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Personality as a risk factor in coronary heart disease

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Abstract

The pioneering work of the authors of the Type A personality concept has now been shown to be seriously flawed, with only the traits of anger, aggression, and hostility remaining as risk factors for coronary heart disease (CHD). There is now evidence for a much stronger relationship between CHD and personality involving a rather different set of concepts and theories. The evidence for such a relationship is summarized, and a causal link suggested. It is also shown that the CHD-prone type of behaviour can be changed by behaviour therapy, decreasing considerably the risk of dying from CHD.

INTRODUCTION

Coronary heart disease (CHD) is the major killer in modern society, and many risk factors have been suggested. Among these, smoking has assumed a leading role, although the evidence on this point is equivocal (Eysenck, 1965, 1980, 1986, 1991). Consider the results of the famous Framingham study, involving a random sample of the adult residents of Framingham, Massachusetts (Dawber, 1980). The final cohort consisted of 2282 men and 2845 women who were aged 29 through 62 years and free from CHD at the initial examination. After a thorough medical examination, members were checked for cardiovascular disease every 2 years. The follow-up period now extends over 30 years, and the major results have recently been summarized by Seltzer (1989.) At the latest examination, the risk ratios for men and women, ages 35–84, were 1.0 for males and 1.0 for females; in other words, smokers and non-smokers showed no differences at all! At various intermediate stages risk ratios for women were at times as low as 0.8, suggesting that smokers had *less* CHD than non-smokers; for men, the risk ratios sometimes reached 1.4, but multivariate analysis taking into account personality variables (Type A) reduced the statistical significance of this finding to $p > 0.05$. In this monumental study there is thus very little evidence of significance for smoking as a risk factor for CHD in originally healthy people (Eysenck, 1991).

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A more promising risk factor than smoking is personality. Clinical observation of CHD patients has linked this disease for a long time with certain personality traits of impatience, hyperalertness, aggressiveness, and proneness to anger (Kemple, 1945; Menninger and Menninger, 1936; Osler, 1910). These observations were put together into the concept of Type A behaviour by two cardiologists (Friedman and Rosenman, 1959), who reported that the majority of CHD patients showed a set of behaviour patterns and emotions similar to those shown in previous clinical observations (Price, 1982). In addition to the Type A personality pattern, stress and tension have been advanced as psychosocial causes of coronary heart disease (Kaplan, 1983; McGuigan, Sime and Wallace, 1984). Much research in the past 30 years has attempted to build on these foundations.

The Western Collaborative Group Study (Rosenman, Brand, Jenkins *et al.*, 1979) was the first major prospective study designed to examine the coronary risk of Type A probands. In this study, 3154 employed men free of CHD were followed for 8½ years. The final follow-up report showed that by the end of the study probands assessed by the Structured Interview at entry as Type A had a risk ratio of 2 to 1 for the development of coronary heart disease compared with Type Bs. In the Framingham Heart Study, 1674 male and female subjects were followed up. It showed that Type A behaviour was an independent predictor of CHD for both men and women of 45–64 years of age in some subgroups (Haynes, Feinleib and Kannel, 1980). A third large-scale study, the Belgian–French Pooling Project (1984) used the Bortner (1969) rating scale to assess Type A behaviour, and found double the incidence of CHD in probands in the highest quarter of the scale as compared with probands in the lowest quarter. The French and Belgian populations had to be combined in this study to reach statistical significance.

Not all studies, however, have been positive in their findings of predictive accuracy for the Type A concept. The largest study done so far has been the Multiple Risk Factor Intervention Trial. It used a population of 12 700 men who were CHD-free at entry and who were followed for an average of 7 years, and showed that Type A behaviour was unrelated to the 7-year incidence of CHD, a failure observed both for the Structured Interview and for the Jenkins' Questionnaire (Shekelle, Hulley, Neaton *et al.*, 1985). (Those findings were based on a highly selected 'high-risk' population that did not, in fact, turn out to be at high risk.) Similar negative results were found in the Aspirin Myocardial Infarction Study, where it was found that in survivors of a first myocardial infarction, Type A men were at no greater risk of a second myocardial infarction, or coronary death, than their Type B counterparts (Ruberman, Weinblatt, Goldberg and Chandbury, 1984), although we may doubt the value of the particular questionnaire used. Both positive and negative results are neatly balanced to show that as a predictor Type A behaviour is of doubtful value, and may be quite useless. Weighting the results by numbers of participants, it is doubtful whether the total can be regarded as significant in a socially meaningful rather than a statistical sense.

Some of the problems with Type A behaviour as a concept are probably due to the psychometric properties of the scales which have been used (Booth-Kewley and Friedman, 1987). As Eysenck and Fulker (1983) have shown, *inter alia*, the concept is not unidimensional but breaks down into a number of factors, and indeed it appears largely to consist of a combination of neuroticism and extraversion. It now seems likely that the concept of Type A behaviour does not hold together,

and that only some of the traits included are in fact relevant to CHD, notably anger, hostility, and aggression (Booth-Kewley and Friedman, 1987; Friedman and Booth-Kewley, 1987; Eysenck, 1988a, 1990). Better theories and personality tests relevant to the concept of CHD-proneness are now available and will be reviewed in the following sections.

PERSONALITY AND CHD-PRONENESS

The theory here only briefly outlined has been described, and confirmatory evidence presented, in a series of papers (Eysenck, 1985, 1987, 1988b; Eysenck and Grossarth-Maticek, 1989; Grossarth-Maticek, 1980a, b; Grossarth-Maticek, Bastiaans and Kanazir, 1985; Grossarth-Maticek and Eysenck, 1990; Grossarth-Maticek, Eysenck and Vetter, 1988; Grossarth-Maticek, Kanazir, Vetter and Jankovic, 1983). Essentially, the theory states that disease, and type of disease, depends on a person's reaction to interpersonal stress. Type 1 (the *cancer-prone* type) is characterized by the inhibition of realizing closeness with loved persons; such a person is unable to create conditions which would enable him/her to fulfil his/her desires in this respect. Type 2 (the *CHD-prone* type) reacts with anger and general excitement to frustration in interpersonal relations, unable to possess or relinquish the loved person. Type 3 (*hysterical* personality) alternates between these two types of reaction, and is thus protected to some degree from both cancer and CHD. Type 4, finally, is a *healthy, autonomous* type, freely able to react appropriately in interpersonal relations.

Personality and interpersonal stress, and in particular the *reaction* of each proband to such stress, were ascertained by trained interviewers on the basis of questionnaires which allocated each proband to one of the four type categories. The questionnaire used has been published (Grossarth-Maticek *et al.*, 1988) and should be consulted in order to make clearer the precise nature of the personality types than is possible through the reading of the very brief description given above. The method of measurement is of course ipsative with all the advantages and disadvantages attending such measurement.

Three major prospective studies were undertaken to test the hypothesis that answers to the inventory questions would be predictive of disease generally, and would also predict more specifically deaths from cancer or from CHD. In each study, a random sample of physically healthy probands was chosen, personality established, and a variety of relevant information obtained, concerning smoking, drinking, cholesterol level, blood pressure, blood sugar, etc. Probands were then left alone for 10 years, at which age they were contacted again by interviewers if alive, and their death certificates consulted if not. Cause of death was then correlated with personality type.

The first group to be studied was made up of the oldest inhabitant in every second household in a small Yugoslav town, Crevenka. The second group was made up of a random sample of citizens in Heidelberg, average age 50 years, and thus 10 years younger than the Yugoslav sample, with an average age of 60. The third sample was made up of Heidelberg citizens who had been nominated by members of the second, 'normal' sample as being highly stressed. This 'stress' group was similar to the 'normal' group in age and other characteristics, and may thus serve as a good control to study the effect of stress on mortality; our expectation was that

the 'stressed' group would show higher mortality than the 'normal' group. We also expected the Yugoslav group to show higher mortality in view of their higher mean age.

Figures 1, 2, and 3 show that all our expectations were in fact borne out (Eysenck, 1988a; Grossarth-Maticek *et al.*, 1988). Type 1 is clearly predisposed to cancer, Type 2 to CHD, while Types 3 and 4 are relatively healthy. There is greater mortality in the Yugoslav group, and in the stressed Heidelberg group, than in the normal Heidelberg group.

The numbers involved, and the size of the difference, makes all the results statistically significant at or beyond the $p < 0.001$ level.

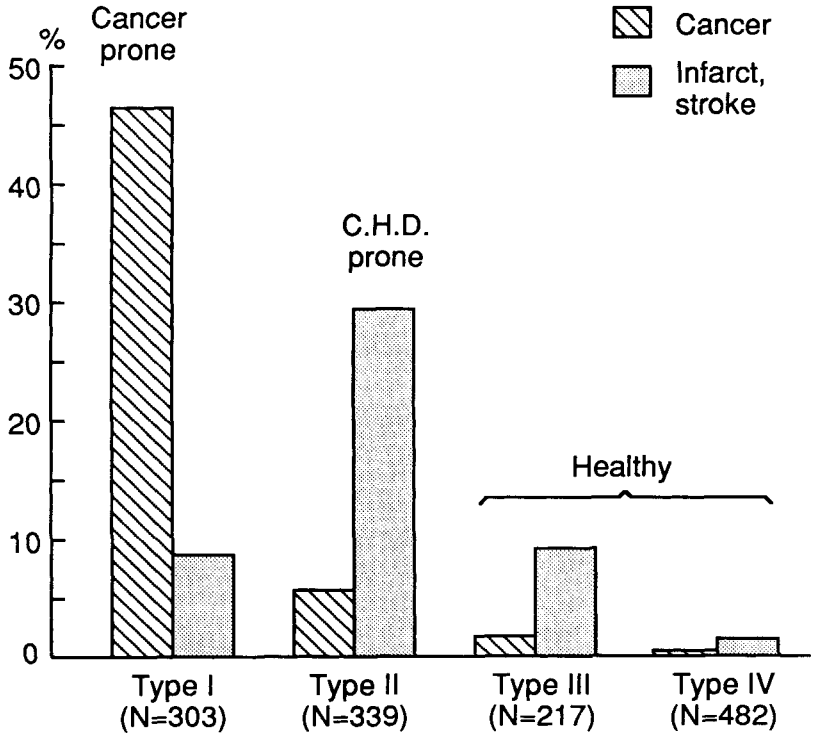

Figure 1. Deaths from cancer and coronary heart disease according to personality type. Yugoslav study (Eysenck, 1988b; Grossarth-Maticek *et al.*, 1988)

To substantiate our major findings, Table 1 sets out the combined data for the three studies, listing the actual numbers dying from cancer, CHD, or other causes, in each of the four type categories. Of the total sample of 3235 probands, 394 died of cancer, 299 of CHD, and 495 of other causes. Nearly all the cancer deaths came from Type 1 probands; the great majority of CHD deaths came from Type 2 probands. The category 'other causes' is difficult to interpret; in view of the well-known lack of accuracy of death certificate diagnoses (Eysenck, 1986), quite a number of these would undoubtedly be due to cancer or CHD not properly diagnosed.

Details concerning these studies have been published in the references already

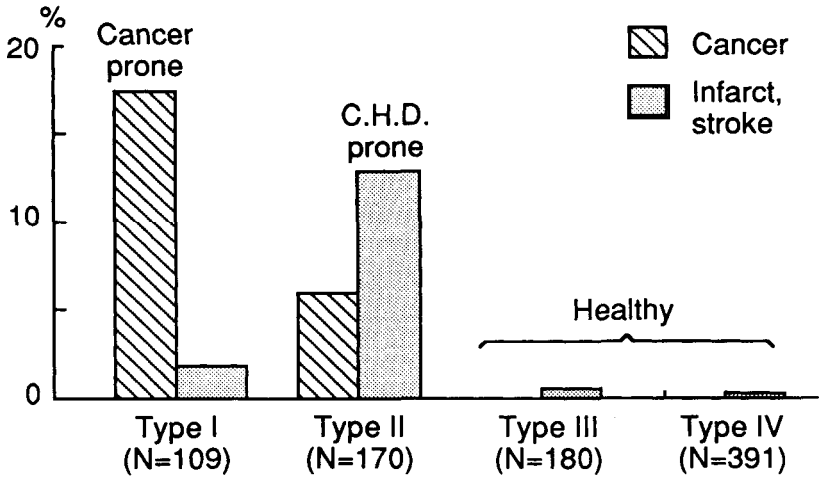

Figure 2. Deaths from cancer and coronary heart disease according to personality type. Heidelberg 'normal' study (Eysenck, 1988b; Grossarth-Maticek *et al.*, 1988)

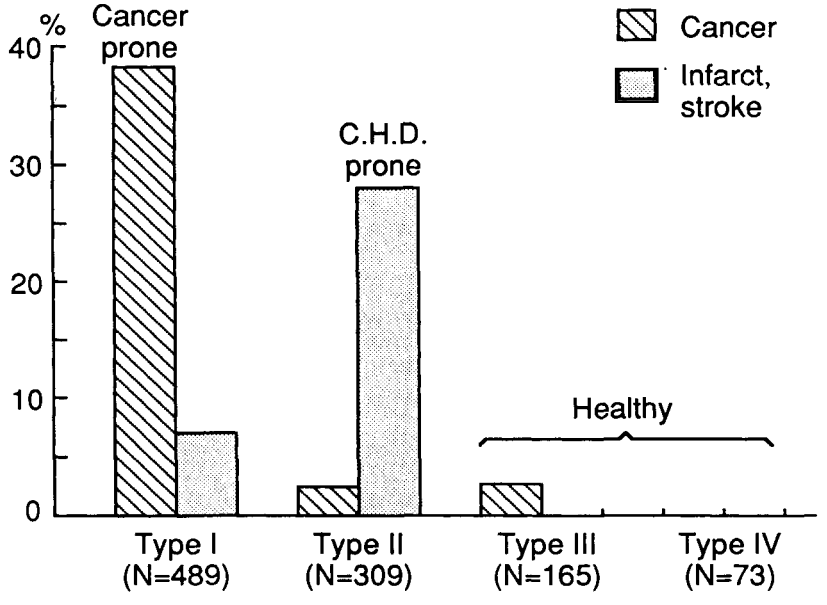

Figure 3. Deaths from cancer and coronary heart disease according to personality type. Heidelberg 'stressed' study (Eysenck, 1988b; Grossarth-Maticek *et al.*, 1988)

given. A more recent study was designed to replicate the results in question, and used a new questionnaire as well as a new method of administration (Grossarth-

Table 1. Mortality according to personality type

		Died of:			
	Still living	Cancer	CHD	Other causes	Total *N*
Type 1	338	347	61	155	901
Type 2	353	36	208	221	818
Type 3	461	8	21	80	570
Type 4	895	3	9	39	946
Total	2047	394	299	495	3235

Maticek and Eysenck, 1990). The actual questionnaire used is printed in full, together with psychometric information, in the above reference; it is probably better suited to routine administration and scoring than the rather more complex inventories used previously. In this questionnaire, two more types have been added to the four types already described: Type 5 is a *rational–anti-emotional* type, also liable to cancer; Type 6 is a *psychopathic* type predisposed to drug-taking. No more will be said about these additional types as they are not relevant to our discussion of CHD-proneness.

The method of administration is based on the hypothesis that stress causes disease, and that successful coping with stress lessens the probability of disease. Hence we administer the personality/stress inventory twice, with 6 months elapsing between the two occasions. If persons improve their score on the disease-prone type categories (i.e. lower it), or increase their score on the healthy type categories, this is considered a favourable development (D). D is defined in terms of a *reduction* of scores on Types 1, 2, and 5; or an *increase* of scores 3, 4, and 6; or both. If a person's scores remain at the same level, or deteriorate, i.e. if scores on Types 1, 2, and 5 *increase*, or scores on Types 3, 4, and 6 *decrease*, or both, this person is stagnating (S), and is more likely to succumb to disease.

These assumptions were tested on a stressed Heidelberg sample of 868 probands. Table 2 shows the results of the follow-up. Clearly, most of the cancer deaths occur as expected among probands of Type 1 (and Type 5); most of the CHD deaths occur as expected among probands of Type 2. But clearly in both cases, the deaths are predominantly among probands categorized S or *stagnant*, rather than among those categorized D or *developing* in a favourable direction. Looking only at Types 1, 2, and 5, i.e. the disease-prone types, the number of deaths of cancer or CHD for S probands is 139; for D probands it is 19. Adding deaths from other causes, the figures are 206 for S and 32 for D probands. These differences are highly significant statistically, and suggest the possible value of this method of administration.

SMOKING AND PERSONALITY AS RISK FACTORS

The possibility that smoking habits might be different for different personality types has of course been considered, and the necessary calculations have been performed to show that eliminating the effects of smoking does little to change the significance

Table 2. Mortality according to personality type and change in type score over 6 months

Type		Died of: 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

Note: S = stagnant (no change or worse score on second administration of questionnaire); D = development (change of score showing improvement on second administration of questionnaire).

of personality as a risk factor for CHD or cancer (Eysenck, 1988a). Comparing the relative predictive accuracy for death from cancer and death from CHD of smoking, blood pressure, and blood cholesterol, all taken together, with personality type, it was found that (1) personality type was six times as important as a risk factor as were the other variables, and (2) the *interaction* of all these variables was *synergistic*. In other words, the effectiveness of one risk factor was increased multiplicatively by the presence of another risk factor. This general principle of non-additivity has already been securely established for other risk factors [for a review, see Eysenck (1990]; apparently it applies equally to the interaction between personality and more usual medical risk factors.

As an example, consider the interaction of systolic blood pressure and a measure of rationality–anti-emotionality, and of smoking and the same measure of rationality–anti-emotionality (Eysenck, 1988a; Grossarth-Maticek, 1980b). The major results of the statistical analysis of the data (which were derived from the Yugoslav study) are given in Table 3. Note that only one of the traits related to CHD, namely Rationality–Anti-emotionality, was used to define the personality variable, yet its presence multiplied the effect of the physical variable (systolic blood pressure; smoking) by a factor indicated in the last column. In all cases, the interaction term was significant. The relative insignificance of smoking as a risk factor *independent* of personality is apparent; in this, our data agree completely with the Framingham data already quoted.

In assessing research in this very complex field, it is always important to bear in mind the interactive nature of all the risk factors investigated, and in particular the synergistic nature of their interaction. Statements like 'Smoking causes CHD'

Table 3. Interaction of physical and personality variables in causation of coronary heart disease (Eysenck, 1988a)

Dependent variable	Physical risk factor	Personality risk factor	Significant interaction factor	Interaction factor
Apoplexy	Systolic blood pressure	Rationality/ anti-emotionality	<0.01	3.9
Infarct	Systolic blood pressure	Rationality	<0.05	6.0
Infarct	Smoking	Rationality/ anti-emotionality	<0.01	34.0

are clearly devoid of any scientific meaning unless this qualification is borne in mind.

PERSONALITY AND SCLEROSIS AS A MEDIATING FACTOR IN CHD

So far, we have dealt only with the taxonomic or descriptive problem of the *correlation* between personality and CHD; clearly, it would be desirable to have some evidence of a *causal* link, and a general theory of how such a link might work. Such evidence, which is of course indicative rather than decisive, can only come from intervention studies, i.e. studies in which an attempt is made to *alter* one of the risk factors in question, i.e. here the type of behaviour which is indicative of CHD-proneness. This has been done in several studies, only one of which will be discussed here for reasons which will become obvious. For a more detailed account of our intervention studies, see Grossarth-Maticek and Eysenck (1991), and Eysenck and Grossarth-Maticek (1991).

In the study to be discussed, 100 probands of Type 1, and 92 probands of Type 2, were matched individually on age, sex, type, and smoking habits. In each of the pairs so created, one was chosen on a random basis for the control, the other for the therapy group. Therapy took roughly 30 h of individual treatment, and was administered at the beginning of the study. Thirteen years later, mortality and cause of death were ascertained, as well as incidence of cancer and CHD, i.e. cases where the disease had been diagnosed, but had not yet resulted in death. Table 4 gives the results of the study. Clearly, therapy has had a powerful prophylactic effect, suggesting that the psychological mechanisms involved provided a *causal* basis for the disease.

As regards the link between personality and CHD, we suspected that sclerosis might be implicated. Consequently, in the study just described, we also added an ophthalmological examination of sclerosis of the fundus of the eye, rated on a 3-point scale, from 1 (Non-existent) through 2 (Mild) to 3 (Severe); the methodology is described elsewhere (Grossarth-Maticek, Eysenck, Gallasch, Vetter and Frentzel-Beyme, in press). The examination was performed twice, at the beginning of the study, and after therapy, or at an equivalent interval for the members of the control group. Figure 4 shows the results.

The main findings are (a) that Type 2 probands have very significantly higher

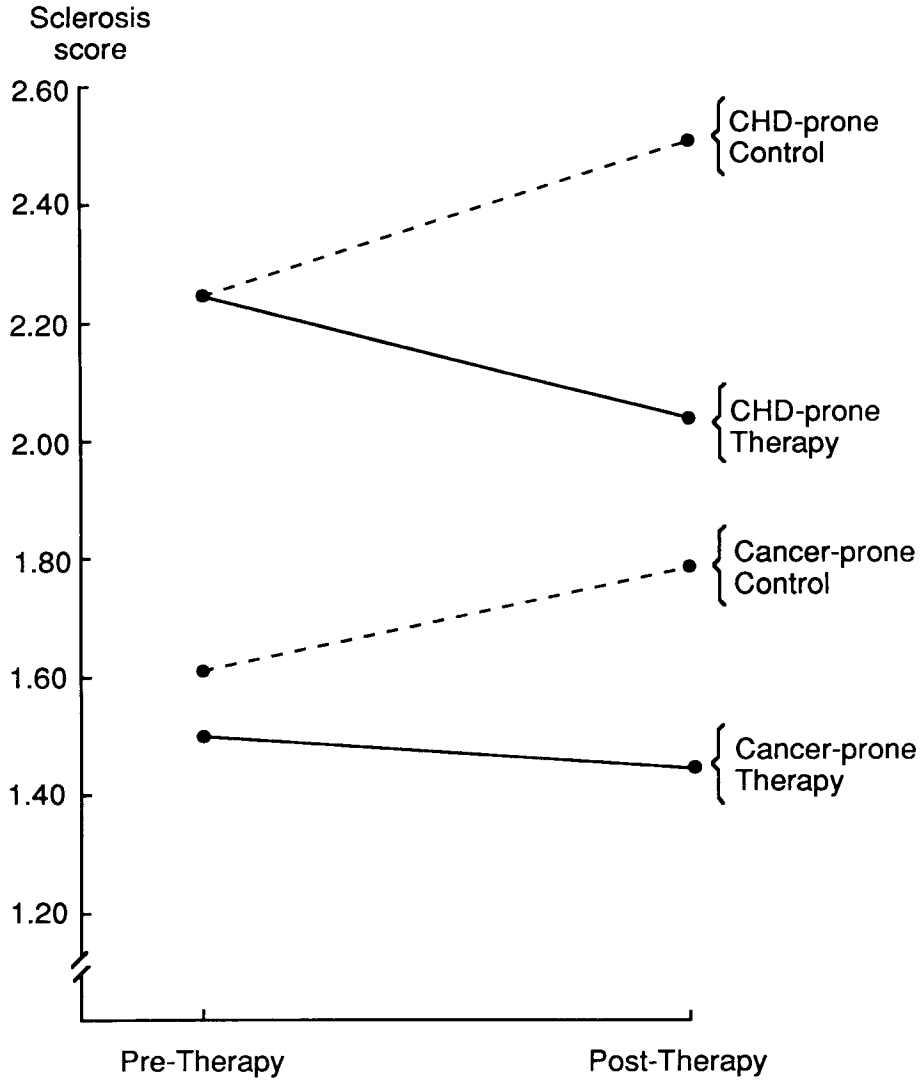

Figure 4. Level and change in sclerosis as a function of personality type and therapy (Grossarth-Maticek *et al.*, 1991)

degrees of sclerosis than Type 1 probands. Degree of sclerosis (b) worsens for the controls, but lessens for therapy probands, again at a high level of significance. Finally, (c) the effects of therapy are stronger for Type 2 than for Type 1 probands, presumably because of threshold effects—Type 1 probands already have very low values for sclerosis.

These data indicate that personality is directly connected with physical factors, i.e. sclerosis, and that these physical factors can be modified by psychological intervention. No doubt the process is very complex, and may involve many bodily-disease mechanisms; no more is claimed than a beginning to a lengthy but exciting and worthwhile process of discovery.

Table 4. Mortality and incidence of cancer and coronary heart disease as a function of personality and absence of therapy (Eysenck, 1990a)

I	*N*	Cancer		Other causes of death	Living
		Deaths	Incidence		
Control	50	16	21	15	19
		32%	42%	30%	38%
Therapy	50	0	13	5	45
		0	26%	10%	90%
Total	100	16	34	20	64
		16%	34%	20%	64%

II	*N*	Cancer		Other causes of death	Living
		Deaths	Incidence		
Control	46	16	20	13	17
		34.8%	43.5%	28.3%	36.9%
Therapy	46	3	11	6	37
		6.5%	23.9%	13%	80.4%
Total	92	19	31	19	54
		20.6%	33.7%	20.7%	58.7%

SUMMARY AND DISCUSSION

In summary, it does seem that theories linking personality and disease-proneness have a solid basis, and that this relation may not be merely *statistical*, but *causal* in nature. Much more research will of course be required to replicate and extend these findings, and in particular the theoretical background for these findings is at present extremely primitive, and requires broadening and augmentation. Nevertheless, the data have a high degree of statistical significance, and suggest great social possibilities for the prevention of coronary heart disease. This alone should encourage psychologists to enter into a field that has not hitherto attracted their attention to any marked degree.

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