generally interacting with them would have beneficial effects; apparently, it is the *content* of the interventions that is the important part of the treatment.

More detailed information concerning our studies of intervention is given by Eysenck 1988a, 1988b, 1989; Grossarth-Maticek, Bastiaans, and Kanazir, 1985; Grossarth-Maticek, Eysenck, and Vetter, 1988; and Grossarth-Maticek, Schmidt, Vetter, and Arndt, 1984. These communications also contain information on the possibilities of prolonging life even after terminal cancer has been diagnosed. It seeems that the use of behavior therapy can delay deaths from cancer and almost double the duration of survival.

Psychological Treatment in Cancer

Table 14 shows data from study to support this assertion (Grossarth-Maticek, 1980a). Twenty-four pairs of patients were formed who suffered from terminal cancer. They were matched on type of cancer, stage of growth, type of treatment, sex, and age; members of each pair were then allocated to treatment or control on a random basis. It will be seen that psychologically treated patients survived 5.07 years as compared with 3.09 years for controls.

In another study (Eysenck, 1988b; Grossarth-Maticek, 1980a), we studied 100 women suffering from terminal cancer of the breast. Half received chemotherapy, half did not; of these two groups, half received psychological therapy, half did not. Of those who received neither type of therapy, duration of survival was 11.28 months. Those who only had chemotherapy survived 14.08 months, while those who received only psychological therapy survived 14.92 months. Statistically, both effects are significant, but the combined effect (survival for 22.40 months) was significantly stronger than the simple addition of the two individual therapy effects would have suggested.

It is sometimes said that the results of behavior therapy in cancer and CHD, either prophylactically or in prolonging life, are too good to be true, yet much outside evidence supports the efficacy of different types of psychological therapy on cancer and CHD, some of which reports results even better than those described here. To take only the most recent study, Spiegel, Bloom, Kraemer, and Gottleib (1989) doubled the life expectancy of female patients with metastatic breast cancer, a result even better than similar studies reported by us (Eysenck, 1988a, 1988b). In the control group of Spiegel et al., life duration was 18.9 months, while in the therapy group it was 36.8 months. Such findings should be seen in the context of studies looking at the influence of psychosocial factors and interventions on the immune system—which presumably mediates the effects of life events, stress, and therapeutic psychological intervention—and on the occurrence

TABLE 14. Duration of survival of treated and control groups

Type of cancer	Number of pair of patients	Survival time, years		Age		
		Therapy group	Control group	Sex	Therapy	Control
Scrotal cancer	1	5.8	3.2+	M	34	35
Stomach cancer	1	4.8	1.8+	M	64	63
	2	2.4	2.3+	M	59	59
Bronchiolar	1	1.7	2.4−	M	42	42
	2	5.6	1.5+	M	59	60
	3	4.2	1.6+	M	60	60
	4	3.2	1.1+	M	47	46
	5	1.7	1.7=	M	39	39
	6	4.5	1.2+	M	58	58
	7	5.2	1.0+	M	63	64
Corpus uteri	1	6.8	4.2+	F	64	65
	2	4.5	4.8−	F	66	66
	3	7.2	3.5+	F	49	48
	4	8.2	3.1+	F	50	51
Cervical	1	5.5	4.2+	F	41	41
	2	6.1	4.0+	F	46	46
	3	3.2	3.3−	F	38	37
	4	4.5	4.1+	F	50	49
	5	2.8	3.6−	F	39	40
Colon and rectum carcinoma	1	9.5	4.2+	M	64	64
	2	7.5	2.1+	F	56	56
	3	6.3	4.9+	M	55	56
	4	4.8	4.3+	F	61	60
	5	5.7	4.1+	F	52	52
Total	24	5.07	3.09			

Note. Data from "Social Psychotherapy and Course of the Disease" by R. Grossarth-Maticek, 1980, *Psychotherapy and Psychosomatics*, *34*, p. 136.

and rigor of cancer. Before turning to a consideration of this evidence, it may be useful to consider studies suggesting the possibility of prophylactic intervention in CHD-prone probands.

Stress Management

Johnston (1989) has reviewed the literature in a very critical spirit but concludes that the evidence for the view that stress management may reduce CHD through the lowering of a large number of stress-related risk factors by moderate, or even small, amounts "is patchy, but a much stronger case can be made than would have seemed possible only ten years ago" (p. 277). The cautions optimism of this quotation is mainly based on

the contributions of M. Friedman (1987); M. Friedman et al. (1984, 1986); Gill et al. (1985); Lovibond, Birrell, and Langeluddecke (1986); and Patel et al. (1985). Most convincing are the data presented by the recurrent coronary prevention project. If the results are less striking than those reported by Grossarth-Maticek, Eysenck, and Vetter (1988), this may be due to the fact that the validity of the type A concept is probably much lower than that of the Grossarth-Maticek type 2, that is, the CHD-prone type (Booth-Kewley & Friedman, 1987). It is also noteworthy that the theory linking stress with cancer and stress reduction with cancer survival has been elaborated much more convincingly (through the innervation of the immune system) than can be said of the relation between stress and stress management and CHD.

Bennett and Carroll (1990) similarly conclude a review of the evidence by saying that stress management techniques "not only reduce individual risk factors, they can also reduce mortality and morbidity to CHD" (p. 1). They also conclude that "risk factors combine multiplicatively, and small decreases on a number of risk factors may reduce the risk of CHD more than if only one risk factor is targeted (Johnston, 1989; D.A. Perkins, 1989), as in most medical interventions" (p. 81). These conclusions are very much in line with our own studies just detailed.

Finally, the conclusions drawn by Taylor (1990) in his review of health psychology, also support this view: "Research that examines whether or not psychological and social factors are involved in health and illness has largely made its point." (p. 46) He goes on to say that investigations have addressed the direct impact of stress and other psychological states on physiological processes, the impact of psychological and social factors on risky health practices, and the impact of psychological and social factors on how people respond to potential illness states, such as whether or not they engage in appropriate illness behavior. He concludes that the field has advanced to an unprecedented level of complexity in research investigations. . . . and that health psychology affords the opportunity to look beyond particular disorders to the broad principles of thought and behavior that cut across specializations of diseases or problems studied to elucidate more fundamental psychosocial mechanisms.

Some of the resulting complexities are illustrated in the work of Brown and McGill (1989) and Scheier et al. (1989). They highlighted the importance of optimism in the recovery of patients from coronary artery disease bypass surgery, demonstrating again the interaction of psychological states, personality, and medical illness or recovery. Brown and McGill demonstrated the complex interaction of personality traits such as optimism, which is closely linked with extraversion (Eysenck & Eysenck, 1985) and stress.

Positive life events are generally believed to have beneficial effects on health, but apparently this is only true when probands have a positive self-concept (optimism). Brown and McGill (1989) outline an identity-

disruption model of stress, which holds that an accumulation of life events that are inconsistent with the self-concept leads to illness. Thus, positive life events in probands with a negative, pessimistic self-concept were predicted (and found) to predict the development of illness over time. Clearly, oversimplified concepts of "stress" may lead to erroneous predictions and may account for many failures to replicate in the literature.

The findings of Brown and McGill (1989) find support in an experimental study by Brebner (1990), who showed that introverts tend to generalize experiences of failure, extraverts experiences of success, thus developing pessimistic or optimistic personality traits. Brebner considers such generalization of failure experiences as an important form of stress, but of course, it is not usually mentioned in traditional stress inventories. It is noteworthy that the characteristics of cancer-prone probands agree to a considerable extent with those of introverts, while CHD probands tend to show more the characteristics of extraverts (Eysenck, 1990b). These apparent relationships between predisposition to disease and well-established personality types are well worth following up along theoretical and experimental lines.

One additional study that confirms the importance of an optimistic, extraverted attitude in mediating beneficial rather than negative health effects has looked at the effects of alcohol consumption, with special emphasis on the *reasons* for drinking (Grossarth-Maticek & Eysenck, in press-c). A group of 1,700 male subjects were tested and followed up for 13 years, when death and cause of death were established. A questionnaire administered at the beginning of the study served to classify drinkers as S-drinkers (drinking to drown their sorrows) or P-drinkers (drinking for pleasure, to celebrate success, for enjoyment). Stress or absence of stress was also noted on the basis of an interviewer-administered questionnaire. Results are shown in Table 15. The table shows that P-drinkers, with or without stress, have a lower mortality than nondrinkers, while S-drinkers, with or without stress, do significantly worse than nondrinkers. These results remain when level of drinking is controlled. It is quite likely that in studying the health effects of smoking, too, one should pay attention to the *reasons* for smoking (Eysenck, 1973; Spielberger, 1986); there might be a similar division there between those who smoke for pleasure and those who want to control their tenseness and anxiety. This is another area disregarded by adherents of present-day "orthodoxy."

Possibly also related to "optimism," and certainly to extraversion (Eysenck & Eysenck, 1985; is mobility (walking, jogging, athletics, sport), and here, too, there is evidence that sports are good for you. Grossarth-Maticek et al. (1990) have shown that people who are actively engaged in sports have enhanced longevity, as compared with people who are not so engaged. Worse off were those who started off in sport but gave up in middle age. It is noteworthy that in our therapy groups, there was a general tendency for probands after therapy to increase significantly participation

TABLE 15. Mortality of drinkers and abstainers as a function of stress and motivation for drinking

	With stress (n = 203) (%)	Without stress (n = 201) (%)
No alcohol		
Cancer	25 (12.3)	8 (3.9)
CHD	23 (11.3)	7 (3.4)
Other causes of death	26 (12.8)	12 (5.9)
Total	74 (36.5)	27 (13.4)
Still living	129 (63.5)	174 (86.6)

Alcohol Consumers	Pleasure drinkers n = 191 27.4%		Sorrow drinkers n = 506 72.6%		Pleasure drinkers n = 481 79.5%		Sorrow drinkers n = 124 20.5%	
		(27.4)		(72.6)		(79.5)		(20.5)
Cancer	18	(9.4)	87	(17.1)	23	(4.7)	11	(8.8)
CHD	11	(5.7)	59	(11.6)	12	(2.5)	7	(5.6)
Other causes of death	20	(10.4)	93	(18.3)	23	(4.7)	17	(13.7)
Total	49	(25.7)	239	(47.2)	58	(12.1)	35	(28.2)
Still living	142	(74.3)	267	(52.8)	423	(87.9)	89	(71.8)

CHD, coronary heart disease.
Note. Unpublished data, R. Grossarth-Maticek and H.J. Eysenck

in sports; there was no such tendency to give up smoking (Eysenck and Grossarth-Maticek, 1991).

A Causal Link Between Cancer and Personality

A brief outline may here be given of the way the connection between personality–stress and disease may be mediated by hormonal and physiological factors. A more detailed outline is given elsewhere (Eysenck, 1986). Figure 9 illustrates the assumed causal pathway. Personality (type 1) and stress combine and interact to produce feelings of helplessness, hopelessness, and depression; these in turn produce hormonal and other reactions of which cortisol is here given as the representative (others are the endogenous opiates, adrenocorticotropic hormone, etc.). These in turn produce immune deficiency, which allows budding cancers to develop. The well-established fact that immune reactions can be conditioned along classic lines suggests one possible way such reactions may be learned (Ader & Cohen, 1975; Solvason, Ghanta, & Hiramoto, 1988). A good deal of evidence supports such a model.

The model owes much to a similar one by Solomon (1985; Solomon, Levine, & Kraft, 1968; Solomon & Moos, 1964), who has argued powerfully for the concept of an "immunosuppression-prone" personality

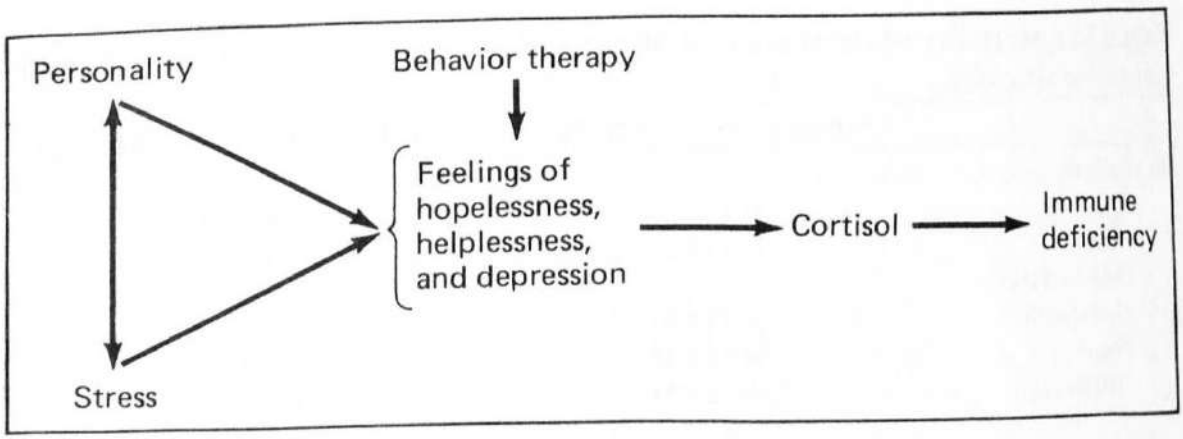

FIGURE 9. The assumed caused pathway whereby hormonal and physiological factors mediate the connection between personality, stress, and disease.

(Solomon, 1985). Having surveyed the literature, he produced first 35, and later another 30 postulates, many of which are relevant to this discussion. The main postulates of interest follow, numbered in sequence, i.e. differently from the way they are stated by him.

Solomon's Postulates (1987)

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

9. Altered CNS neurotransmitter receptor-site sensitivities believed to be associated with mental illnesses should be reflected in lymphocyte receptors.
10. The "functional" modes of expression of CNS and immune system should be similar.

Linn, Linn, and Jensen (1981) have shown that stress and anxiety are associated with depressed immunological response. Levy (1985) and Levy, Herberman, Lippman, and d'Angelo (1987) found that natural killer (NK) cell activity in breast cancer patients was strongly correlated with psychosocial stress indicators, which accounted for 51% of the baseline NK activity variance. Green and Green (1987) reported that relaxation increases salivary immunoglobin A1. Bandura, Cioffi, Taylor, and Brouillard (1988) found that perceived self-inefficacy in exercising control over cognitive stressors activated endogenous opioid systems. Kiecolt-Glaser, Rickers, et al. (1984) found that distressed and lonely probands had significantly higher cortisol levels and a lower level of NK cell activity. Glaser et al. (1986) discovered stress-related impairments in cellular immunity, and Glaser and Kiecolt-Glaser (1985) found that even "relatively mild stress" depressed cellular immunity in healthy adults. Kiecolt-Glaser, Rickers, et al. (1984) found that high scorers on stressful life events and loneliness had significantly lower levels of NK cell activity. Herberman (quoted in Solomon, 1985); Irwin, Vale, and Britton (1987); Nemeroff et al. (1984); and Rou, Rose, Sunderland, Moritisa, and Murphy (1988) found impaired immune reaction in depressed groups. Linn, Linn, and Klimas (1988); Arnetz et al. (1987); Glaser, Kiecolt-Glaser, Speicher, and Holliday (1985); and Shavit, Lewis, Terman, Gale, and Leibeskind (1989) found impaired immune reactions to stress. Jemmott and Magloire (1988) found that stress lowered salivary concentrations of S-IgA, while social support increased them. Grossarth-Maticek and Eysenck (1989b) found that behavior therapy significantly increased the percentage of lymphocytes in terminally ill women suffering from cancer and also increased their survival time. Pennebaker, Kiecolt-Glaser, and Glaser (1988) found that self-disclosure improved cellular immune functioning. Kiecolt-Glaser et al. (1985) found an enhancement of immunocompetence by relaxation and social contact.

Irwin, Daniels, Bloom, Smith, and Weiner (1987) have shown that life events can cause depression and can reduce the effectiveness of the immune function. Similarly, Murphy, Monson, Sobol, and Leighton (1987), in a prospective study of 1,003 adults, found a significant correlation between depression and mortality. Rodin (1984, 1986) showed that appropriate psychotherapy reduced depression *and* cortisol level through psychotherapy. Dabbs and Hopper (in press) showed that cortisol levels correlated with anxiety, depression, and high heart rate.

Of particular interest in relation to this theory are, of course, studies of cancer patients in which physiological treatment shows both improvement

in psychological status and immune function when treatment patients are compared with control patients. Cousins (1989) summarizes one such study, which showed a significant decline in depression and psychological distress generally and a significant increase in "quality of life" scores; at the same time the functioning of the immune system exhibited significant improvement as shown by the number of immune cells in the NK cells family. (This of course is not necessarily a good index of immune functioning). A paper by Levy, Herberman, Lippman, and d'Angelo (1987) has already been mentioned, relating stress factors with sustained depression of NK cell activity, but equally important is another paper by Levy and her colleagues (Levy, Herberman, Maluish, Schlien, and Lippman, 1985) on prognostic risk assessment in primary breast cancer by behavioral and immunological parameters. They found that depressed cancer patients tend to have poorer NK-cell activity and greater likelihood of tumor spread. Also relevant is an article by Temoshok (1985), relating psychological and immune-system response to cutaneous malignant melanoma. She found that patients whose attitudes and emotions were active instead of passive exhibited better immune function and slower tumor spread.

Another relationship is between social support and immune function. Thus, in a community sample of the elderly, higher levels of social support were associated with total lymphocyte count and the ability of lymphocytes to subdivide when stimulated with mitogen (Thomas, Goodwin, & Goodwin, 1985.) Of particular interest is a quite recent study by Baron, Cutrona, Hicklin, Russell, and Lubaroff (1990), who investigated the effect of social support on immune functioning among spouses and cancer patients. They found that participants who had greater social support had faster T-cell proliferation when stimulated by the mitogen PHA and also were more effective in destroying target tumor cells in comparison with individuals who were below the median on reported social support. Neither the incidence of negative life events nor the existence of depressive symptoms were found to mediate this relation, but perhaps personality variables might have done so; the medium involved in producing these effects is at present unknown.

Pennebaker (1985, 1989) has described the way in which inhibition can be viewed as a chronic stressor, resulting in chronic autonomic and corticol arousal. This, in turn, leads to endocrinal activity that compromises the immune system, increasing susceptibility to disease. Pennebaker (1989) and his colleagues have reported that inhibiting one's desire to confide about traumatic events is associated with heightened electrodermal responding, decreased immunocompetence levels, and increase in disease. Other studies demonstrating the role of inhibitory mechanisms in decreased immune functions and in the development of disease (Cox & McKay, 1982; Jemmot, 1987) support this view, as does McClelland (1989).

Finally, the relationship between mood and the immune-system response has been established in a series of studies (e.g., Baker, 1987; Dillon & Baker, 1985–1986; Linn, Linn, & Jensen, 1984; McClelland, Floor, Davidson, & Saron, 1980; McClellon, Ross, & Patel, 1985; Stone, Cox, Valdimarsdottir, Jandorf, & Neale, 1987). Animal studies, too, have contributed to the formulation of the model (e.g., Borysenko & Borysenko, 1982; Glaser, Thorn et al., 1985; Laudenslager, Ryan, Drugan, Hyson, & Maier, 1983; and for a review, Justice, 1985.)

The studies cited are among only the most recent; for reviews of the older and perhaps less convincing material, the following are suggested: Antoni (1987); Baker (1987); Jemmott and Locke (1984); Kennedy, Kiecolt-Glaser, and Glaser (1988); Korneva, Klimenko, and Shkhinek (1985); N. Miller (1983, 1985); Plotnikoff, Faith, Murgo, and Good (1986); Steptoe (1989); and Teshina (1986). Taking all the published data together, they do seem to support the sort of model suggested by Eysenck (1986) Dilman and Ostroumova (1984), and Kanazir et al. (1984) and briefly outlined previously. There is evidence that (a) personality and stress produce immunodestructive substances in the bloodstream; (b) that these substances do have such an immunodestructive function, and that (c) behavioral manipulations can reverse this process. Thus, there appears to exist at least a preliminary model to explain along causal lines the effectiveness of behavior therapy in prophylaxis for cancer and in prolonging life in cancer sufferers.

There is one apparent objection to this argument. As Zonderman, Costa, and McCrae (1989) have shown, there is no evidence in a nationally representative sample for any correlation between depressive symptoms and cancer morbidity. The answer to this is very simple. Depression is a multifaceted set of symptoms, like fever, which may have diverse causes and relate to different disorders; the difference between reactive and endogenous depression is perhaps the best known. The type of depression referred to in the theory discussed here is subclinical and might be defined as "hopelessness depression" (Alloy, Abramson, Metalsky, & Hartlage, 1988). This concept is largely based on the work of Seligman (1975) and L.Y. Abramson, Seligman, and Teasdale (1978) and is essentially a cognitive diathesis-stress theory of depression (Alloy, Clements, & Kolden, 1985). According to this theory, "a proximal sufficient cause of depression is an expectation that highly desired outcomes are unlikely to occur, or that highly aversive outcomes are likely to occur, and that no response in one's repertoire will change the likelihood of occurrence of these outcomes" (Alloy et al., 1988, p. 7). It is in this sense that the term has been used in our research. Other varieties of depression may or may not be relevant, and it is important to note that animal work has also emphasized the importance of differentiating between escapable and inescapable shocks and the vital contribution of predictability (S.M. Miller, 1981).

CHD and Sclerosis: Psychological Therapy Effects

As far as CHD is concerned, there is less material to review, but sclerosis is an obvious intermediary. Grossarth-Maticek, Eysenck, Gallasch, Vetter, and Frentzel-Beyme (in press) have reported a study in which 100 cancer-prone and 92 CHD-prone probands had the degree of sclerosis in the fundus of the eye measured on a 3-point scale by a leading ophthalmologist, before and after therapy (for a randomly selected 50% of probands in each case) and at similar points of time for probands in the control group. Figure 10 shows the results. Type 2 probands had significantly *higher* levels of sclerosis than type 1 probands, and the therapy group had a significantly *lower* degree of sclerosis; more so for CHD-prone type 2 than for cancer-prone type 1 probands. This experiment is in urgent need of replication.

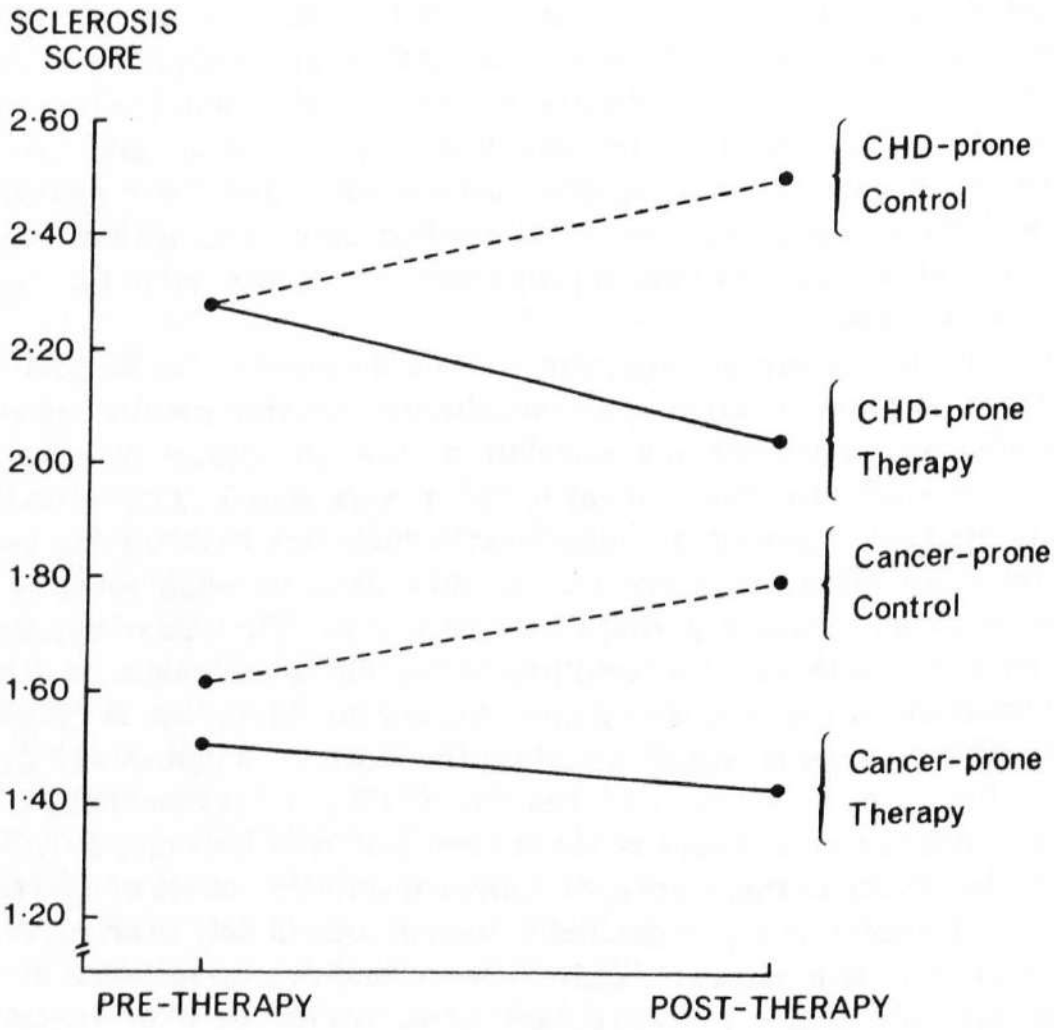

FIGURE 10. Sclerosis in cancer-prone and coronary heart disease-prone probands and effects of behavior therapy Heidelberg study. (*Note*. From "Changes in Degree of Sclerosis as a Function of Prophylactic Treatment in Cancer-Prone and CHD-Prone Probands" by R. Grossarth-Maticek, H.J. Eysenck, B. Gallasch, H. Vetter, and R. Frentzel-Beyme, in press, *Behaviour Research and Therapy*. Copyright 1991 by H.J. Eysenck. Reprinted by permission.)

It is of course necessary to preserve caution in interpreting the results shown in Figure 10. As in the case of epidemiology generally, there are no singular causes producing single results. Thus, Cobb and Rose (1973) showed that air traffic controllers have a higher incidence of hypertension than controls and produce the condition at a younger age. This could be interpreted as a direct effect of the greater stress under which air traffic controllers work. Hypertension, however, is strongly related to alcohol consumption in air traffic controllers (De Frank, Jenkins, & Rose, 1987). It is possible that the drinking may be the important determinant, because alcohol intake is known to raise blood pressure (MacMahon, 1987). Complications of this kind are the rule in this field, rather than the exception.

Clearly, the Grossarth-Maticek and Eysenck data concerning the prophylactic effects of behavior therapy for cancer and CHD are equally subject to this caution. We have shown (Grossarth-Maticek et al., in press) that after therapy there is a very significant shift from type 1 or type 2 behavior to type 4 behavior; there is no change in smoking habits. As Pearl (1925) has pointed out, however, there is a life-style that embraces many different behavior patterns, and changing one may change many others, one of which, or any combination of which, can carry the burden of changes in cancer proneness or CHD proneness. Research is only at the beginning of the scientific study of these complex nomological networks and should not pretend to a greater or more secure understanding of these causal effects than in reasonable under the circumstances.

In Eysenck (1990a), I have discussed the social implications of this type of work. Clearly, preventive medicine is much more humane in its consequences, as well as much cheaper, than traditional medicine, which waits until the disease has manifested itself before attempting any intervention. Many practical, social, and ethical problems are raised when one considers the possibility of introducing methods of this kind for the prevention of cancer and CHD into the social systems at present concerned with health, but it is time that the issues were taken seriously and debated in a meaningful fashion.

What is most important from the point of view of this book, of course, is the demonstration of the heightened probability of a *causal* relation between stressed personality and cancer and CHD. The failure to demonstrate such a relationship between smoking and disease, shown in previous chapters is the most vulnerable point in the orthodox view. These results show that it may be possible to demonstrate causal relations following accepted scientific methodology and to establish personality and stress as important risk factors in cancer and CHD.