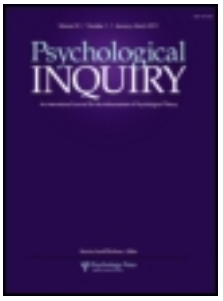

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TARGET ARTICLE

Personality, Stress, and Disease: An Interactionist Perspective

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It is argued that there is now sufficient evidence to regard psychosocial variables, in particular personality and stress, as important risk factors for cancer and coronary heart disease (CHD), equal in importance to smoking, heredity, cholesterol level, blood pressure, and other physical variables. Furthermore, it is now clear that both types of factors act synergistically; that is, each by itself is relatively benign, but their effects multiply to produce high levels of disease. Last but not least, it is argued that there is now good evidence to show that psychological treatment can modify a person's reaction to stress, so that risk of cancer and CHD can be greatly diminished, and duration of survival significantly increased in those terminally ill with cancer. Psychological influences on physical diseases are much greater than suspected in the past; we are only now beginning to trace the causal pathways.

There is a long history of belief in the existence of a cancer-prone personality, characterized by a tendency to suppress the expression of emotions, and an inability to deal with stress, leading to feelings of hopelessness/helplessness, and finally of depression (Eysenck, 1985). There is also a long history of belief in the existence of a coronary heart disease (CHD)-prone personality, characterized by strong, easily aroused feelings of anger, hostility, and aggression (Eysenck, 1990). Most of the published studies have followed the example of Kissen and Eysenck (1962) in studying the personality of patients already ill, comparing them with patients suffering from more benign forms of disease, a methodology that opens the door to arguments that perhaps the disease process has led to cancer-prone or CHD-prone personality, rather than the other way around.

Ideas concerning the importance of personality and stress have in recent years been incorporated into several theories leading to highly focused investigations. That Type A behavior might be related to and predictive of CHD has received a good deal of attention (Rosenman & Chesney, 1980), but reviews such as those by H. S. Friedman and Booth-Kewley (1987) have suggested that only certain traits of the Type A personality, such as anger and aggression, might be related to CHD (Eysenck, 1991). Our own work (Grossarth-Maticek, Eysenck, & Vetter, 1988) certainly supports this view.

As regards the cancer-prone personality, this has often been described as appeasing, unassertive, overcooperative, overpatient, harmony seeking and conflict avoiding, compliant, and defensive (Baltrusch, Stangel, & Waltz, 1988). The two most frequently noted characteristics are (a) suppression of emotional expression, and denial of strong emotional reaction, and (b) failure to cope successfully with stress, and the reaction of giving up, linked with feelings of hopelessness and helplessness (Baltrusch et al., 1988; Eysenck, 1985). This type has sometimes been called "Type C," to distinguish it from the CHD-prone Type A and the healthy Type B (Temoshok, 1987).

The CHD-prone personality does seem to have some of the characteristics of Type A, but, on the whole, Type A has not been found very prognostic of CHD (Eysenck, 1990). Only anger, hostility, aggression, and a generally contumacious attitude have been found to give positive predictions in this field (H. S. Friedman & Booth-Kewley, 1987).

Personality as a Predictor of Cancer and CHD

In several prospective studies, we have avoided the difficulty of disease affecting personality by selecting physically healthy probands at the beginning of the study, ascertaining details concerning personality, stress, smoking and drinking habits, cholesterol level, blood pressure, and blood sugar by interview, questionnaire, and measurement, then following up probands for 10 years before noting mortality and cause of death (Eysenck, 1987a, 1987b, 1987c, 1988a, 1988b; Grossarth-Maticek, 1980a, 1980b; Grossarth-Maticek, Eysenck, Vetter, & Schmidt, 1988). Three major studies have been recorded—the first carried out in Yugoslavia, the other two in Heidelberg, Germany. In each case, cause of death was ascertained by death certificate.

The personality inventory used has been published elsewhere (Grossarth-Maticek, Eysenck, & Vetter, 1988). Essentially, it divides the population into four types: Type 1 is cancer prone according to theory; Type 2 is CHD prone; Type 3 is alternating between behaviors characteristic of Types 1, 2, and 4 and is thereby protected to some extent; and Type 4 is a healthy, autonomous type hypothesized to survive best. Interview ratings and other types of inventory were also used, but gave essentially similar results and are not discussed here. Table 1 shows the results of the first of these studies. It is clear that Type 1 probands die mainly from cancer, Type 2 from CHD, whereas Type 3 and particularly Type 4 probands show a much lower death rate.

Table 2 shows similar results for a Heidelberg proband

Table 1. *Personality Type and Death From Various Causes in the Yugoslav Study*

Group	*n*	Living (%)	Died of: Cancer (%)	CHD (%)	Other Causes (%)
Incomplete	18	33.3	11.1	22.2	33.3
Type 1	316	30.4	36.1	10.4	23.1
Type 2	342	33.3	9.4	24.9	32.5
Type 3	260	58.5	4.6	9.6	27.3
Type 4	417	87.8	1.4	2.2	8.6
Total	1,353	54.2	12.3	11.5	22.0

Notes: Coefficient of association = .34. From "Personality Type, Smoking Habit and Their Interaction as Predictors of Cancer and Coronary Heart Disease" by R. Grossarth-Maticek, H. J. Eysenck, and H. Vetter, 1988, *Personality and Individual Differences, 9,* p. 486. Copyright 1988 by H. J. Eysenck. Adapted by permission.

group representing a quota sample of the population, but with an age limit—young probands were excluded because they were unlikely to contract cancer or CHD in the next 20 years. This group was some 10 years younger than the Yugoslav group, which was selected on the basis of being the oldest inhabitant of randomly chosen households. There are consequently significantly fewer deaths in this group, but the comparison between Type 1 and Type 2 probands shows similar differences.

Table 3 shows results for the second Heidelberg group. Members were similar in age and sex composition to those of the original Heidelberg group, but were chosen as being severely stressed, on the basis of reports of members of the original Heidelberg study who suggested suitable friends and relatives to take part in the investigation of stressed probands. It will be seen that, whereas 89% survived in the normal group, only 54% survived in the stressed group, a difference of 35% presumably due to the effect of stress. Comparative total percentages for deaths from cancer are 3% versus 19%; for deaths from CHD, 3% versus 12%. For other causes, the figures are 5% versus 15%. Clearly, stress is a powerful cause of death (Eysenck, 1987a, 1987b).

Table 2. *Personality Type and Death From Various Causes in the Heidelberg Study*

Group	*n*	Living (%)	Died of: Cancer (%)	CHD (%)	Other Causes (%)
Incomplete	6	100.0	—	—	—
Type 1	194	82.5	9.8	1.5	6.2
Type 2	227	72.7	4.4	10.1	12.8
Type 3	184	99.5	—	—	0.5
Type 4	261	99.2	—	0.4	0.4
Total	872	88.6	3.3	3.1	4.9

Notes: Coefficient of association = .24. From "Personality Type, Smoking Habit and Their Interaction as Predictors of Cancer and Coronary Heart Disease" by R. Grossarth-Maticek, H. J. Eysenck, and H. Vetter, 1988, *Personality and Individual Differences, 9,* p. 486. Copyright 1988 by H. J. Eysenck. Adapted by permission.

Table 3. *Personality Type and Death From Various Causes: Heidelberg Study of "Stressed" Probands*

Group	*n*	Living (%)	Died of: Cancer (%)	CHD (%)	Other Causes (%)
Incomplete	10	80.0	10.0	10.0	—
Type 1	421	40.8	32.6	9.9	16.7
Type 2	317	53.0	10.7	17.7	18.6
Type 3	213	75.1	8.9	7.0	8.9
Type 4	76	73.7	7.9	7.9	10.5
Total	1,042	54.3	19.1	11.5	15.1

Notes: Coefficient of association = .21. From "Personality Type, Smoking Habit and Their Interaction as Predictors of Cancer and Coronary Heart Disease" by R. Grossarth-Maticek, H. J. Eysenck, and H. Vetter, 1988, *Personality and Individual Differences, 9,* p. 487. Copyright 1988 by H. J. Eysenck. Adapted by permission.

These three studies are mentioned only briefly because they have been documented in detail elsewhere (Eysenck, 1988a, 1988b; Grossarth-Maticek, Eysenck, & Vetter, 1988). They also showed that, as a predictor of disease, personality type was about six times more effective than smoking, cholesterol level, and blood pressure, singly or in combination. The data also indicated that the relationship between physical variables, like smoking, and psychosocial variables, like personality type, was synergistic. Consider Table 4, which shows data from the Yugoslav and the Heidelberg stressed groups for deaths from lung cancer; there were too few of these in the Heidelberg normal group to include it.

If we combine the two studies in Table 4, we find the following results. Among nonsmokers, as expected, there are very few deaths from lung cancer, but, of the 13 that occur, 10 occur in persons of Type 1. For smokers, there are 74 deaths, only 6 of which occur in persons other than Type 1. These results give rise to an association between Type 1 and lung cancer of $p = .0001$ for both samples considered. It is clear that quite independent of smoking, individuals of Type 1 are cancer prone, as compared with individuals of

Table 4. *Smoking and Personality Type as Risk Factors in Lung Cancer*

Group	Yugoslavia Deaths from: Lung Cancer	Other Causes	Total	Heidelberg (Stressed) Deaths From: Lung Cancer	Other Causes	Total
Nonsmokers						
Type 1	1 (0.8%)	118	119	9 (3.8%)	227	236
Others	0	550	550	3 (1.0%)	297	300
Smokers						
Type 1	31 (16.9%)	153	184	37 (14.6%)	216	253
Others	6 (1.2%)	482	488	0	247	247

Note: From "Personality Type, Smoking Habit and Their Interaction as Predictors of Cancer and Coronary Heart Disease" by R. Grossarth-Maticek, H. J. Eysenck, and H. Vetter, 1988, *Personality and Individual Differences, 9,* p. 488. Copyright 1988 by H. J. Eysenck. Adapted by permission.

Types 2, 3, and 4 (see also Grossarth-Maticek, Eysenck, & Vetter, 1988).

Table 4 also makes clear that there is a *synergistic interaction* between smoking and typology. The only group having a high proportion of deaths from lung cancer is that of smokers of Type 1; smokers not of Type 1 and nonsmokers (either of Type 1 or of the other types) have negligible rates of cancer deaths. Of the two factors, smoking and personality, personality seems to be the stronger. Of 735 smokers not of Type 1, only 6 were found to have died of lung cancer; this figure is not very different from the 3 nonsmokers not of Type 1 who died out of the total set of 850 nonsmokers. Clearly, smoking appears to represent a danger to health, as far as lung cancer is concerned, mainly for individuals of Type 1.

More research data, as yet unpublished, support these conclusions. In a new study we selected from a large Heidelberg population of over 20,000 male and female probands who had scored exceptionally high on one of the four personality-type scales. The 35 members of each type group were matched for age and sex, and for smoking habits; the mean ages of Types 1 to 4 were 53, 55, 52, and 55 years, respectively, and the proportion of males was 46%.

Table 5 shows the main results. Of Type 1, 21 probands died of cancer, as compared with 3 of Type 2, 1 of Type 3, and 1 of Type 4. Of Type 2, 18 died of CHD, as compared with 5 of Type 1, 1 of Type 3, and 1 of Type 4. Other causes of deaths show Type 4 probands as less affected than Types 1, 2, and 3. These data strongly support our previous studies, in demonstrating a close relationship between personality type and disease. Overall significance by chi-square test is well beyond the $p = .001$ level.

In a recent study, we have altered the method of administering the questionnaire and added two type categories. Type 5 is a rational-antiemotional type, showing characteristics common to Type 1 and Type 2. Type 6 shows psychopathic tendencies and has been found to be prone to drug addiction and AIDS (Grossarth-Maticek & Eysenck, 1990a). The new questionnaire used in the studies to be described is given in full in this reference.

The questionnaire can, of course, be administered in a single session and scored, but the method used in our study and recommended for prediction of proneness to disease is rather more complex. According to this "dynamic" method, the questionnaire is administered on two occasions, separated by 6 months; what is of interest is the change in type score. The change can lead to what may be called stagnation, in which a person having a certain score establishing him as a member of a type liable to certain diseases, retains that score or increases it; this leads to an unfavorable prognosis. Alternatively, the score can show development in a favorable direction, by a reduction in the number of questions answered in the disease-prone direction. Stagnation (S) is identified when the sum of scores on Types 1, 2, and 5, minus the sum of scores for Types 3, 4, and 6, shows an increase in the scores of the first set of types, or a decrease in the second set of types. Favorable development (D) is indicated when there is an increase on Occasion 2 in the scores of the second group of types or a decrease in the scores of the first group of types, leading to a positive difference.

The reason for grouping together Types 3, 4, and 6 as relatively healthy, as opposed to Types 1, 2, and 5 as unhealthy, rests in part on theoretical grounds and past findings but more strongly on psychometric grounds. We took a random sample of 262 women and 486 men from our stressed group, correlated the scores for each sex, separately for Occasions 1 and 2, and then factor-analyzed the matrices. The unrotated values clearly oppose Types 1, 2, and 5 to Types 3, 4, and 6, as hypothesized, in all matrices.

The new questionnaire had been administered to a large sample from which we selected 216 probands, half men and half women, equated for age, on the basis of their scores on the questionnaire. Our aim was to find equal numbers for each of the six types, such that a person given a type was characterized by having a perfect score of 10 for that type, and no score higher than 2 on any other type. The number of probands satisfying this requirement was not too large, and we chose the first probands in our lists who fulfilled the requirement.

Probands were followed up over a 13-year period, mortality and incidence of a variety of disorders being the dependent variables. Diagnoses were obtained from the physicians in charge of persons who were suffering from any kind of illness, with the agreement of the patient. Addiction was diagnosed according to interviews with relatives of the probands. In the case of death, physicians were consulted and death certificates examined.

Table 6 shows the major results of the study. It will be seen that, as predicted, cancer is particularly frequently diagnosed in Type 1; in fact, it is as frequent in persons of Type 1, as in those of all other types taken together. CHD, as expected, is most frequent in Type 2, being about three times as frequent as in all other types. Type 2 also shows significantly higher incidence of ulcer, hypertonia, and diabetes. Type 3 and Type 4, as expected, are relatively healthy, with few medical diag-

Table 5. *Mortality for a Subsample Consisting of Exceptionally High Scorers on a Single Personality Type*

Group	*n*	Cancer	CHD	Other	Living
Type 1	35	21	5	5	4
Type 2	35	3	18	11	3
Type 3	35	1	1	8	25
Type 4	35	1	1	1	32

Table 6. *Relation Between Personality Type and Diagnosis 13 Years Later*

	Personality Type					
Diagnosis	**1**	**2**	**3**	**4**	**5**	**6**
Endogenous Depression	1	1	3	1	27	1
Drug Addiction	9	8	11	0	0	25
Rheumatoid Arthritis	3	1	2	0	16	1
Ulcer Ventriculi et Duodeni	2	19	1	1	2	0
Hypertonia	1	29	4	0	1	1
Diabetes	1	15	1	0	1	0
Infarct/Stroke	3	14	1	0	0	1
Cancer	11	2	1	1	4	3
Total Number of Probands	36	36	36	36	36	36
Mean Age (in Years) at Beginning of Study	47	48	46	47	46	46

noses, although the high scores of Type 3 for addiction might be worthy of a follow-up. Type 5 shows clearly elevated scores for endogenous depression, but also for rheumatoid arthritis. Type 6 only has a high score for addiction, there being about as many addicts of this type as for all other types combined. It is clear that the questionnaire does possess a certain amount of validity, the major diagnostic criteria for the different types agreeing with prediction derived from previous research.

The predictive accuracy of the dynamic procedure, considering death from cancer, CHD, and other causes, was established in a group of 868 probands, assigned to type according to the score on the first occasion. They were then divided into those who showed stagnation (S) or development (D). A follow-up was instituted 13 years later; results are shown in Table 7. Subjects were allocated to a given type if their score for that type exceeded their score for any other type. There were no significant age differences between types.

Totals in the various columns are given at the bottom of the tables; it will be seen that out of 868 probands, 536 are still alive, 95 having died of cancer, 107 of CHD, and 130 of other causes. These results are based on examination of death certificates.

Of those who died of cancer (across all types), 6 were in the development category, 89 in the stagnation category. Of those who died of CHD, 18 were in the development category, 89 in the stagnation category. Of those who died of other causes, 16 were in the development category, 114 in the stagnation category. It is clear that this new dynamic way of prediction is highly successful, primarily, no doubt, because it charts the progress of the way the individual deals with stress. Clearly, if stress is an important cause of death, then a "D" score indicates that the individual is coping well with stress and shows psychological improvement, whereas an "S" score shows the opposite.

Table 7. *Mortality According to Personality Type and Change in Stress Reaction Over Time*

Type	Nature of Change[a]	Cancer	CHD	Other Causes	Still Living
1	S (82)	35	11	15	21
	D (71)	3	4	5	59
2	S (104)	17	49	25	13
	D (84)	1	10	7	66
3	S (56)	8	9	10	29
	D (72)	0	1	0	71
4	S (46)	5	2	3	36
	D (85)	0	1	0	84
5	S (57)	18	9	27	3
	D (91)	1	0	1	89
6	S (57)	6	9	34	8
	D (63)	1	2	3	57
Total	868	95	107	130	536

[a]S = stagnant; no change or worse score on second administration of type questionnaire. D = development; change of score showing improvement on second administration of type questionnaire. Numbers having each kind of change score are shown in parentheses.

Synergistic Interaction Between Risk Factors

The interaction between smoking and personality as risk factors for cancer were made the topic of a special large-scale study. Earlier work, already mentioned, had suggested that such interaction would be synergistic, but the data collection had not been planned with this hypothesis in mind (Grossarth-Maticek, 1980a, 1980b; Grossarth-Maticek, Eysenck, & Vetter, 1988). The present study was designed to give more definitive answers to the question of relationship between several risk factors for cancer.

The view that risk factors for cancer and CHD interact synergistically is of course not new. In a recent article, D. A. Perkins (1989) argued that "interactions among the major coronary heart disease risk factors of smoking, hypertension, and elevated cholesterol may contribute substantially to the prediction of CHD risks over and above the sum of the independent risks due to their factors" (p. 3). He surveyed results showing that "the interaction of smoking and cholesterol and of hypertension and cholesterol may each as much as double the risks of CHD which might be expected if these factors acted only additively" (p. 3). He also commented on "the strong possibility of an interaction between chronic psychosocial stress and elevated cholesterol" (p. 3). Other authors who have argued in favor of synergistic interaction effects are Kannel et al. (1986), Kleinbaum, Kupper, and Morganstern (1982), Rothman (1974), and Saracci (1987); Kooperman (1981), K. A. Perkins (1985, 1987), and Walker (1981) have also added to the methodological analysis of synergistic effects.

An example will illustrate methods and results. Kannel et al. (1986) reported data on more than 300,000 White males selected for inclusion in the Multiple Risk Factor Intervention Trial (see Multiple Risk Factor Intervention Trial Research Group, 1982). For men between 35 and 45 years of age, in the lowest quintile of diastolic blood pressure (76 mm Hg), the 6-year risk of CHD mortality among nonsmokers without elevated cholesterol (i.e., background risks) was 0.6/1,000, whereas the risk due to smoking alone was 1.0/1,000, and the risk for nonsmokers with cholesterol in the highest quintile (above 2.44 mg/dl) was 2.0/1,000. If the effects of smoking and elevated cholesterol were only additive, the risk for those with both factors present would be 2.44/1,000—that is, the background risk of 0.6/1,000 plus the risk attributable to smoking (0.4/1,000) and to cholesterol (1.4/1,000). The actual mortality rate for this subgroup, however, was 4.8/1,000—twice that expected on an additive model, indicating an excess risk of 100%!

This is clearly an important area of research, and the concepts involved may explain why single risk-factors so often fail to give consistent positive results. It is certainly completely inadmissible to use simple univariate analyses for risk-factor studies in epidemiology; multivariate analyses are an absolute must (Wilhelmsen, Wedel, & Tibblin, 1973). Certainly smoking emerges as a risk factor in conjunction with other risk factors, rather than by itself. For this, as well as for other reasons already adumbrated, talk of smoking as "causing" cancer of CHD is scientifically meaningless (Eysenck, 1987c, 1991).

The established facts of synergistic interaction between risk factors also have profound consequences for prophylaxis and prevention generally (D. A. Perkins, 1989). The relative failure of quitting smoking to reduce cancer and CHD mor-

tality may be due to the fact that such quitting would only have pronounced effects in people with more than one risk factor present (Eysenck, 1986). For people with no other risk factors except smoking, quitting smoking would have little effect on mortality, and their inclusion in any study would disguise any impact that quitting smoking might have on mortality (Eysenck, 1991). The issue is discussed in detail in Eysenck (1991) and in Eysenck, Grossarth-Maticek, and Everitt (in press).

In our own study (Grossarth-Maticek, Eysenck, & Vetter, 1988), we started with a randomly selected population of 16,250 men and 3,670 women in Heidelberg, a small German university town. Of these, we selected 512 matched pairs of probands, half with stress, half without. Stress was defined as having a higher score for Types 1 + 2 + 5 than for Types 3 + 4 + 6. None of the probands smoked, suffered from bronchitis, or had a close family member die of cancer. Matching was in terms of sex and age. Results of a 13-year follow-up are shown in Table 8. It will be seen that in the "no stress" group, 25 probands died; in the "stress" group, 136 died. This is a direct measure of stress as a risk factor, uncomplicated by smoking or hereditary disposition.

Table 9 shows data for death from lung cancer, for groups taken from the same sample, again matched between "stress" and "no stress" groups, as already defined. There are five groups differing in number of cigarettes smoked, varying from *none* to *41 to 60 daily*. The probands have no hereditary predisposition and do not suffer from bronchitis. There is a clear synergistic relation between stress and smoking.

Figure 1 makes clear the interactions involved. There are linear dose–response relationships for smoking and lung cancer, but the slopes for stressed and nonstressed probands are very different, being much steeper for the former. This difference in steepness is the index of synergism; smoking is a risk factor for lung cancer at all levels but becomes much stronger when it occurs in stressed probands than when it occurs in nonstressed probands.

Table 10 shows a similar type of analysis for the interaction between personality and genetics as risk factors for lung cancer. Hereditary influences are indexed by the number of close relatives (parents, grandparents) who died of lung cancer. Here too there is a monotonic increase in lung-cancer mortality with increased hereditary disposition, and here too this increase is more rapid for probands with, as compared to those without, stress. The postulation of synergistic interaction is inescapable (Eysenck et al., in press).

As a final study, consider the following. Probands were selected as showing only one, mixtures of two, mixtures of three, or all four of the following risk factors of smoking (C = more than 20 cigarettes per day, for more than 10 years),

Table 8. *Mortality According to No Stress or Stress in Nonsmokers*

		Cause of Death			
	n	Lung Cancer	Other Cancers	CHD	Other Causes
No Stress	512	1 (0.19)	4 (0.7)	7 (1.3)	13 (2.5)
Stress	512	1 (0.19)	38 (6.2)	32 (6.2)	65 (12.6)

Note: Percentages are given in parentheses.

Table 9. *Mortality as a Function of Stress and Smoking Category*

	Died of Lung Cancer					
	Without Stress			With Stress		
Category	*n*		%	*n*		%
Nonsmoker	512	1	0.2	512	1	0.2
Smoker (Daily Cigarettes)						
10 to 20	271	1	0.4	271	2	0.7
21 to 35	271	3	1.1	271	8	2.9
36 to 40	101	5	4.9	101	11	10.9
41 to 60	101	7	6.9	101	17	16.8

heredity (H = at least one first-degree relative suffering from or having died of cancer), chronic bronchitis (B), and stress (S = probands of Types 1 or 2). Table 11 shows the results. In these probands (who were on average around 52 years of age at the beginning of the study), 13 years later, none of those who showed only one risk factor had died of lung cancer. Of those showing two risk factors, only about 1% had died of lung cancer. Combinations of three risk factors showed quite elevated death rates from lung cancer, varying from 7.6% through 9.8% to 20%. A combination of these four risk factors raised the death rate from lung cancer to 31%, demonstrating the strong synergistic effect of multiplying risk factors.

Of particular interest here is the group of four-risk-factor probands in parentheses; they had received prophylactic behavior therapy (BT) and, accordingly, had a death rate from lung cancer only about one third as high as the group of four-risk-factor probands who received no therapy. Thus, even for those most exposed to lung cancer, prophylactic treatment is

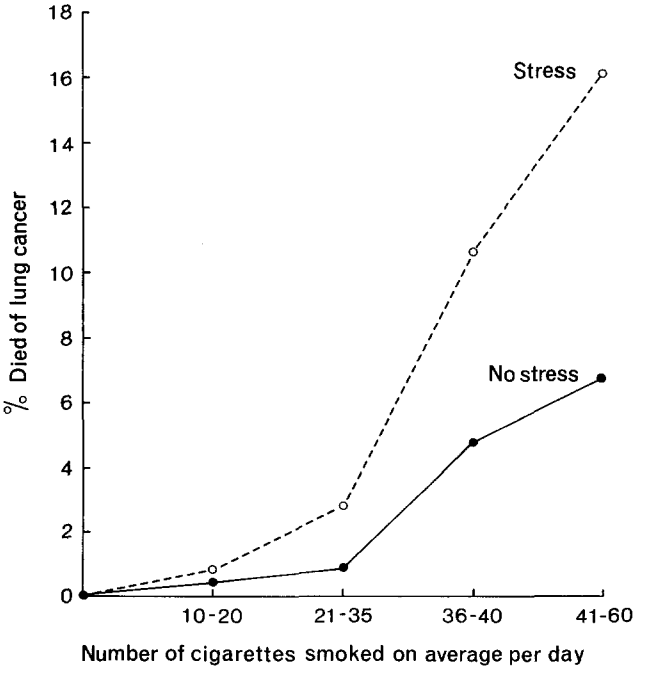

Figure 1. Lung cancer as related to smoking and stress in a synergistic fashion.

Table 10. *Mortality as a Function of Stress and Hereditary Disposition (From Family History)*

Number of Close Relatives Having Died of or Suffering From Lung Cancer	Died of Lung Cancer					
	Without Stress			With Stress		
	n		%	*n*		%
1	191	2	1.1	191	2	0.5
2	98	2	2.2	98	4	4.1
3	33	2	6.7	33	5	15.2
4	33	3	9.1	33	6	19.4
5	25	4	16.0	25	8	40.0
6	16	5	31.3	16	7	58.3

possible and can be efficacious. The next section deals with the prophylactic effects of behavior therapy.

Prevention of Cancer and CHD Through Behavior Therapy

There has been much interest in recent years in the possibility of prolonging life in cancer sufferers, through instilling a "fighting spirit" type of reaction and, quite generally, in the importance of mental attitudes for survival. The evidence certainly suggests that mental attitudes constitute an important prognostic factor for cancer (Eysenck, 1988a, 1988b; Greer, Morris, & Pettingale, 1979; Grossarth-Maticek, 1980a; Nelson, L. C. Friedman, Baer, Lane, & F. E. Smith, 1989; Pettingale, Morris, Greer, & Haylittle, 1985; Pettingale, Philalithis, Tee, & Greer, 1981; Spiegel, J. R. Bloom, Kraemer, & Gottheil, 1989).

If our own views concerning the prognostic importance of cancer-prone behavior and CHD-prone behavior are broadly acceptable, it would seem to follow that appropriate methods of behavior therapy might succeed in changing such behaviors in the direction of Type 4 behavior—by teaching people to express their emotions more freely, in a socially acceptable manner, become more autonomous and able to stand up for their rights and interests, and deal with stress-producing situations more successfully. Such a version of behavior therapy has been described in detail elsewhere (Grossarth-Maticek & Eysenck, 1991).

Three major interventions studies have been published (Eysenck & Grossarth-Maticek, 1991). In the first of these, pairs were formed of healthy probands, matched for sex, age, type, and smoking habits. In each pair, one proband was assigned on a random basis to the control group, the other to the therapy group. Therapy involved about 30 hr of individual treatment during the first few months after initiation of the study; probands were not contacted again until 10 years later, and again 3 years after that, to inquire into mortality. Cause of death was ascertained on the basis of death certificates. The data in Table 12 show the outcome for 100 Type 1 and 92 Type 2 probands. Incidence was ascertained by contacting the physician in charge of the patient.

The effectiveness of the therapy in preventing death from cancer and CHD is obvious. Similarly, incidence is greatly reduced in both therapy groups, as compared with the control groups. All comparisons are highly significant statistically. Particularly impressive are the differences in survival ("living"), a statistic more objective than death-certificate diagnosis (Eysenck, 1986).

A second study attempted to test the effectiveness of group therapy. Groups of 20 to 25 probands were seen a number of times, varying dependent on the wishes of the group. Therapy and control groups were formed as already explained, and the results of the study are shown in Table 13. Clearly, the outcome is very similar for both mortality and incidence to that for individual therapy; 80% surviving in the therapy

Table 11. *Mortality as a Function of the Combination of Different Risk Factors*

Combination of Risks	*n*	Lung Cancer	%	Other Causes of Death	%	Average Age
Only H	50	0	0	5	10	51
Only C	100	0	0	12	12	52
Only S	59	0	0	16	27	52
H + C	50	1	2	4	8	53
H + B	52	0	0	8	15	51
C + B	55	0	0	11	20	52
C + S	100	2	2	21	21	53
H + S	49	0	0	9	18	54
B + S	50	0	0	8	16	53
C + H + B	26	2	8	5	19	51
C + H + S	50	10	20	14	28	51
C + B + S	51	5	10	10	20	51
H + C + B + S	26	8	31	8	31	52
(H + C + B + S + BT)	26	3	12	4	15	52

Note: H = heredity, C = cigarettes, S = stress, B = bronchitis, BT = behavior therapy.

Table 12. *Mortality of Control and Therapy Probands: Individual Behavior Therapy*

Type 1 Group	*n*	Cancer		Other Causes of Death	Living
		Deaths	Incidence		
Control	50	16 (32%)	21 (42%)	15 (30%)	19 (38%)
Therapy	50	0 (0%)	13 (26%)	5 (10%)	45 (90%)
Total	100	16 (16%)	34 (34%)	20 (20%)	64 (64%)

Type 2 Group	*n*	CHD		Other Causes of Death	Living
		Deaths	Incidence		
Control	46	16 (34.8%)	20 (43.5%)	13 (28.3%)	17 (36.9%)
Therapy	46	3 (6.5%)	11 (23.9%)	6 (13%)	37 (80.4%)
Total	92	19 (20.6%)	31 (33.7%)	19 (20.7%)	54 (58.7%)

Note: From "Creative Novation Behaviour Therapy as a Prophylactic Treatment for Cancer and Coronary Heart Disease: II. Effects of Treatment" by H. J. Eysenck and R. Grossarth-Maticek, 1991, *Behaviour Research and Therapy, 29*, p. 19. Copyright 1991 by H. J. Eysenck. Adapted by permission.

Table 13. *Mortality of Control and Therapy Probands: Group Behavior Therapy*

	Therapy[a]		Control[b]	
	Mortality	Incidence	Mortality	Incidence
n	239	235	234	231
Cancer	18 (7.5%)	75 (31.9%)	111 (47.4%)	129 (55.8%)
CHD	10 (4.2%)	29 (12.3%)	36 (15.4%)	45 (19.5%)
Other Causes of Death	20 (8.4%)	—	33 (14.1%)	—
Living	191 (79.9%)		56 (23.9%)	

Note: From "Creative Novation Behaviour Therapy as Prophylactic Treatment for Cancer and Coronary Heart Disease: II. Effects of Treatment" by H. J. Eysenck and R. Grossarth-Maticek, 1991, *Behaviour Research and Therapy, 29,* p. 20. Copyright 1991 by H. J. Eysenck. Adapted by permission.
[a]Of the original $N = 245$, 6 were not contacted. [b]Of the original $N = 245$, 11 were not contacted.

group, as compared with 24% in the control group, shows the effectiveness of the intervention. It also gives addition proof of the importance of personality and stress as risk factors for cancer and CHD; intervention studies suggest a causal relationship rather than a purely statistical one (Eysenck, 1991).

Our third study used a mixture of bibliotherapy and short-term behavior therapy, together with the use of a placebo therapy group. Two sets of 600 probands each were matched as before, and allocated at random to a therapy or control group. Therapy consisted of giving each member of the therapy group a written pamphlet outlining the principles of behavior therapy as applied to better, more autonomous living, and avoidance of stress. The contents of the pamphlet were explained in detail during a 1-hr interview, and its specific application to each person's circumstances was discussed in three 1-hr interviews later on. Of the 600 members of the control group, 100 were randomly chosen to constitute a placebo group; they were given a pamphlet setting out a set of psychoanalytic principles that were judged to be irrelevant to the prevention of cancer and CHD, yet seemed valuable on the face of it. The administration of the pamphlet was also accompanied by personal visits and explanation. Results of this study are shown in Table 14.

The results of this study are surprisingly positive and not very different from those of the individual and group therapy studies. Again we see that treated probands survive much better than controls and have lower incidence. Placebo treatment is no better than no treatment. Clearly, bibliotherapy of this kind accompanied by detailed explanation, is a very cost-effective form of treatment.

A less formal but nevertheless illuminating comparison is afforded by our study of the effects of psychoanalysis on various samples of probands. As is well known, psychoanalysis constitutes a severe stress on neurotic patients, and often has very negative effects (Mays & Franks, 1985). In addition, psychoanalysis usually makes patients more dependent on the analyst, rather than increasing their autonomy, which is the aim of behavior therapy. On the basis of these considerations, we predicted that patients under analysis would be more likely than probands matched for personality type and other variables, but not undergoing analysis, to suffer and die of cancer and CHD.

Results have supported this hypothesis (Grossarth-Maticek & Eysenck, 1990b). Table 15 shows what percentage of probands survived a 7-year follow-up in the control group, in a group of patients terminating psychoanalysis in less than 2 years, and in a group of probands continuing psychoanalysis for more than 2 years. Clearly, there is a dose–response relation; the more psychoanalysis, the greater the mortality! No such deleterious effects were noted for patients undergoing various forms of short-term psychotherapy.

The term *psychoanalysis* at present does not denote a consistent body of theory and treatment; many different aims are expressed, and many different types of treatment offered. In view of the importance of increased autonomy in our system of treatment by behavior therapy (Grossarth-Maticek & Eysenck, 1991), we questioned the patients undergoing psychoanalysis concerning the following alternatives: Did the treatment increase or decrease their autonomy? Results showed that of those who felt that the treatment had decreased their autonomy, 86% of Type 1, 75% of Type 2, and 87% of Type 3

Table 14. *Mortality of Control and Therapy Probands: Bibliotherapy*

	Causes of Death									
	Cancer		CHD		Other					
Group	D	I	D	I	D	I	Total	Living	Not Investigated	
Control ($N = 500$)	106 (21.5%)	162 (33.4%)	145 (29.4%)	203 (41.8%)	164 (33.3%)	—	415 (84.2%)	78 (15.8%)	7 (1.4%)	15 (3%)
Placebo Control With Use of Psychoanalytic Text ($N = 100$)	22 (22%)	37 (37.7%)	31 (31%)	40 (40.8%)	28 (28%)	—	81 (81%)	19 (19%)	0 (0%)	2 (2%)
Therapy Group With Behavior Therapy Text ($N = 600$)	27 (4.5%)	99 (16.9%)	47 (7.9%)	132 (22.5%)	115 (19.2%)	—	189 (31.6%)	409 (68.4%)	2 (0.3%)	14 (2.3%)

Notes: D = died, I = incidence. From "Creative Novation Behavior Therapy as a Prophylactic Treatment for Cancer and Coronary Heart Disease: II. Effects of Treatment" by H. J. Eysenck and R. Grossarth-Maticek, 1991, *Behaviour Research and Therapy, 29,* p. 20. Copyright 1991 by H. J. Eysenck. Adapted by permission.

Table 15. *Percentage of Probands Surviving a 7-Year Follow-Up as a Function of Time Spent in Psychoanalysis*

	Survivors		
	Type 1	Type 2	Type 3
Control Group	96	92	95
Psychoanalysis Terminated in Less Than 2 Years	84	84	84
Psychoanalysis Continued for More Than 2 Years	74	70	78

Note: From "Prophylactic Effects of Psychoanalysis on Cancer-Prone and Coronary Heart Disease-Prone Probands, as Compared With Control Groups and Behaviour Therapy Groups" by R. Grossarth-Maticek and H. J. Eysenck, 1990, *Journal of Behaviour Therapy and Experimental Psychiatry, 21,* p. 94. Copyright 1990 by H. J. Eysenck. Adapted by permission.

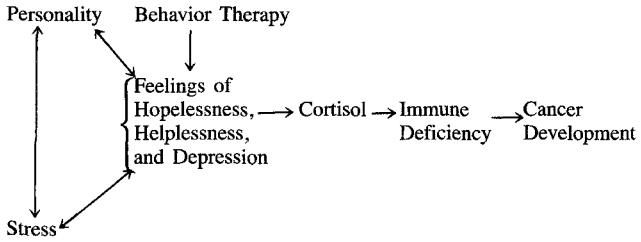

Figure 2. Diagrammatic representation of theory linking personality and stress with cancer.

were still alive. Of those who felt that treatment had increased their autonomy, the figures are 97%, 97%, and 96%. These data support our theory that autonomy (i.e., the ability to be independent in one's thoughts, feelings, and actions, even under stress) is important for survival and is a valuable countermeasure as far as cancer and CHD are concerned. Clearly, not all treatments called *psychoanalysis* are equivalent in their conception or their effects; they are dangerous only insofar as they impair autonomy.

Interpretation of these data suffers of course from the possible objection that patients undergoing psychoanalysis may have suffered greater mortality because of whatever caused them to seek help in the first place. That seems unlikely; the usual reasons for undergoing psychoanalysis have never been found to lead to cancer and CHD, and of course patients and controls were matched for personality type, the most predictive risk factor of all. Apparently, we should not consider psychoanalysis as a placebo treatment for prophylaxis cancer and CHD, but as a very active treatment constituting an additional risk factor to those usually studied.

Beginnings of a Theory Linking Personality and Disease

We have shown that a considerable body of evidence supports the view that:

1. There exists a cancer-prone personality.
2. There exists a CHD-prone personality.
3. Personality type as a risk factor for disease interacts synergistically with other risk factors such as smoking and heredity.
4. Behavior therapy can *reduce* significantly the likelihood of cancer or CHD mortality.
5. Psychoanalysis can *increase* significantly the likelihood of cancer and CHD mortality.

Acceptance of these conclusions would obviously be greatly enhanced if we could provide at least the beginnings of a theory linking the two sides of the equation—that is, psychosocial factors on the one side, physical disease on the other.

A brief outline may here be given of the way the connections between personality, stress, and disease may be mediated by hormonal and physiological factors. A more detailed outline is given elsewhere (Eysenck, 1986, 1991). Figure 2 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 given here as the representative (others are the endogenous opiates, adrenocorticotrophic hormone [ACTH], etc.). These in turn produce immune deficiency, which allows budding cancers to develop. There is a good deal of evidence to support such a model.

B. S. Linn, M. W. Linn, and Jensen (1981) showed that stress and anxiety are associated with depressed immunological response. Levy, Herberman, Lippman, and d'Angelo (1987) and Levy, Herberman, Maluish, Schlien, and Lippman (1985) 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 A^1. Bandura, Cioffi, Taylor, and Brouillard (1988) found that perceived self-inefficacy in exercising control over cognitive stressors activated endogenous opioid systems. 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 et al. (1984) found that high scorers on stressful life events and loneliness had significantly lower levels of NK cell activity. Loneliness suggests a lack of social support due to, to some extent no doubt, inefficient coping mechanisms. Herberman (1988), Irwin, Vale, and Britton (1987), Nemeroff et al. (1984), and Rou, Rose, Sunderland, Moritisa, and Murphy (1988) found impaired immune reaction in depressed groups, and Arnetz et al. (1987), Glaser, Kiecolt-Glaser, Speicher, and Halliday (1985), B. S. Linn, M. W. Linn, and Klimas (1988), and Shavit, Lewis, Terman, Gale, and Leibeskind (1989) found impaired immune reactions to stress. Jemmott and Magloine (1988) found that stress lowered salivary concentrations of S-IgA, whereas social support increased them. Grossarth-Maticek and Eysenck (1989) found that behavior therapy significantly increased the percentage lymphocyte count 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 enhanced immunocompetence from relaxation and social contact.

Irwin, Daniels, T. L. Bloom, H. Smith, and Weiner (1987) showed that life events can cause depression, and 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 (1990) showed that cortisol level correlated with anxiety, depression, and high heart rate.

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; M. W. Linn, B. S. Linn, & Jensen, 1984; McClelland, Floor, Davidson, & Saron, 1980; McClelland, Ross, & Patel, 1985; Stone, Cox, Valdimarsdottir, Jemdorf, & Neale, 1987). Animal studies, too, have contributed to the formulation of the model (e.g., Borysenko & Borysenko, 1982; Glaser, Thorn, Tarr, Kiecolt-Glaser, & D'Ambrosia, 1985; Laudenslager, Ryan, Dougan, Hyson, & Maier, 1983; for a review, see Justice, 1985).

The studies cited are only among 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 Dilman and Ostroumova (1984) and Eysenck (1986) and briefly outlined in the preceding discussion. There is evidence (a) that personality and stress produce immunodestructive substances in the bloodstream, (b) that these substances do have such an immunodestructive function, and (c) that behavior 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) showed, 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 our theory is subclinical and might be defined as "hopelessness depression" (Alloy, Abramson, Metalsky, & Hartlage, 1988). This concept is largely based on the work of Abramson, Seligman, and Teasdale (1978) and Seligman (1975) 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).

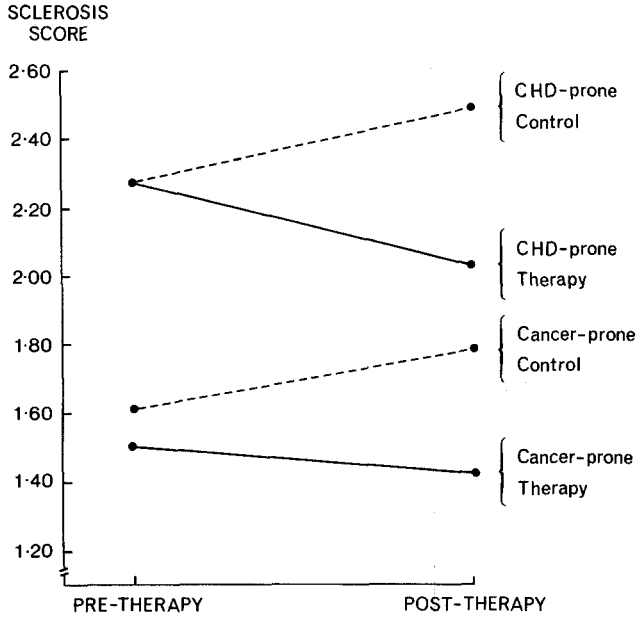

Figure 3. Degree of sclerosis as a function of personality type and treatment. From "Changes in Degree of Sclerosis as a Function of Prophylactic Treatment in Cancer-Prone and CHD-Prone Probands" by R. Grossarth-Maticek, H. J. Eysenck, G. Gallasch, H. Vetter, and R. Frentzel-Beyme, 1991, *Behaviour Research and Therapy, 29*, p. 347. Copyright 1991 by H. J. Eysenck. Reprinted by permission.

As far as CHD is concerned, there is less material to review, but sclerosis is an obvious intermediary. Grossarth-Maticek and Eysenck (1991) 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 3 shows the results. Type 2 probands had significantly higher levels of sclerosis than Type 1 probands, and the therapy group 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.

The theories here adumbrated are of course grossly oversimplified; cortisol is only one of several substances (e.g., ACTH, endogenous opiates) that interact in complex ways to affect the immune system. What the theory does is direct attention to areas worthy of study; in particular, any replication of the investigations here reviewed should include regular immune assays to monitor the effects of stress, and of therapy; similarly, regular assays of sclerosis should be included.

Discussion and Conclusions

The results presented here briefly constitute the first points of a large and long-continued research effort that began more than 25 years ago. Prospective studies are of course the most rewarding in the epidemiological field, but they also take a long time to come to fruition. Nevertheless, even the partial results at hand at present indicate the truth of the words

written by an Indian sage more than 4,000 years ago: "There are two classes of disease—bodily and mental. Each arises from the other. Neither is perceived to exist without the other. Mental disorders arise from physical causes, and likewise physical disorders arise from mental causes" (*Mahabharata:* Santi Parva, XVI 8–9). Modern biological psychiatry attests to the truth of the first of these statements; the work here presented attests to the truth of the second.

It is becoming more and more clear that we cannot continue to base our hypotheses on Cartesian dualism, separating rigidly the body and the mind. Physicists had to learn to deal with a space–time continuum, and similarly we will have to learn to deal with a mind–body continuum. Philosophical arguments notwithstanding, it would be unreasonable to dismiss the obvious instances of interaction. Mental states, cognitions of one kind or another, emotions and moods are produced by physical causes, and in turn influence physical events in the body. Behavior therapy uses behavior to influence mental states and cognitions, but it also uses cognitions to influence behavior; modern learning theory has shown beyond a doubt that cognitions are an ever-present accompaniment of conditioning and learning processes, even in animals (Eysenck & Martin, 1987). There is no meaningful distinction between behavior therapy and cognitive therapy; both are closely interacting with each other.

It should be clear that no finality can be claimed for the studies reported here. It is not clear, for instance, to what degree the success of the intervention is due to the *method* used, the *personality* of the therapist, or the *circumstances* under which treatment took place. Much remains to be done, but at least the parameters of the problem are becoming more apparent. A proper solution would have far-reaching consequences (Eysenck & Grossarth-Maticek, 1991).

Note

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