R-A) SCALE

	R–A Scale			
	Low Score		High Score	
	Obs	Exp	Obs	Exp
Lung Cancer	0	26	38	12
Other Cancer	8	84	120	44

cancer, the figures are 68.8 for smoking, and 84.1 for personality, showing that personality is a more important predictive variable than smoking as far as lung cancer is concerned. Much larger differences were found for all cancers, when smoking and personality were compared as predictor variables. Grossarth-Maticek (1980) has also presented evidence to show that these effects are synergistic, in the sense that the effects of smoking are disproportionally greater in those people whose personality predisposes them to cancer.

A theory is briefly stated below which may be used to explain the observed effects, as far as the relationship between uncontrollable stress, personality, and cancer are concerned, and may be used to make predictions along causal lines. The theory is discussed in some detail elsewhere (Eysenck, 1985a, 1985b), but Figure 1 may be used to show it in diagrammatic form. We start out with the observation that certain personality variables (neuroticism, introversion, psychoticism) appear to protect humans against cancer, in the sense that they are found less frequently in people who develop cancer, and more frequently in those who do not, and seem to be protected against it. It would seem that the endocrine system may be partly responsible *both* for the development of the relevant personality traits, and also for the enhancement or suppression of the immune system, which prevents the development of malignant tumours. Thus cortisol is associated with acute stress, the development of learned helplessness and depression, and immunosuppression. Chronic stress, on the other hand, results in the tolerance of endogenous opiates and cortisol, and enhances ACTH, which in turn is related to neuroticism and introversion, and is known to increase immune reaction.

The differentiation between acute stress and chronic stress is very important. It is *acute* stress which has immunosuppressive effects, and is associated with the development of cancer; *chronic* stress has been shown in several studies and experiments to have the opposite effect, possibly giving rise to what I have called the "inoculation effect" (Eysenck, 1983, 1984a, 1984b). This differentiation makes plain the apparently paradoxical fact that neuroticism and psychoticism, which are personality traits liable to be found very stressful by their possessors, appear to be correlated *negatively* with the development of carcinomas. These

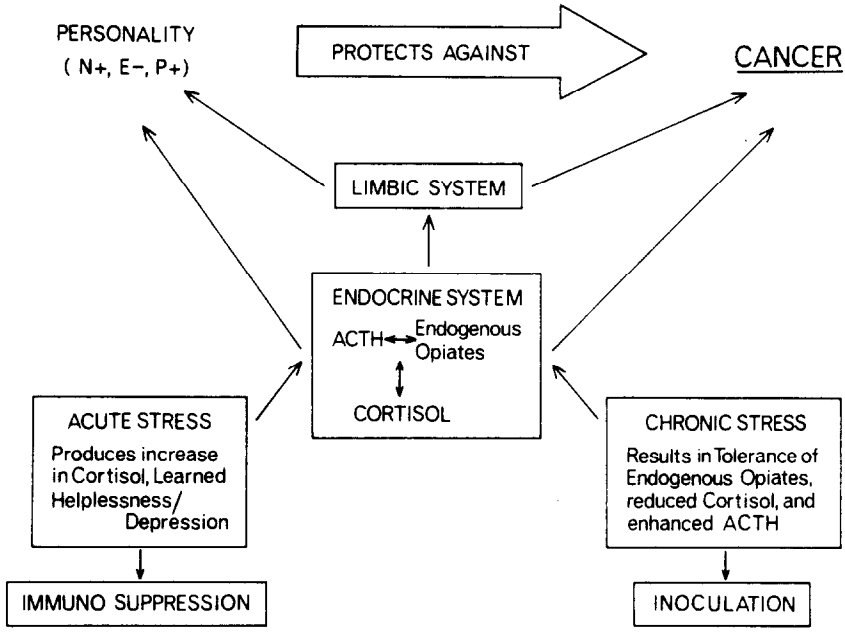

FIG. 1. Hypothetical links between personality, stress, neurohormones, and cancer.

personality traits imply a *chronic* stress, and hence seem to help the individual to become inoculated against stressors.

In the above, we have suggested a causal set of relationships, and this is important because the usual epidemiological type of study relies entirely on the discovery of correlations which may or may not have any kind of causal effect. The virtue of a causal hypothesis is that it leads to experiments which can support or disprove the hypothesis. What is the position here?

Let us first consider the recent work of Rodin (1980, 1986), which is very relevant to this hypothesis. Put briefly, Rodin studied elderly people living in a home subjected to a variety of stresses, which, according to the theory, might lead to helplessness, depression, and death. Rodin introduced methods of coping with the stress involved in various experimental groups, and compared their fate with that of members in control groups receiving either no or placebo instructions and treatments. The outcome was very clear-cut. Subjects who received appropriate instruction in coping behaviour showed significantly higher nurses' ratings on such items as "happy," "actively interested," "sociable," "self-initiating," and "vigorous." They also survived significantly longer than the nontreated subjects. Rodin also looked at cortisol levels, arguing that many deaths in elderly persons can be attributed to a general weakening

of the immune system, since corticosteroids display immunosuppressive properties (Gabrielson & Good, 1967). Indeed, as she points out, there appears to be an inverse relationship between plasma corticosterone level and the capacity of the spleen to synthesize antibodies (Gisler, 1974). In healthy organisms there are usually homeostatic regulatory mechanisms effectively counteracting the suppressive properties of corticosteroids (Nothey, 1965; Rose & Sabison, 1971; Solomon, 1969). These homeostatic regulatory mechanisms may be less effective in the elderly (Timiras, 1972), so that stress without effective coping may have an even more debilitating effect on health in such people, through its effects on the pituitary-adrenal system.

As Rodin points out, "This would occur because the magnitude of pituitary adrenocortical response to stress becomes greater with aging and environmental uncontrollability might exacerbate this condition" (p. 195). To test this hypothesis, and the influence of effective coping on cortisol levels, she measured hypthalamo-pituitary adrenal activity by means of 24-hour urine samples. Three months prior to and four to six weeks after the intervention phase, urine was collected for free-cortisol (USC) measurements, the cortisol being measured in urine by radioimmunoassay. Rodin first noted the initial high level of USC in the whole sample, followed by a significant reduction in the intervention groups, but not the no-treatment group. The self-instructional group, which showed a significant increase in coping responses after treatment as well as increased problem-solving activity and reduced stress from actual problems, showed high correlations between increased perceived control and increased problem-solving activity, with decreases in urinary free cortisol levels of $r = .62$ and $r = .54$, respectively. The groups also, unlike the placebo-treatment groups, did not show a return to baseline after a one-year follow-up as far as cortisol levels are concerned. Thus, Rodin's work clearly supports the general hypothesis under investigation.

Turning now to cancer specifically, rather than survival in general, we shall consider the important work of Grossarth-Maticek, Kanazir, Vetter, and Jankovic (1983a). He was concerned to supplement his prospective studies, which in spite of their superior methodology, could still only result in correlational findings with a more experimental line of research which would give direct evidence on the *casual* role of stress and coping mechanisms. In the first of these studies, women suffering from terminal cancer of the breast were given chemotherapy if they so desired. Of those who declined chemotherapy, half were treated by Grossarth-Maticek; using a type of behaviour therapy called "creative novation therapy." This is a kind of cognitive behaviour therapy specially designed to relieve depression and hopelessness, and encourage the expression of emotions. This method contains elements of Wolpe's (1958) method of desensitization, Beck's (1976) cognitive therapy of emotional disorders, and Lazarus and Folkman's (1984) method of teaching "coping strategies." In his own words, quoting from an unpublished manuscript, "Creative novation

therapy is a form of cognitive behaviour therapy, uniting the principles of learning underlying conventional behavioural techniques with certain cognitive principles."

The therapy is designed to enable the patient to express needs that had previously been inhibited, and to engage in more satisfying social interactions. It is assumed that undesirable behaviour patterns are guided by cognitive-emotional programmes (values and assumptions) which can be modified. Through careful analysis the conflicting needs of the patient are identified. These are considered to be approach–avoidance, conflicts, but are also similar to double-blind conflicts (e.g, "I love my husband who died years ago. I believe that I cannot live without him, therefore I wish to die to be reunited with him. But I also love to live and have good relations with my children"). The next therapeutic step is to define with the patient alternative behaviours and patterns of cognitive interpretation. No attempt is made to dismantle the structure of emotional needs (as in depth psychotherapy), but rather to bring resolution by substitution of new cognitive programmes (e.g., "I love my mother, but I have always thought I would betray her if I loved another woman. Now I realise that I am able to love both at the same time. Therefore I do not feel guilty anymore"). In addition, a programme of complete behavioural changes is developed with the patient, and he or she is encouraged to work on these at home.

The design of the study includes four groups of 25 patients each, as shown in Table 2. Some received both behaviour therapy and chemotherapy, some neither, and some one but not the other.

The table shows main length of survival in months. Clearly the group receiving neither type of therapy, with a mean survival time of 11.28 months, did worst, while the group receiving both therapies did best, with a mean survival time of 22.40 months. Those receiving only one or the other type of therapy showed a mean survival length of about 14.50 months, with the two types of therapy apparently equally successful in prolonging life. It is interesting that the combination of both is clearly synergistic, survival length being greater than the simple addition of the effects of the two types of therapy.

This synergistic effect may be illustrated by reference to the following figures. The mean survival time of all 100 patients was 15.7 months, with a standard deviation of 7.3 months, total survival time varying from 6 to 38 months. The relationship between behaviour therapy, chemotherapy, and survival time was as follows: chemotherapy alone increased survival time by 2.80 months ($p < .001$), while behaviour therapy alone increased survival by 3.64 months ($p < .001$). If the two effects were additive, one would expect a survival time of $11.28 + 2.80 + 3.64 = 17.72$ months for the groups with combined therapies. However, the mean survival time of the chemotherapy plus behaviour therapy group was 22.40 months, exceeding the additive value by 4.68 ($p < .005$). This indicates that a positive interaction between chemotherapy and behaviour therapy has taken place, and that they operate synergistically.

TABLE 2
SURVIVAL TIME IN MONTHS OF TERMINALLY ILL WOMEN SUFFERING FROM CANCER OF THE BREAST AND GIVEN DIFFERENT TYPES OF THERAPY

	Chemotherapy: no	Chemotherapy: yes	totals
Behaviour Therapy: no	mean = 11.28 N = 25	mean = 14.08 N = 25	mean = 12.68
Behaviour Therapy: 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

Additional patients, who had declined chemotherapy, were treated by orthodox behaviour therapy or by psychoanalysis. Results for these methods were significantly worse than for creative novation therapy, and indeed dynamic psychotherapy did significantly worse than no therapy at all.

Is it possible to use creative novation therapy in a prophylactic manner for cancer, as Rodin used the teaching of coping behaviour as a prophylactic treatment for stress in old age? In his Heidelberg prospective study, Grossarth-Maticek, Eysenck, Vetter, and Frentzel-Beyme (1986) used 91 high cancer-prone subjects, 45 of whom were treated by means of creative novation therapy as a prophylactic measure, while the other 46 received no such therapy. Table 3 shows the results. It will be seen that of the patients who received behaviour therapy, none died of cancer, whereas of the control group 12 died. Altogether, 40 in the therapy group are still living, as compared with 25 in the control group. Overall, the difference is significant at the .001 level.

Grossarth-Maticek et al. (1986) carried out a similar study with 82 people who were prone to cardiovascular diseases. Of the 43 people in the therapy group, three died of heart disease, whereas of the 39 in the

TABLE 3
DEATHS FROM CANCER IN GROUPS TREATED PROPHYLACTICALLY BY BEHAVIOUR THERAPY, AND GROUPS NOT SO TREATED

	Cancer-Prone Groups			
	Living	Died	Died of other causes	Total
Control Group	25	12	9	46
Therapy Group	40	0	5	45
Total	65	12	14	91

control group, 14 died. Detailed results are given in Table 4. Again the difference between the two groups was very highly significant at the .01 level. These results obviously need replication, but they do suggest that there is a causal link between personality-related behaviour patterns, their alteration by means of behaviour therapy, and liability to contract various types of diseases.

Apart from the work of Rodin and Grossarth-Maticek, there are other studies which show that it is possible to affect the development of physical disease by using behaviour therapy methods to change habitual behaviour and emotional reactions. Friedman et al. (1984) and Gill et al. (1985) have shown that groups of post-myocardial infarction patients who received group Type A behaviour counselling, in comparison with control groups not receiving such counselling showed significant reduction in Type A behaviour. Cumulative cardiac recurrence rates also were significantly less than that observed in participants not receiving counselling.

It also was found (Gill et al., 1985) that subjects undergoing a profound reduction in the intensity of the Type A behaviour pattern also exhibited a significantly lowered serum cholestrol value as the study continued, as compared with subjects who exhibited no change in their Type A behaviour. Interesting as these data are, they are rather less impressive than those reported by Grossarth-Maticek, possibly because of the less satisfactory definition of the personality correlates involved in the "Type A" concept (Eysenck & Fulker, 1983).

TABLE 4
DEATHS FROM CARDIOVASCULAR DISEASE IN GROUPS TREATED PROPHYLACTICALLY BY BEHAVIOUR THERAPY, AND GROUPS NOT SO TREATED

	Heart-Disease Prone Groups*			
	Living	Died of HD	Died of other causes	Total
Control Group	20	14	5	39
Therapy Group	34	3	6	43
Total	54	17	11	82

* $p < .0090$

The studies mentioned above deal with human subjects, where of course, proper experimental investigations cannot be carried out for ethical reasons, and where one has to rely on observational and epidemiological arguments. The use of behaviour therapy is one possible experimental intervention which resembles a proper experimental design, hence its great value. With animals, on the other hand, it is possible to carry out proper experiments, and some of these have given powerful support for the relationship between stress, disease, and immunosuppression.

In one series of experiments, Sklar and Anisman (1979) injected mice with tumour cells and then gave them 60 shocks. Some of the mice were allowed to terminate each shock by performing an escape response, and thus had a degree of control over the shock. Other mice were given inescapable shocks, and thus had no control. Even though both groups received identical shocks in physical terms, tumour growth was *enhanced* in the inescapable shock group, while the escapable shocks had no effect. Visintainer, Volpicelli, and Seligman (1982) reported similar results for the rejection of Walker's 256 sarcoma in rats.

In another experiment by the Seligman group (Laudenslager, Ryan, Dougan, Hyson, & Maier, 1983), 12 rats were given an average of one escapable shock per minute for a total of 80 shocks. The rats were placed in a small "wheel-turn" box and shock was applied through fixed tail electrodes. Each shock ended when the subject turned the wheel in the front of the chamber. The second group of 12 rats received inescapable shock. Each was paired with an escapable shock subject; shock began at the same time as for the escapable shock subject and ended when the latter responded. A third group was restrained in the apparatus, but was not shocked. Twenty-four hours later all three groups were given five 5-second foot shocks in a shuttle box, and blood was collected immediately afterwards. There was also a home cage control group which received no experimental treatment before blood was collected. Two immune-reaction relevant assays were carried out on the blood. T-cell mitogens Concanavalin A (ConA) and phytohemagglutinin (PHA) were used to stimulate lymphocyte proliferation. Inescapable shock was the only treatment associated with suppression of lymphocyte proliferation. Results are shown in Figures 2 and 3.

The results for the ConA-stimulated cultures were slightly different, with the escapable shock apparently somewhat facilitating lymphocyte proliferation relative to the restrain control condition. However, inescapable shock again depressed lymphocyte proliferation, thus reducing the immune reaction. These results, taken together with those of Weiss (1968) (Weiss et al., 1981), suggest that uncontrollable stressors produce higher levels of corticosteroids than controllable stressors. Thus, there is support for the hypothesis that stress not alleviated by coping mechanisms is responsible for the production of cortisol and other corticosteroids, leading to depression of immune functioning. These and many other animal experiments provide some support for the general theory here put forward.

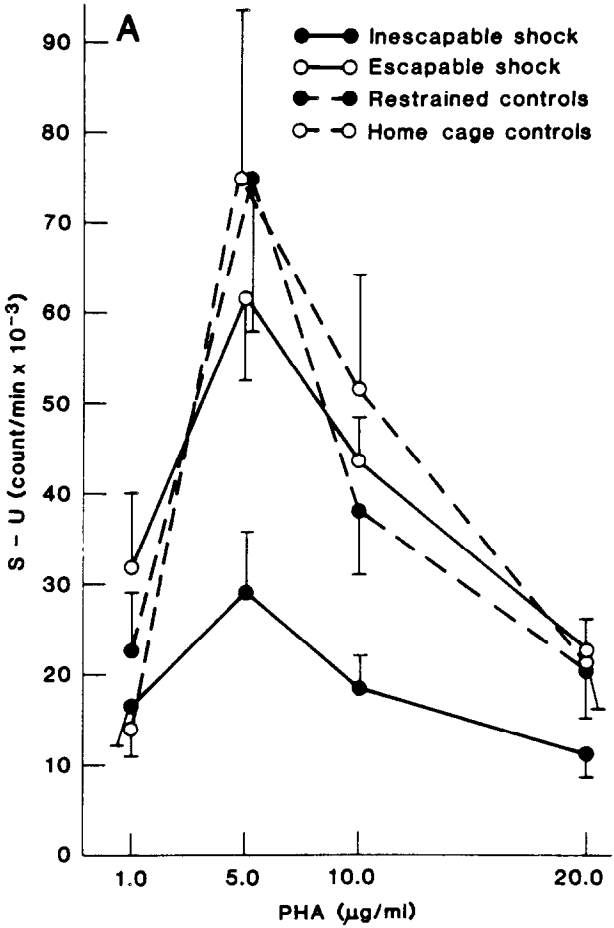

FIG. 2. Suppression of lymphocyte proliferation by inescapable shock, using PHA to stimulate proliferation.

SUMMARY

It will be clear that in relating anxiety and depression to a physical disease, such as cancer, we are confronted with a very complex nomological network which has not been worked out in sufficient detail to enable us to say with any certainty that the underlying theories are correct. Nevertheless, enough has been done to show that the theory in question points in the right direction, as otherwise it would be difficult to explain the many successful tests of deductions from the theory which have been carried out. It may be useful to state theory, deductions, and findings in a sequential manner.

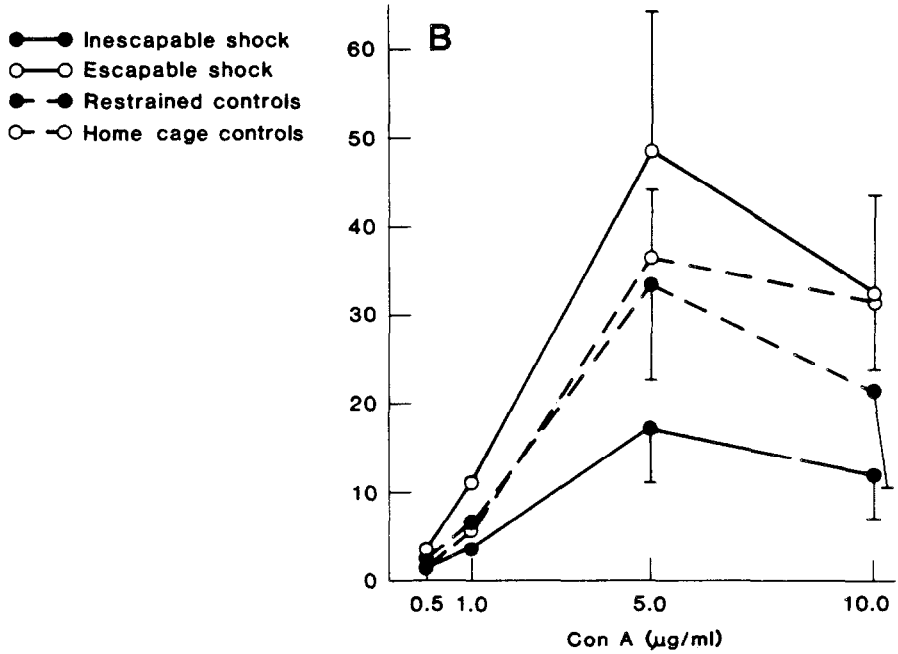

FIG. 3. Suppression of lymphocyte proliferation by inescapable shock using ConA to stimulate proliferation.

1. Malignant growths occur all the time in animals and humans, but are kept in check by the immune system. The immune system declines in effectiveness with age, which explains the rapid increase of carcinomas with increasing age.
2. The workings of the immune system are influenced and to some extent controlled by peptides and hormones. Corticosteroids such as cortisol act in an immunosuppressive fashion, and endogenous opiates may also may be involved in mediating some of the immunosuppressive effects of stress. ACTH, on the other hand, may have an immuno-enhancing effect.
3. Uncontrollable stress ("learned helplessness") is significantly related to the occurrence of disease and death, whereas controllable stress does not seem to act in a similar fashion.
4. Chronic and acute stress also seem to be somewhat dissimilar in their relation to disease and death, with *acute* stress increasing the probability of malignant growths developing, whereas chronic stress, through a process of inoculation, may have the opposite effect.
5. The peptides and hormones mentioned as affecting the immune system also seem to be related to certain behaviour patterns. Thus ACTH is related to anxiety, neuroticism, and introversion, whereas

cortisol is related to depression, hopelessness, and helplessness. This may explain the *positive* relationship between hopelessness/helplessness and cancer, and the *negative* relationship between anxiety, neuroticism and introversion, on the one hand, and cancer, on the other.

6. If there is a causal relationship between the personality traits just mentioned, and the behaviour patterns on which they are built, on the one hand, and the mediation of malignant growth through immunosuppression and immunoenhancement, respectively, then alteration of these behaviour patterns through behaviour therapy should be able to reverse ongoing trends.
7. Creative novational therapy, working along these lines, has, in fact, been shown to act in a prophylactic manner in preventing the occurrence of carcinomas, and prolong the life of incurably ill cancer patients.
8. There is direct evidence that by making uncontrollable stressors controllable, not only is there a significant decrease in death, but there is also a significant decline in cortisol level.

I have not tried in this brief paper to review the large literature relating to each of these points; but have given reference to summary articles which attempt to do so. The issues arising in connection with each of the eight points listed are always complex, and the evidence is not always as clear cut as one might wish. Nevertheless, it seems clear that there are relations between personality, stress, disease, and the immune system, which suggest than traditional medical opinion, which pays no attention to psychosocial factors, has neglected a very important area. The data further suggest that methods of treatment of the psychological kind (behaviour therapy), taking account of these relations, can have powerful prophylactic effects in preventing cancer, and equally powerful effects in prolonging life in incurable patients. Furthermore, these methods of treatment appear to be synergistic with chemotherapy and other traditional medical treatments. These facts seem to suggest the need for further and more rigorous investigations of the theories briefly discussed above.

These developments are part and parcel of an extension of the work of clinical psychologists beyond psychiatry, and into general medical and social problems, as advocated by Eysenck and Rachman (1973). Individual differences in personality, differential reactions to stresses and strains, conditioned emotional reactions, and the interactions between these, neurohormonal processes and immune reactions, the learning of coping strategies and other behavioural methods of treatment, all point to functions of psychological factors going well beyond the traditional psychiatric ones, and extending far into the medical field. Anxiety and depression obviously have effects which go well beyond those we have been dealing with in the past, and should always be seen in the context of their physiological, biochemical, and neurohormonal correlates. Such a

point of view will help to eradicate the traditional Cartesian-type dichotomy between body and mind, a dichotomy that has been disastrous from the philosophical as well as the scientific point of view, and which has prevented psychology and medicine from entering into a more fruitful relationship.

REFERENCES

Abramson, L. Y., Seligman, M., & Teasdale, J. D. (1978). Learned helplessness in humans: Critique and reformulation. *Journal of Abnormal Psychology*, **87**, 49–74.

Bahnson, C. B., & Bahnson, M. B. (1964). Cancer as an alternative to psychosis. In D. M. Kissen & L. LeShan (Eds.), *Psychosomatic aspects of neoplastic disease* (pp. 104–116). Philadelphia, PA: Lippincott.

Bammer, K., & Newberry, B. (1981). *Stress and cancer*. Toronto: Hogrefe.

Beck, A. T. (1976). *Cognitive therapy and the emotional disorder*. New York: International Universities Press.

Borysenko, M., & Borysenko, J. (1982). Stress, behaviour and immunity: Animal models and mediating mechanisms. *General Hospital Psychiatry*, **4**, 59–67.

British Registrar General. (1958). *Decennial Supplement, England and Wales, 1951, Occupational Mortality*. Part 11, Vol. 1. London: HMSO.

Brittain, R. W., & Weiner, N. I. (1985). Neural and Pavlovian influences on immunity. *Pavlovian Journal of Biological Science*, **20**, 181–194.

Brown, F. (1966). The relationship between cancer and personality. *Annals of the New York Academy of Sciences*, **123**, 865–875.

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

Dattore, P. J. (1978). *Premorbid personality characteristics associated with neoplasms: An archival approach*. Unpublished doctoral dissertation, University of Kansas.

Dattore, P. J., Stontz, F. C., & Coyne, L. (1980). Premorbid personality differentiation of cancer and noncancer groups: A test of the hypothesis of cancer proneness. *Journal of Consulting & Clinical Psychology*, **48**, 388–394.

Dobson, C. B. (1982). *Stress: The hidden adversary*. Lancaster: MTP.

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

Eysenck, H. J. (1984a). Personality, stress and lung cancer. In S. Rachman (Ed.), *Contributions to medical psychology* (Vol. 3, pp. 151–171). Oxford: Pergamon Press.

Eysenck, H. J. (1984b). Lung cancer and the stress-personality inventory. In C. L. Cooper (Ed.), *Psychosocial stress and cancer* (pp. 49–71). London: John Wiley & Sons.

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

Eysenck, H. J. (1985b). Smoking and health. In R. Tollison (Ed.), *Smoking and society* (pp. 17–88). Lexington, MA: D.C. Heath.

Eysenck, H. J. (in press). Psychotherapy and clinical medicine: Applications to cancer and cardiovascular disease. *Prescriptive Psychotherapy*. (International journal of the International Academy of Electric Psychotherapists.)

Eysenck, H. J., & Fulker, D. (1983). The components of Type A behaviour and its genetic determinants. *Personality and Individual Differences*, **4**, 499–505.

Eysenck, H. J., & Rachman, S. (1973). The future of clinical psychology. *Bulletin of the British Psychological Society*, **26**, 113–116.

Fox, B. H. (1978). Premorbid psychological factors as related to cancer incidence. *Journal of Behavioral Medicine*, **1**, 45–133.

Fox, B. H. (1983). Current theory of psychogenic effects on cancer incidence and prognosis. *Journal of Psychosocial Oncology*, **1**, 17–31.

Friedman, G. D., 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., & Bourg, E. (1984). Alterations of Type A behaviour and reduction in cardiac references in postmyocardial infarction patterns. *American Heart Journal*, **108**, 237–248.

Gabrielson, A. E., & Good, R. A. (1967). Chemical suppression of adaptive immunity. *Advanced Immunology*, **6**, 91.

Gill, J. J., Friedman, M., Thoreson, C., Powell, L., Vernon, D., Brown, B., & Drews, F. (1985). Reduction in Type A behaviour in healthy, middle-aged Austrian military officers. *American Heart Journal*, **110**, 503–514.

Gisler, R. H. (1974). Stress and the hormonal regulation of the immune response in mice. *Psychotherapy and Psychosomatics*, **23**, 197.

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

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

Grossarth-Maticek, R., Eysenck, H. J., Vetter, H., & Frentzl-Beyme, R. (1986). The Heidelberg prospective intervention study. Paper presented at the First International Symposium on Primary Prevention and Cancer, March 19–22 1986, Antwerp.

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

Grossarth-Maticek, R., Kanazir, D. T., Schmidt, P., & Vetter, H. (1982a). Psychosomatic factors in the process of cancerogenesis. *Psychotherapy and Psychosomatics*, **28**, 284–302.

Grossarth-Maticek, R., Kanazir, D. T., Vetter, H., & Jankovic, M. (1983). *Psychotherapy and Psychosomatics*, **39**, 94–105.

Grossarth-Maticek, R., Kanazir, D. T., Schmidt, P., & Vetter, H. (1985). Psychosocial and organic variables as predictors of lung cancer, cardiac infarct and apoplexy: Some differential predictors. *Personality and Individual Differences*, **6**, 313–321.

Grossarth-Maticek, R., Sigrist, J., & Vetter, N. (1982). Interpersonal repression as a predictor of cancer. *Social Science and Medicine*, **16**, 493–498.

Hodapp, V., & Weyer, G. (1982). Zur Stress-Hypothese der Essentiellen Hypertonie. In D. Vaitl (Ed.), *Essentielle Hypertonie: Psychologische-medizinische Aspekte* (pp. 130–141). Berlin: Springer.

Kissen, D. M. (1963a). Personality characteristics in males conducive to lung cancer. *British Journal of Medical Psychology*, **36**, 27–36.

Kissen, D. M. (1963b). Aspects of personality of men with lung cancer. *Acta Psychotherapentica*, **11**, 200–210.

Kissen, D. M. (1964a). Relationship between lung cancer, cigarette smoking, inhalation and personality. *British Journal of Medical Psychology*, **37**, 203–216.

Kissen, D. M. (1964b). Lung cancer, inhalation, and personality. In D. M. Kissen & L. Le Shan. (Eds.), *Psychosomatic aspects of neoplastic disease*. Philadelphia, PA: Lippincott.

Kissen, D. M. (1964c). The influence of some environmental factors on personality inventory scores in psychosomatic research. *Journal of Psychosomatic Research*, **8**, 145–149.

Kissen, D. M. (1966). The significance of personality in lung cancer in men. *Annals of the New York Academy of Sciences*, **125**, 820–826.

Kissen, D. M. (1967). Psychological factors, personality and lung cancer in men aged 55–64. *British Journal of Medical Psychology*, **40**, 29–34.

Kissen, D. M. (1969). Present status of psychosomatic cancer research. *Geriatrics*, **24**, 129–137.

Kissen, D. M., Brown, R., & Kissen, M. (1969). A further report on personality and psychosocial factors in lung cancer. *Annals of the New York Academy of Sciences*, **164**, 535–545.

Kissen, D. M., & Eysenck, H. J. (1962). Personality in male lung cancer patient. *Journal of Psychosomatic Research*, **6**, 123–137.

Korneva, E. A., Klimenko, V. M., & Shkhinek, E. K. (1985). *Neurohumoral maintenance of immune homeostasis*. Chicago: University of Chicago Press.

Laudenslager, M. L., Ryan, S. N., Dougan, R. C., Hyson, R. C., & Maier, S. F. (1983). Coping and immunosuppression: Inescapable but not escapable shocks suppresses lymphocyte proliferation. *Science*, **221**, 568–570.

Lazarus, R. S., & Folkman, S. (1984). *Stress, appraisal, and coping*. New York: Springer.

LeShan, L. (1959). Psychological states as factors in the development of malignant disease: A critical review. *Journal of the National Cancer Institute*, **22**, 1–18.

Maier, S. F., & Seligman, M. (1976). Learned helplessness: Theory and evidence. *Journal of Experimental Psychology: General*, **105**, 2–46.

Miller, W. R., Seligman, M., & Kurlander, H. (1975). Learned helplessness, depression, and anxiety. *The Journal of Nervous and Mental Disease*, **161**, 347–357.

Morrison, F. R., & Paffanbarger, R. A. (1981). Epidemiological aspects of biobehaviour in the aetiology of cancer: A critical review. In S. M. Weiss, J. A. Herd, & B. N. Fox (Eds.), *Perspectives on behavioural medicine* (pp. 135–162). New York: Academic Press.

Nothey, W. T. (1965). Studies on the interrelationship of cold environment, immunity and resistance to infection. 1. Qualitative and quantitative studies on the immune response. *Journal of Immunology*, **94**, 649.

Perrin, G. M., & Pierce, I. W. (1959). Psychosomatic aspects of cancer: A review. *Psychosomatic Medicine*, **21**, 397–421.

Rodin, J. (1980). Managing the stress of aging: The role of control and coping. In S. Levine & H. Ursin (Eds.), *Coping and health*, (pp. 171–202). New York: Plenum.

Rodin, J. (1986). Health, control and aging. In M. M. Baltes & P. B. Baltes (Eds.), *Aging and the psychology of control*. Hillsdale, NJ: Erlbaum.

Rose, J. E. M. St., & Sabiston, B. H. (1971). Effects of cold exposure on the immunologic response of rabbits to human serum albumin. *Journal of Immunology*, **107**, 339.

Rosellini, R. A., & Seligman, M. (1975). Frustration and learned helplessness. *Journal of Experimental Psychology: Animal Behaviour Process*, **104**, 149–157.

Seligman, M., Rosellini, R., & Kozak, M. (1975). Learned helplessness in the rat: Time versus, immunization, and reversibility. *Journal of Comparative and Physiological Psychology*, **88**, 542–547.

Sklar, L. S., & Anisman, H. (1979). Stress and coping factors influence tumour growth. *Science*, **205**, 513–515.

Solomon, G. F. (1969). Stress and antibody response in rats. *International Archives of Illness*, **35**, 97.

Selzer, C. C., & Jablon, S. (1977). Army rank and subsequent mortality by cause: 23-year follow-up. *American Journal of Epidemiology*, **105**, 559–566.

Taylor, S. (1983). Adjustment to threatening events: A theory of cognitive adaptation. *American Psychologist*, **38**, 1161–1174.

Timiras, P. S. (1972). *Developmental physiology and aging*. New York: Macmillan.

Visintainer, M. A., Volpicelli, J. R., & Seligman, M. E. P. (1982). Tumour rejection in rats after inescapable and escapable shocks. *Science*, **216**, 437–439.

Weiss, J. M. (1968). Effects of coping responses on stress. *Journal of Comparative and Physiological Psychology*, **65**, 251–260.

Weiss, J. M., Goodman, P. A., Nosito, B. G., Coorigan, S., Charry, J. M., & Bailey, W. N. (1981). Behavioural depression produced by an uncontrollable stressor: Relationship to non-epinephrine, dopamine and serotonin levels in various regions of rat brain. *Brain Research Review*, **3**, 167–175.

Weyer, G., & Hodapp, V. (1979). Job stress and essential hypertension. In I. G. Sarason & C. D. Spielberger (Eds.), *Stress and anxiety*. (Vol. 6, pp. 337–349). Washington, D.C.: Hemisphere.

Wolpe, J. (1958). *Psychotherapy by reciprocal inhibitions*. Stanford, CA: Stanford University Press.