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PREDICTION OF CANCER AND CORONARY HEART DISEASE MORTALITY BY MEANS OF A PERSONALITY INVENTORY: RESULTS OF A 15-YEAR FOLLOW-UP STUDY[1]

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Summary.—This paper reports on the 1982-1986 follow-up of two samples of healthy persons first studied in 1972 and followed up in 1982 when mortality and cause of death were established ($N = 2,146$). Both were related to stress and personality type according to clearly elaborated theories, and results were very much in accordance with theory. The second follow-up was instigated to answer criticisms of the first study and to test whether results would still support the theories involved. The data support the previous results strongly and show that psychosocial data can predict with considerable accuracy mortality and cause of death over 14 years ahead.

It used to be taken for granted during the nineteenth century and even earlier, in fact going back over two thousand years to Hippocrates and Galen, that psychosocial factors played a prominent part in the causation and development of such diseases as cancer and coronary heart disease (see Le Shan, 1959; Rosch, 1979, 1980a, 1980b). This belief was substantiated by careful systematic observations but not by statistical or experimental researches, unknown in clinical medicine at the time. The discoveries of microbes as infective agents by Pasteur gave rise to a quite different approach to disease, specifying unitary causes, although as late as 1911 Sir William Osler, known as "the father of British medicine," could state that it was often more important to know which person had the disease than which disease the person had.

These old beliefs were resurrected and tested with some rigour by Le Shan (1959, 1977), Friedman and Rosenman (1959), Kissen and Eysenck (1962), and Schmale and Iker (1971). These early studies gave promising results (Eysenck, 1985; Rosenman & Chesney, 1980), yet reviews of the literature usually led to conclusions that the prevalence of negative results precluded any over-all positive evaluation (Fox, 1978, 1981). I have suggested that such negative evaluations are unjustified because they are based on erroneous assumptions (Eysenck, 1990). We cannot reasonably count studies which are not based on good theoretical foundation, which do not use appropriate methods of investigation, and which do not use relevant inventories, and which inevitably give negative results, as outweighing stud-

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ies which are based on good theoretical foundations, use appropriate methods of investigation, which use relevant inventories, and which give positive results. To think otherwise is to commit the fundamental error underlying most meta-analyses (Eysenck, 1984b, 1992).

I have taken as an example the Schmale and Iker (1971) article in which they reported testing with considerable success the hypothesis that cervical cancer was correlated with feelings of hopelessness, using interviewers' assessments. They also used two other methods; the MMPI and a projective technique, both of which failed miserably! This is hardly surprising; both are general-purpose instruments, irrelevant to the purpose in hand (so that it would not have been reasonable to expect positive results). Furthermore, it seems probable that quite generally interviewing procedures are more likely to generate positive results than impersonal administration of questionnaires; the most positive results of the Type A–Type B studies have been those based on interviews (Rosenman & Chesney, 1980). Hence no meta-analysis type of review, which does not discard studies methodologically flawed by lack of a good theory, a proper methodology, and a relevant instrument, can claim to have disproved the correctness of the theories in question.

A favourite ploy of critics has been to suggest that certain results are "too good to be true," even though they may replicate earlier results with marked success. Thus the predictive accuracy of Le Shan (1959) of 86% in discriminating between cancer and noncancer patients on the basis of three psychological factors was better than that of Grossarth-Maticek (1979), yet the latter was said to have obtained results "too good to be true." In another study Le Shan (1977) searched for three personality factors (very similar to those used by Grossarth-Maticek) to discriminate between 152 cancer patients and 125 controls; he found loss of a crucial relationship in 72% of the former, 12% of the latter; inability to express hostility in 47% of the former, 25% of the latter; not getting over the death of a parent in 38% of the former, 11% of the latter. The results of Schmale and Iker, and of Kissen and Eysenck already mentioned, are equally clear (Eysenck, 1990). It becomes an interesting question just how many replications of a successful study, testing a long-established and widely held theory, are necessary to carry conviction? How many badly carried out studies, irrelevant to the theory, can block the acceptance of that theory? There is now a large body of research, including proper experimental tests of the theories linking personality with cancer and coronary heart disease, to suggest that objections are not based entirely on rational considerations (Temoshok & Dreher, 1992; Eysenck, 1991d; Dixon & Dixon, 1991).

It should never be forgotten that what appeared to be revolutionary new developments in medicine have often attracted violent, occasionally unrea-

sonable, and strongly prejudiced opposition—witness the fate of William Harvey, whose discovery of the circulation of the blood was greeted with obloquy (Keele, 1965); Semmelweis, whose discovery of the importance of antiseptic procedures was greeted with ridicule (Slaughter, 1950), and Pasteur, whose discovery of microbes was greeted with derision (Dubos, 1950). Orthodoxy always invokes the danger of Type One errors to ensure the occurrence of Type Two errors! In this paper I am concerned with the criticisms that have been made of the work of Dr. Grossarth-Maticek (1989) and his demonstration that psychosocial factors (e.g., stress, personality) are more important as risk factors for cancer and coronary heart disease than physical factors like smoking, drinking, cholesterol level, blood pressure, and blood sugar (Grossarth-Maticek, 1986, 1989; Grossarth-Maticek, Bastiaans, & Kanazir, 1985; Grossarth-Maticek & Eysenck, 1990; Grossarth-Maticek, Kanazir, Schmidt, & Vetter, 1982; Eysenck, 1983, 1984a, 1984c, 1988), that psychosocial and physical factors interact synergistically (Grossarth-Maticek, 1980b, 1989; Grossarth-Maticek, Eysenck, & Vetter, 1988; Eysenck, 1988, 1991b; Eysenck, Grossarth-Maticek, & Everitt, 1991), and that a special kind of behaviour therapy (autonomy training) can largely prevent cancer and coronary heart disease in people predisposed to develop these diseases and can prolong life in those terminally ill (Grossarth-Maticek & Eysenck, 1991; Eysenck & Grossarth-Maticek, 1991; Grossarth-Maticek, 1980a; Eysenck, 1991d).

Grossarth-Maticek Studies

In this section I am concerned mainly to describe the prospective studies carried out by Grossarth-Maticek (Grossarth-Maticek, 1989; Grossarth-Maticek, Bastiaans, & Kanazir, 1985; Grossarth-Maticek & Eysenck, 1990; Grossarth-Maticek, Kanazir, Schmidt, & Vetter, 1982; Grossarth-Maticek, Vetter, & Heller, 1986; Grossarth-Maticek, Schmidt, Vetter, & Arundt, 1989; Eysenck, 1991b). Three major studies were carried out, one in Yugoslavia, the other two in Heidelberg, a small university town in Germany. All started with elderly probands, selected on a complex but largely random basis, using healthy probands averaging around 60 years in the Yugoslav study and 50 years in the Heidelberg studies. One of the Heidelberg studies used a random sample; in the other, probands were nominated as suffering from stress by members of the first, random sample. Probands were followed for 10 years, and mortality and cause of death ascertained on the basis of their death certificates. Data were collected by over 100 trained interviewers, and probands were assigned scores on a 7-trait questionnaire and were also allocated to one of four types, again on the basis of a questionnaire. The 7-trait questionnaire is available in full (Grossarth-Maticek, 1979), as is the type-questionnaire (Grossarth-Maticek, Eysenck, & Vetter, 1988).

A target article describing this research (Eysenck, 1991b) was followed

by critical discussion on the part of a large number of experts in the field, followed in turn by my replies (Eysenck, 1991a, 1991c). Some of these criticisms relate to errors of analysis or of methodology; it is impossible in large-scale follow-up studies using thousands of subjects to avoid occasional mistakes, such as erroneous gender attributions, etc. What seems to be important is the direction of such errors. If they are indifferent to the hypothesis being tested, i.e., not favouring it and not increasing the probability of its being accepted, then such errors, while regrettable, are not of major basic importance (although they should, of course, be corrected in later computations). It is when errors are directional, i.e., favour the hypothesis under investigation, that they may suggest undue manipulation of data and make the data unsuitable for scientific examination. Although the original data have been made freely available to critics and although these data have been analysed and re-analysed by eminently critical statisticians like H. Vetter and equally critical epidemiologists like van der Ploeg, the few errors that have been discovered have all been of the indifferent kind, i.e., they did not favour the theory under investigation but were nondirectional (1991c).

The same appears to be true of methodological errors. In his first (Yugoslav) study, Grossarth-Maticek took the oldest inhabitant in every second household in a small Yugoslav town. Later he added a number of highly stressed probands, a move that has been criticized rightly as mixing together two different samples. It was also suggested that this might have been done to rescue nonsignificant results from the original sample by the addition of the second sample. C. D. Spielberger (private communication) subjected the original data to a re-analysis and found that far from improving the results from the original sample, the addition of the second sample actually made them worse. In other words, far from having positive directional effects, the methodological error had the opposite effect. I have seen no methodological error in this work that could be said to have had a positive directional error. Again, all such errors are regrettable, but they must be evaluated in relation to any effect they might have had on the general outcome of the study.

In view of a number of criticisms of the first 10-year follow-up of the two Heidelberg studies, and the general importance of the topic, C. R. Reynolds agreed to support a further follow-up of the two samples for another four-and-a-half years, the argument being that, as all the original data were now in the public domain and had been used by H. Vetter to produce the statistical evaluation of the original 10-year follow-up, there was no possibility of manipulation of the later follow-up, particularly as the collection and interpretation of the death certificates was supervised by an independent member of the Karlsruhe Institute of Statistics, who also interviewed a random sample of the paid interviewers who had collected the original data. Professor Spielberger was asked by C. R. Reynolds to undertake such re-anal-

ysis as that mentioned above and generally to make a thorough search of the original data; I was asked to supervise the new follow-up of the Heidelberg samples, aided by Vetter, who would carry out the statistical analysis, and by Dr. W-D. Heller, who would supervise the interpretation of the death certificates. The hypothesis under investigation was that this second follow-up would continue to show the same effects as had the first 10-year follow-up, namely, that persons diagnosed as Type 1 (cancer-prone) on the basis of the interviewer-administered inventory would tend to die of cancer, persons diagnosed as Type 2 (coronary heart disease-prone) would tend to die of heart disease, while persons of Type 4 (autonomous, healthy psychologically) would tend to survive, as would Type 3, who showed an hysterical type of personality not tending toward either cancer or coronary heart disease. Type 2 is similar to the Type A, and Type 1 is similar to the Type C concept of Temoshok (Temoshok & Dreher, 1992); Type 4 is similar to their Type B.

Several independent replications have tended to support the view that Types 1 and 2 are disease-prone, Type 4 healthy (Quander-Blaznick, 1991; Schmitz, 1992; Ranchor, Sanderman, & Bouma, 1992; Amelang & Schmidt-Rathjens, 1992), and that Types 1 and 2 are high on neuroticism, while Type 4 is low. In addition, Type 1 appears to be introverted, Type 2 extraverted. These epidemiological types thus not only have independent support, but they also fit well into an existing system of personality description (Eysenck & Eysenck, 1985). The relationship of stress to neuroticism has been discussed by Bolger and Schilling (1992). Henry (1992) has summarized the work done on the biological basis of the stress response.

The 1982-1986 Follow-up

The results of the 1982 follow-up, ten years after the start of the Heidelberg studies, showed clearly that the data supported the original hypothesis; Figs. 1 and 2 give a diagrammatic view for the random and the stressed sample, respectively (Eysenck, 1991d). Both demonstrate clearly that Type 1 probands succumb mainly to cancer, Type 2 probands to coronary heart disease, while Types 3 and 4 remain healthy. Fig. 3 shows results for the 1982-1986 follow-up; the two Heidelberg samples have been combined to achieve a group large enough (Eysenck, 1991d) for further analysis.

Table 1 (Eysenck, 1991b) gives details concerning the various groups when the 10-year follow-up was completed in 1982. The data show clearly that (1) the stressed group has a significantly higher mortality from cancer, coronary heart disease, and other causes (46% as opposed to 11%) and (2) that prophylactic treatment of cancer-prone and coronary heart disease-prone probands was successful in reducing mortality. Health instruction, i.e., advice on healthier living and coping with stress, had a similar prophylactic effect.

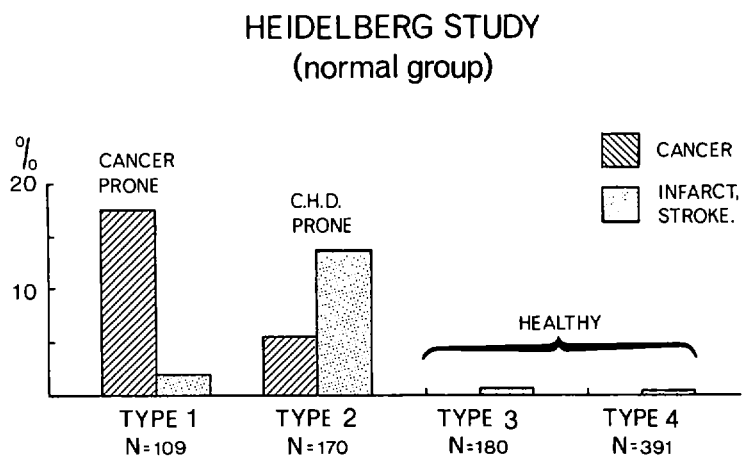

FIG. 1. Proportion of Types 1, 2, 3, and 4 probands dying of cancer and coronary heart disease, 1972–1982—Heidelberg normal sample (from Eysenck, 1991a)

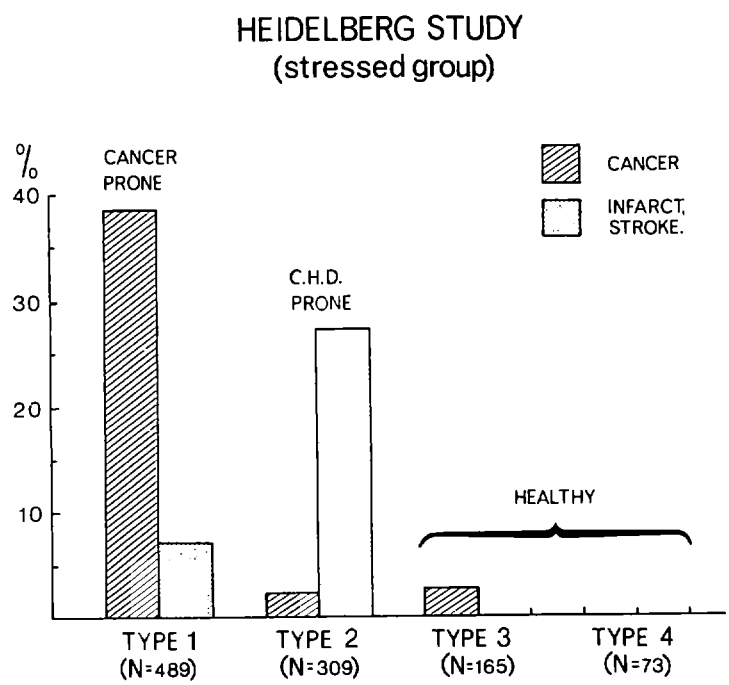

FIG. 2. Proportion of Types 1, 2, 3, and 4 probands dying of cancer and coronary heart disease, 1972–1982—Heidelberg stressed sample (from Eysenck, 1991a)

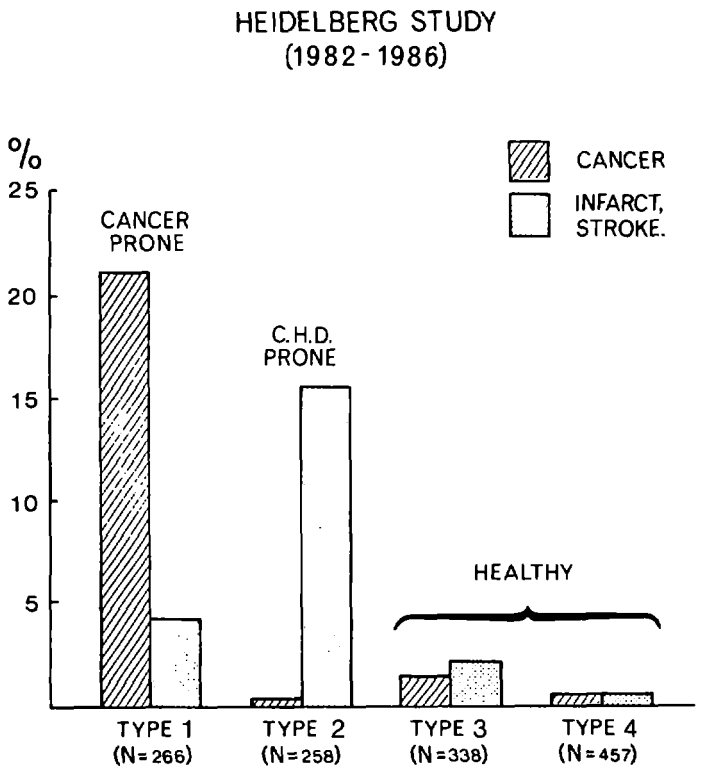

FIG. 3. Proportion of Types 1, 2, 3, and 4 probands dying of cancer and coronary heart disease, 1982–1986—Heidelberg combined samples (from Eysenck, 1991a)

Table 2 gives similar details for the follow-up of the same groups until 1986, i.e., another 4.5 years. There are many similarities. For the 10-year follow-up, the average yearly mortality was 29.54% divided by 9.5 = 3.1% per annum. For the second 4.5 year follow-up it was 13.28% divided by 4.5 = 3.0%. The difference in mortality of the stressed and normal samples is 45.7%/11.4% = 4.0 in the 10-year follow-up and 25.0%/4 5% = 5.6 for 1982 to 1986. The yearly rate of mortality, on the same comparison, is 4.8% and 5.6% in the stressed sample, and 1.2% and 1.0% for the normal sample. The differences between stressed and normal samples are clearly markedly different for both time intervals by chi-squared test.

Looking at the therapy groups, i.e., the cancer-prone and the coronary heart disease-prone subjects, a comparison with the control group shows a marked reduction in mortality for 1982-1986, equally as marked as the 1973 to 1982 reduction. Chi-squared tests for all causes of death were never less than $p < .025$. Thus the major observations of the original follow-up are preserved in the succeeding 4.5-year follow-up.

TABLE 1
STATUS OF GROUPS AT 1982 FOLLOW-UP (EYSENCK, 1991A)

Group		Still Living	Other Causes of Death	Coronary Heart Disease	Cancer	Total: % Mortality
Normal	*n*	773	43	27	29	872
	%	88.65	4.93	3.10	3.33	11.35
Stressed	*n*	566	157	120	199	1,042
	%	54.32	15.07	11.52	19.10	45.68
Cancer Control	*n*	25	4	5	12	46
	%	54.35	8.70	10.87	26.09	45.65
CHD Control	*n*	20	5	14	0	39
	%	51.28	12.82	35.90	0.00	48.72
Cancer Treated	*n*	40	3	2	0	45
	%	88.89	6.67	4.44	0.00	11.11
CHD Treated	*n*	34	5	3	1	43
	%	79.07	11.63	6.98	2.33	20.93
Health Instruction	*n*	54	1	1	3	59
	%	91.53	1.69	1.69	5.08	8.47
Total	*N*	1,512	218	172	244	2,146
	%	70.46	10.16	8.01	11.37	29.54

We must next turn to the predictive accuracy of the normative and the semi-ipsative questionnaires over the second follow-up period. Data for the 10-year follow-up period have been given in detail elsewhere (Eysenck, 1991b, and references included therein). Consider first the various trait scales and their effectiveness in predicting mortality and cause of death,

TABLE 2
STATUS OF GROUPS AT 1986 FOLLOW-UP (EYSENCK, 1991A)

Group		Still Living	Other Causes of Death	Coronary Heart Disease	Cancer	Total: % Mortality
Normal	*n*	738	10	12	13	773
	%	95.47	1.29	1.55	1.68	4.53
Stressed	*n*	425	40	50	52	567
	%	74.96	7.05	8.82	9.17	25.04
Cancer Control	*n*	16	1	0	8	25
	%	64.00	4.00	0.00	32.00	36.00
CHD Control	*n*	11	1	6	2	20
	%	55.00	5.00	30.00	10.00	45.00
Cancer Treated	*n*	39	0	0	2	41
	%	95.12	0.00	0.00	4.88	4.88
CHD Treated	*n*	31	0	3	0	34
	%	91.18	0.00	8.82	0.00	8.72
Health Instruction	*n*	53	0	0	1	54
	%	98.15	0.00	0.00	1.85	1.85
Total	*N*	1,313	52	71	78	1,514
	%	86.72	3.43	4.69	5.15	13.28

1982-1986 (Table 3). The statistical tests applied are *eta* for Scales 1 to 5, and Cramer's coefficient for Scales 6 and 7. Scale 1 is a hopelessness questionnaire, Scale 2 deals with annoyance, Scale 3 is a hopelessness rating made by the interviewers on a 10-point scale, Scale 4 is a similar rating of annoyance. Scale 5 is one of rationality-antiemotionality, used for the stressed group only. Comparisons 6 and 7 relate to the four-type classification and a three-step rating of autonomous (healthy) behaviour, respectively. It is clear that as far as mortality is concerned all scales and ratings retain statistical significance on this follow-up ($p < .001$). As far as differential prediction is concerned, Scales 1, 2 and 5 are statistically significant, suggesting that the interviewers' ratings are slightly less useful for the purpose than are the questionnaire scores. Dr. Vetter, who carried out the statistical evaluations, described the methods used (Eysenck, 1991a) as follows: "For the quantitative variables (1 to 5) I calculated *eta* against (living—cancer mortality—CHD mortality—other causes of death). For Scales 6 and 7, I calculated Cramer's coefficient of association *V*, assessing significance using chi squared. Analysis of variance was calculated for contrasts between living and dead, and cancer versus CHD" (Eysenck, 1991a, p. 320).

TABLE 3
EFFECTIVENESS OF VARIOUS PERSONALITY SCALES IN PREDICTING MORTALITY AND CAUSE OF DEATH, 1982 TO 1986 (EYSENCK, 1991A)

Scale*	*eta* or Cramer's Coefficient, *V*	*p*	*p*	
			Living v Deceased	Cancer v Coronary Heart Disease
1	.32	<.001	<.001	<.001
2	.31	<.001	<.001	<.001
3	.28	<.001	<.001	ns
4	.27	<.001	<.001	ns
5	.20	<.001	ns	<.001
6	.29	<.001	not calculated	
7	.22	<.001	not calculated	

*1 = hopelessness questionnaire; 2 = annoyances; 3 = hopelessness rating, 10-point scale; 4 = annoyances rating, 10-point scale; 5 = rationality/antiemotionality (stressed only); 6 = four-type classification; 7 = three-step autonomy rating.

The particular relationships involved for the four-type classification are treated in more detail in Table 4, which deals with the normal and the stressed Heidelberg samples, excluding all intervention groups. This table gives the data concerning the accuracy of predictions made on the basis of the four-type classification. Clearly the accuracy of the original 10-year follow-up is preserved, cancer mortality being closely related to Type 1 personality, coronary heart disease mortality being more closely related to Type 2 personality, and Types 3 and 4 showing much lower mortality over-all (particularly Type 4).

TABLE 4
MORTALITY IN DIFFERENT PERSONALITY TYPES, 1982 TO 1986 (EYSENCK, 1991A)

Type		Living	Other Causes of Death	Coronary Heart Disease	Cancer	Total
Type 1	*n*	179	20	11	56	266
	%	67.29	7.52	4.14	21.05	
Type 2	*n*	195	22	40	1	258
	%	75.58	8.53	15.50	0.39	
Type 3	*n*	321	5	7	5	338
	%	94.97	1.48	2.07	1.48	
Type 4	*n*	448	3	3	3	457
	%	98.03	0.66	0.66	0.66	
Total	*N*	1,143	50	61	65	1,319
	%	86.66	3.79	4.62	4.93	100.00

Vetter also carried out an analysis of the interactions between physical and psychosocial risk factors, paralleling the earlier study of the 1973-1982 follow-up (Grossarth-Maticek, Eysenck, & Vetter, 1988). His report was as follows (Eysenck, 1991a): "To investigate the interactions between physical and psychosocial risk factors, multiple-regression analysis was carried out, and significance levels calculated according to Vetter's formula (Vetter, 1988). The first analysis concerned smoking (number of cigarettes smoked per day) and as the chosen psychosocial factor a linear combination of hopelessness (1 and 3) and rationality/antiemotionality (5). The interaction (product term) was significant at the $p = .018$ level. The picture is as follows: For probands whose psychosocial risk scores lie 1 *SD* below the average, the risk for smoking lies hardly at all above 0; it increases with increasing scores on the psychosocial variable" (p. 321).

"Concerning coronary heart disease and smoking, the psychosocial variable was a linear combination of the two anger-annoyance indicators (Scales 2 and 4). The interaction (product term) was significant at $p < .0001$, with similar regression slopes as in the case of cancer.

"Looking finally at the interaction between blood pressure and psychosocial factors for coronary heart disease mortality, there was no interaction either for systolic or for diastolic measures. This is unlike the results of the analysis of 1973-1982 mortality data" (Eysenck, 1991a, p. 321).

It will be clear that in all major respects the second follow-up study gives results not dissimilar to those of the original 1972-1982 investigation.

A Special Test for the Genuineness of the Data

Do those calculations remove all doubts about the genuineness of the Grossarth-Maticek data? Vetter (1991c) raised one further question which he dubbed the "morbidity hypothesis" in the spirit of the *advocatus diaboli*. I quote his own words: "Doubts about the prospective validity of the data

were raised by the fact that practically all predictors worked better for deaths from 1978 to 1982 than for deaths from 1973 to 1977, whereas I should have expected that an initial state measured in 1972 should become progressively less relevant to deaths occurring at greater temporal distance from it because it may have changed in the meantime. This led me to the hypothesis that Dr. Grossarth-Maticek's assistants, on the occasion of finding out about deaths in 1982 (for which the causes of death were ascertained by personnel from the two independent university institutes mentioned), might also have learned from relatives and neighbours about causes of death, as well as the presence, in survivors, of cancer or a cardiac infarction or stroke history; and that on the basis of this information, psychosocial (and physical) data might have been assigned to subjects, thus securing 'predictions' of deaths not only up to 1982, but also continuing from 1982 to 1986 on the basis of premorbidity. The information being more accurate for years shortly before 1982 than for years further back, relationships were stronger for 1978-1982 than for 1973-1977. When I communicated this hypothesis to Dr. Grossarth-Maticek, he said that the morbidity part of the hypothesis could, and should, be tested empirically (preferably by independent researchers) by asking relatives and/or doctors of subjects deceased in 1982-1986 when cancer or cardiovascular disease first became known. In fact, if prediction worked approximately as well for first knowledge after 1982 as for knowledge before 1982, I am prepared to admit that the morbidity part of my thesis is refuted" (p. 287).

Analysing a small sample of probands, Vetter (1991a) used the following method. He divided all deaths that occurred from 1973 to 1986 into those from the periods 1973-1978, 1979 to mid-1982, and mid-1982 to 1986, and discriminated each of these groups from the survivors on the basis of the personality inventories. "The result was that all correlations were higher for the first period than for the second period and that they dropped then from the second period to the third period. This is exactly the expectation I uttered in my commentary. Hence, the motivation for the 'morbidity hypothesis,' which had been disproved in the limited test anyway, evaporates, and I herewith wish to retract it" (Vetter, 1991a, p. 323).

Vetter (1991b) took the testing of the morbidity hypothesis one stage further. If it be agreed that there is a possibility that Grossarth-Maticek's psychosocial predictions of death and causes of death in Heidelberg after 1982 might have been due to artificial assignment by the interviewers of the predictors on the basis of existing cancer or a history of cardiovascular disturbances in the subjects, the best test of such an hypothesis, it was agreed at a meeting between Grossarth-Maticek, Vetter, and myself, was to compare the predictive successes for cancer in which this first knowledge was available prior to 1982 ("prior group") with cases for which it was available only

later ("later group"). For the latter group, for instance, cancer was not diagnosed until after information had been collected for the individuals concerned and could therefore have played no part in the prediction.

Concerning the method of analysis to be used, Vetter (1991b) argued as follows: "It was decided to predict the cancer deaths with a multiple regression model. The predictors had to be selected from the variables [scales] handed over to me in 1982 and deposited by me at two independent university institutes. Selection was made in terms of the bivariate correlations with cancer deaths after mid-1982. Prediction was made separately in the normal and in the stressed samples, and the sets of available predictors were not quite identical. Two sets of predictors were used in each case, a smaller one including only variables related to cancer and a larger one containing also variables related to other causes of death. Furthermore, in the stressed sample, where a considerable number of cancer deaths besides those in the two prediction groups were available, two prediction strategies were used. The first was the same as the only one available in the normal sample, namely, using the given set of predictors to discriminate the 'prior' group and the 'later' group of cancer deaths, respectively, from all subjects who had not died of cancer, and to compare the two multiple correlations. The other strategy, available in the stressed sample only, consisted in deriving the regression coefficients of the predictors from those cancer deaths after mid-1982 for which neither 'prior' nor 'later' information was available, to use this regression function for discriminating the 'prior' and the 'later' group, respectively, from the noncancer subjects, and to compare the two correlations" (p. 322).

The smaller and the larger sets of predictors in the two subsamples are shown in Table 5 (Vetter, 1991b, p. 323).

Vetter described the method used as follows: "Among the cancer deaths that occurred in the Heidelberg cohort from mid-1982 to 1986 there were a few for which the duration or first diagnosis of cancer was recorded on the

TABLE 5
CANCER-RELATED AND OTHER PREDICTORS OF CANCER DEATHS BY SUBSAMPLE (VETTER, 1991B)

Subsample	Smaller Set (Cancer Predictors Only)	Additional in Larger Set
Normal Only	Interviewers' rating of rationality with respect to withdrawing objects	Interviewers' rating of rationality with respect to disturbing objects
Stressed Only	Rationality/Antiemotionality	
Both Samples	Number of life events leading to hopelessness	Number of life events leading to anger
	Intensity of hopelessness	Intensity of anger
	Interviewers' rating of hopelessness	Interviewers' rating of anger
		Need for harmony and closeness
		Nonhypochondria

death certificate. The idea of the test was to compare the predictive success for cases in which this first knowledge was available prior to 1982 with cases in which this knowledge was available only later. In the Heidelberg stressed sample (including control groups, but excluding intervention groups), first knowledge prior to 1982 was recorded for 10 cases and later for 9 cases; in the normal sample, prior knowledge was recorded for 1 case and later for 4 cases. Because a comparison of the prediction for 4 cases and a single case did not seem advisable, the 4 later cases were compared with all of the rest, namely, 13 cancer deaths.

The unequal size of the two groups makes necessary some caution with respect to the possible size of the (point-biserial) correlations. We have generally: $R^2 = V_s/V_t = V_s/(V_s + V_e)$, where V_s is the systematic variance, V_e is the error variance, and $V_t = V_s + V_e$ is the total variance of the dependent variable. In the point-biserial case, the error variance is the variance of the dependent variable within the two groups. The systematic variance is $p(1 - p)d^2$, where d is the difference of the means of the two groups, and p, $1 - p$ are the relative frequencies of the two groups. We consider the influence of p on R^2 when d and V_e are constant. We have

$$R^2 = \left[p(1 - p)d^2\right] / \left[p(1 - p)d^2 + V_e\right] .$$

When R^2 is small, it is approximately proportional to $p(1 - p)d^2$; and when p is small, $p(1 - p)$ is approximately proportional to p. Hence, when R^2 and p are small, R^2 is approximately proportional to p. Under these conditions, then, R^2 is approximately proportional to the size of the smaller group when the group means and the within-group variance are constant. So in the group of 4 subjects we have to expect an R^2 less than one-third of that in the group of 13 subjects when other things (i.e., the goodness of the prediction in terms of d and V_e) are equal" (Vetter, 1991b, p. 322).

Results are shown in Table 6. This shows the squared multiple correlations produced by the sets of predictors used. All the squared multiple correlations (or squared bivariate correlations in the case of the regression coefficients derived from different subjects) are significantly different from zero ($p < .05$), with the exception of those in the stressed sample, prior group, where $p = .07$ and $p = .17$, respectively.

Vetter comments: "I cannot think of any possibility of manipulating the predictors supplied in 1982 with respect to deaths afterward once the use of morbidity is ruled out. Hence, even given all the doubts put forward in the commentary, it should be admitted that Grossarth-Maticek has succeeded in making at least some genuine psychosomatic predictions. It is deemed highly desirable, however, to extend the test to all (or most) of the deaths that occurred from 1982 to 1986 by collating information from doctors and/or rela-

TABLE 6
PREDICTOR SQUARED MULTIPLE CORRELATIONS WITH CANCER DEATHS (VETTER, 1991B)

Samples	*n*	Set of Predictors	
		Smaller	Larger
Normal Sample			
Not Later Group	13	.0263	.0415
Later Group	4	.0142	.0357
Stressed Sample			
Prior Group	10	.0157[a]	.0234[b]
Later Group	9	.0260	.0354
Regression Coefficients Derived From Rest of Cancer Deaths			
Prior Group	10	.0135	.0169
Later Group	9	.0191	.0218

Note.—Values differ significantly from zero ($p < .05$) except for the stressed sample, prior group.
[a] $p = .07$. [b] $p = .17$.

tives, when the disease that led to death first became known" (Vetter, 1991b, p. 323).

Since then, an extended test of the hypothesis has been carried out by the Surgical University Clinic of Heidelberg, collating information on further subjects who had died of cancer in 1982-86 as to when the disease had first become known. Results were analysed by Vetter, who sent me the following reports. It will be seen that the morbidity hypothesis is again refuted.

"The method applied to the new subjects was precisely the same as that described by Vetter in 1991, with the addition of applying the regression function derived from the rest of the cancer deaths to the 'known before mid-1982' and 'known after mid-1982' groups also in the normal sample. The following table (Table 7) reports the squared multiple correlations with cancer death and their significance for the various groups, two sets of predictors and two methods of prediction.

"If, in view of the smallness of the groups, a generous significance level of .10 is admitted, all the multiple correlations are significantly different from zero with the exception of those derived from the rest of the cancer deaths in the 'prior' group of the normal sample. When comparing R^2 for the 'prior' and the 'later' groups, it must be borne in mind that for the same relative mortality difference, R^2 is approximately proportional to the group size. Hence, in the normal sample, an equivalent R^2 in the 'later' group should be twice that in the 'prior' group, and in the stressed sample it should be nearly three times that in the 'prior' group. This requirement is fulfilled three out of four times in the normal sample, and in the fourth case where it is not (larger set of predictors, regression coefficients not derived from the rest of the deaths), R^2 at any rate is greater and more strongly significant in the 'later' group. In the stressed sample, the requirement is

TABLE 7
PREDICTOR SQUARED MULTIPLE CORRELATIONS WITH CANCER DEATHS

	Set of Predictors			
	Smaller		Larger	
	R^2	p	R^2	p
Normal Sample				
Prior Group (3 cases)	.011	.090	.022	.100
Later Group (6 cases)	.023	.002	.031	.011
Regression coefficients derived from rest of cancer deaths				
Prior Group (3 cases)	.000	1.000	.001	1.000
Later Group (6 cases)	.007	.019	.007	.022
Stressed Sample				
Prior Group (10 cases)	.017	.06	.033	.035
Later Group (29 cases)	.067	.000	.076	.000
Regression coefficients derived from rest of cancer deaths				
Prior Group (10 cases)	.015	.003	.023	.001
Later Group (29 cases)	.054	.000	.056	.000

fulfilled for the two comparisons involving the smaller set of predictors, and in the two other cases, R^2 in the 'later' group at any rate is more strongly significant. To summarize, the above extended test of the 'morbidity hypothesis' again on the whole speaks against it quite clearly" (private communication, June 19, 1992).

DISCUSSION

The demonstration here offered of the genuineness of the Grossarth-Maticek data is important because potentially his work is more extensive, theory-based, and successful than most of the prior studies that have reported on psychosocial factors in the causation of cancer and coronary heart disease. His work has been criticized on many points, but these criticisms were demonstrably not in accord with reality (Eysenck, 1991c; Bachman, 1981), as these two references make clear. However, the existence of captious and irrational criticism does not mean that there cannot be any well-based and meaningful criticisms; indeed, work on the scale carried out by Grossarth-Maticek, involving tens of thousands of subjects and some 3 million data entries, would be unique if careful scrutiny unearthed no investigative errors, methodological inadequacies, and occasional lapses on the part of the workers engaged to collect the data. Many valid criticisms can be found in the commentaries (Commentaries, *Psychological Inquiry*, 1991) following my target article (Eysenck, 1991b) describing his work. However justified, these criticisms do not invalidate the major conclusions reached by Grossarth-Maticek.

Obviously what is needed to establish the credibility of Grossarth-Maticek's work is a design which does not admit of any manipulation and is carried out by independent agents on data made available prior to the mor-

tality observations. This would seem to be the case in the study here reported, where mortality data were collected by an independent medical group and the original psychosocial data were held by the major critic (Vetter) who also carried out the statistical analysis. It would be difficult to argue with his conclusion that this analysis would seem to establish the genuineness of the Grossarth-Maticek data. All the data collection was, of course, carried out by Grossarth-Maticek, who also framed the theories on which the work is based and constructed the instruments by which to test them. My own function was simply that of supervision, aided by several people already named, and of trying to integrate this work into a thought-structure more familiar to English-speaking readers. This paper thus contains my evaluation of Grossarth-Maticek's data, as analysed by Vetter, who articulated the only testable counter-hypothesis to the Grossarth-Maticek series of studies. The fact that the data disproved his own hypothesis is an important fact in this long-continued debate.

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