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SELF-REGULATION AND MORTALITY FROM CANCER, CORONARY HEART DISEASE, AND OTHER CAUSES: A PROSPECTIVE STUDY

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Summary—This article introduces a new personality inventory dealing with self-regulation. This is in some ways the opposite of neuroticism, and measures personal autonomy or independence, particularly as far as emotional dependence is concerned. Our concern was the relation between self-regulation and health, and large samples of healthy men and women were tested and followed up to demonstrate high predictability of mortality from cancer, coronary heart disease and other causes of death from scores on the questionnaire. It was also demonstrated that psychological risk factors were largely independent from physical risk factors, and could be changed by behavioural–cognitive treatment, reducing mortality.

INTRODUCTION

The ancients had a motto for happiness: *Mens sana in corpore sano*. They believed, following Hippocrates, that the sound mind was related to the sound body, and that there were cancer-prone personalities predisposed to develop this disease more readily, and die of it more quickly, than others not so prone (Mettler & Mettler, 1947; Kowal, 1955; Greer, 1983; Rosch, 1979, 1980). In recent years, many studies have given support to the idea of a cancer-prone personality (Eysenck, 1991, 1994a; Temoshok & Dreher, 1992), as well as a coronary heart disease-prone personality (Friedman, 1991; Johnson, 1990; Turner, Sherwood & Light, 1992). The latter is often referred to as Type A, contrasted with the healthy Type B (Eysenck, 1990); the cancer-prone type is sometimes referred to as Type C. Disease-prone types share certain similarities, but can be differentiated successfully both experimentally (Kneier & Temoshok, 1984) and by interview/questionnaire (Eysenck, 1988).

While most interest has been directed towards Types A and C, there has also been some interest in the study of the healthy type of person, Type B (Friedman & Rosenman, 1974) or Type 4 (Grossarth-Maticek, Eysenck & Vetter, 1988). This may be defined *negatively* in terms of the absence of traits characteristic of cancer-prone and CHD-prone personalities, or *positively* in terms of active health-promoting traits. The difference is of course of little practical importance; a given trait may be formulated positively, or negatively, and scored in the health-giving or disease-prone direction. Table 1 shows some of the concepts related to the disease-prone and the healthy personality (Friedman & Booth-Kewley, 1987), respectively. Obviously these varied conceptions have a great deal in common, and the present study reports an attempt to bring this consensus to a focus, and demonstrate its relevance to actual physical health. What seemed to us to be the defining feature of the healthy personality was autonomy, emotional independence, and self-regulation, i.e. the ability to actively regulate one's own life, without a degree of emotional dependence on other people that acted in such a way as to thwart one's needs and aims. The concept of 'locus of control' has some similarity to self-regulation, but is rather narrower in its meaning. Both are clearly related to *low neuroticism* (Eysenck, 1994a).

The term 'self-regulation' has been used in the past with similar but somewhat differing meaning (Schwartz, 1983; Leventhal, Nerenz & Strauss, 1980; Carver & Scheier, 1982) as an aspect of control theory. These authors review self-regulation in terms of coping with symptoms or medical treatments

Table 1. Healthy and disease-prone psychological types

Disease-prone personality	Healthy personality
Alexithymia (Lesser, 1981; Taylor, 1994)	Self-directedness (Carver, Peterson, Follanbee & Scheier, 1983)
Lack of self-attention (Suls & Fletcher, 1985; Mullen & Suls, 1982)	Hardiness (Kobasa, 1979)
Helplessness (Seligman, 1975)	Self-efficacy (Bandura, 1977)
Infantile personality (Ruesch, 1948)	Type B (Friedman & Roseman, 1974)
Negative affect (Cohen, Gwaltne, Doyle, Shoner, Firman & Newson, 1955)	Autonomous type (Grossarth-Maticek, 1976, 1989)
Exploratory style (Peterson & Seligman, 1987)	Self-regulation (Mithaug, 1993; Taylor, 1992)
Expression (Kreitler & Kreitler, 1990; Kissen & Eysenck, 1962)	Control (Carver & Scheier 1982; Langer, 1981)
	Self-control (Barrios, 1985)

as a part of a complex system of negative feedback loops. On this view people take action when they experience symptoms that suggest the existence of a discrepancy between their present state and their standard of comparison (i.e. good health). The experience of distress is a negative feedback process that serves as a prerequisite for engaging in appropriate health-seeking behaviour. Failure to monitor critical cues or to attend to them can result in the breakdown of the system. Such a disconnection between input and output Schwartz (1983) has labelled 'disregulation'. Following up this line of argument has led Suls and Fletcher (1985) to stress the importance of self-attention for health, meaning by this attention to internal signs of a psychological or somatic nature, leading to some cognitive behavioural action to reduce the discrepancy.

Our own use of the term 'self-regulation' goes well beyond this definition. In labelling this trait 'self-regulation' and calling it a personality trait, we are obviously choosing a short-hand term to cover a conglomerate of concepts. Personality is a central concept that describes, inter alia, certain behaviours that determine one's reaction to a variety of *stressors*, and includes a variety of *coping mechanisms*. The very definition of *stress* is tied up with the concept of personality; what is a stress to one person may be a welcome motivator to another, and quite neutral to a third. *Stress* can only be defined in terms of *strain*, i.e. the reaction of a given person to an objective occurrence, say the death of a loved one, or the loss of one's job. Personality, stress and coping are intimately tied together, and the term 'personality' is used here to cover a multitude of factors each of which can also be independently studied.

'Healthy' and 'unhealthy' typologies

The intimate connection of healthy and unhealthy typologies, coping types, personality and behaviour is illustrated in Tables 2 and 3. These summarize investigations published by Schmitz (1992, 1993) and Sandin, Chorot, Jimenez and Santad (1993a); Sandin, Chorot, Santad and Jimenez (1993b), referring to correlational studies of various inventories with the six Grossarth Maticek types (Grossarth-Maticek & Eysenck, 1990). Of these, present interest is in Type 1 (cancer-prone behaviour), Type 2 (CHD-prone behaviour), and Type 4 (healthy, autonomous behaviour). Type 3 is an hysterical type, Type 5 shows rational, anti-emotional behaviour, and Type 6 is a sociopathic type. Positive relations are indicated by + signs, negatives by − signs, and absence of any relation by a 0.Number of + or − signs indicates the strength of a relation. As several studies involving different samples are being summarized, the indicators are overall estimates and may not exactly mirror the details of any one study. To these two studies, one German, the other Spanish, could be added a Japanese one by Shigehisa (1994) which shows interesting cultural similarities and differences.

There are two problems in this type of work that should always be borne in mind. The first is that the validity of a questionnaire depends crucially on the mode of administration (Grossarth-Maticek, Eysenck & Barrett, 1993; Grossarth-Maticek, Eysenck & Boyle, 1995). Simply handing out questionnaires demanding complex introspections and important self-revelations has the least validity;

Table 2. Personality and other correlates of the six Grossarth-Maticek types (Schmitz, 1992, 1993)

Types	1	2	3	4	5	6
N	+ + +	+ + +	+ +	− − −	+	+ + +
E	− −	−	−	+ +	−	+
P	=	+	=	−	− −	+ +
L	=	=	=	=	+	−
Autonomic anxiety	+ +	+	=	−	+	=
Cognitive anxiety	+ +	+ +	+	− −	+	+ +
State anxiety	+ +	+ +	=	− −	+	+
Dogmatism	+ + +	+ +	+ +	− −	+	+ +
Alienation	+ + +	+ +	+ +	−	+	+ +
Can't describe feelings	+ + +	+ + +	=	− −	=	+
Can't communicate feelings	+ + +	+	+	−	+ +	+
Alexithymia	+ +	=	=	−	+	=
Task-oriented	=	−	=	+ +	+	−
Emotion-oriented	+ + +	+ + +	+ +	− −	=	+ +
Coping						
Avoidance-oriented	+ +	+ +	+	=	=	=
Distraction	+ +	+ + +	+	=	=	=
Social diversion	=	=	+	=	=	=
Psychosomatic complaints						
Physical exhaustion	+ +	+ +	+	− −	+	+
Insomnia	+	+	+	− −	+ +	+ + +
Cardiovascular problems	+ +	+ +	+	−	+ +	+ +
Depressive tendencies	+ +	+ + +	+ +	− −	+ +	+ + +
Impulsiveness	+	+ + +	+ +	− −	=	+ + +
Abuse of drugs						
Psychopath.	+	+ +	=	−	=	+
Alcohol	+ +	+ +	=	−	+ +	+ + +
Drugs	+ +	+ +	=	− −	=	+
Smoking	+	+	+		=	+ +

interviewer-administration involving the establishment of *trust*, and the *explanation* of obscure or complex questions, has the greatest validity. The second problem is that response may depend on the circumstances leading to the establishment of a given sample; a sample consisting of people coming for psychotherapy is more likely to give truthful answers than a random sample uncertain about the relevance of the questions asked. These problems are closely linked to the hypothesis that a major aspect of the cancer-prone personality, for instance, is the suppression of feelings and emotional responses; such denial may lead to differential responding in different conditions of test

Table 3. Personality of other correlates of the six Grossarth-Maticek types (Sandin *et al.*, 1993a, b)

Types	1	2	3	4	5	6
Immunological	+	+	+	−	=	+
Cardiovascular	+	+ + +	+	−	=	+
Respiratory	+	+ + +	=	−	=	+
Gastrointestinal	+	+ +	+	−	=	+
Neurology: sensorial	+ +	+ + +	+ +	−	−	+ +
Skin	+	+ +	+	=	=	+
Musculoskeletal	+ +	+ + +	+	−	−	+
Genito-urinary	+	+ +	+	−	=	+
N	+ +	+ +	+ +	−	=	+
E	− −	=	=	+	=	=
P	=	+ +	=	=	=	+
Alexithymia	+ +	=	=	=	=	=
Coping						
Task-oriented	−	−	−	+ +	=	=
Emotion-oriented	+	+	+	−	=	+ +
Avoidance-oriented	+	+ +	+	−	=	+
Social support	− −	=	+	+	=	=
		STAXI				
Anger-state	=	+	=	=	=	+
Anger-strait	+	+ +	+ +	−	=	+ +
Anger-in	+ +	+	+ +	+	=	+ +
Anger-out	=	+ +	+ +	=	=	+ +
Anger-control	=	−	−	+	=	−
Anger-ex	+ +	+ +	+ +	=	=	+

administration. Intelligence, too, may play a part; complex questions embodying complicated theories may not be easily understood by persons with below-average IQs.

If we may take the results of the studies summarized in Tables 2 and 3 as suggesting the nature of the 'healthy' (Type 4) and 'unhealthy' (Types 1 and 2) personality, we see that the 'healthy personality' is low in psychopathology (neuroticism and psychoticism), extraverted, task-oriented rather than emotion-oriented, and controlled in his anger. We may compare these characteristics with those noted in an early but still valuable study that played a pioneering role in this field (Hinkle & Wolff, 1957). They studied three rather homogeneous groups, composed of over 4000 men and women, looking at their history of major and minor illnesses, as well as their circumstances, personalities, and stresses and stress reactions. Their first finding was that the distribution of illnesses was not Gaussian, but negative binomial, a sort of distribution that occurs in groups when the members of the group have different 'risks' of becoming ill. In other words, people *differ in their predisposition to become ill.* In addition, those so predisposed showed an increased susceptibility to illness *in general*; they developed many different types of minor or major illness, not just one or two. (Number of major illnesses correlated 0.40 with number of minor illnesses.) There was a clear correlation between number of illnesses and stress experienced, in terms of objective events like divorces, separations, conflicts with family members, uncongenial living and working arrangements, etc. Further, clusters of illness often occurred during periods of significant stress. Constitutional differences predisposing to disease have not been found to differentiate the 'healthy' from the 'diseased'. The subjectivity of the 'stresses' involved becomes apparent in the conclusion drawn by the authors "that illness often occurs when a person perceives his life situation as peculiarly threatening to him, even though this life situation may not appear to be threatening to an outside observer, and that people who maintain good health in a setting of what are 'objectively' difficult life situations do not usually perceive these situations as difficult."

The study closely targeted psychological factors similar to those found in Tables 2 and 3 as related to illness predisposition. "Those people who had the greater number of bodily illnesses, regardless of their nature and regardless of their etiology, were the ones who experienced the greater number of disturbances of mood, thought, and behaviour. For example, not uncommonly, persons were seen with recurrent episodes of anxiety, depression, chronic obsessive and compulsive symptoms, or character disturbances; symptoms of this type, with exacerbations and remissions, might predominate in their illness pattern throughout life. But such people, as a group, also *had more bodily illnesses of all types* than were found among those who had few or no disturbances of mood, thought, or behaviour. This can be put in other terms by saying that ... *there was a parallelism between the occurrence of psychoneuroses and psychoses and the occurrence of bodily illness.*" (p. 446; italics not in original).

THE SELF-REGULATION INVENTORY (SRI)

To investigate the hypothetical relationship between personality and illness, a self-regulation inventory was constructed using questions based on those that had in past research proved useful in predicting good health or poor health respectively, reversing the scoring for the latter so that a high score indicated good health, a low score poor health. Likert-scale scoring on a six-point scale was used. Scores can vary between 105 and 630. The Cronbach α reliability for various groups centred on 0.80. For purposes of presentation scores were grouped into six groups, from 1 (low self-regulation) to 6 (high self-regulation). The six steps are coded in multiples of 105. Thus a score of 1 is obtained when the total point score is between 105 and 209; a score of 2 is obtained when the total point score is between 210 and 314, etc. The number of men and women with each score is given in Table 4. A detailed statistical analysis of the questionnaire will be given in a later publication; here we shall be concerned with the *validity* of the questionnaire as regards predictive accuracy of mortality. *Ss* were tested by trained interviewers in 1973, and mortality established in 1988; thus the study reports a 15-year follow-up. Data were collected by 116 trained students in all. *Ss* were randomly selected on the basis of lists of inhabitants in Heidelberg, Germany, at the time. (Copies of the questionnaire can be obtained from H. J. Eysenck.)

Table 4 shows the degree of self-regulation for the men and women who took part in the study.

Table 4. Degree of self-regulation and mortality in women and men

Group	1	2	3	4	5	6	Total
Women	150	316	535	912	502	193	2608
	5.7%	12.1%	20.5%	34.9%	19.2%	7.4%	
Men	154	509	1221	813	308	103	3108
	4.9%	16.3%	39.2%	26.1%	9.9%	3.3%	
Total	304	825	1756	1725	810	296	5716
	5.3%	14.4%	30.7%	30.1%	30.1%	5.1%	

Table 5. Degree of self-regulation and mortality in women

	Group					
	Score 1	Score 2	Score 3	Score 4	Score 5	Score 6
N	150 5.7%	316 12.1%	535 20.5%	912 34.9%	502 19.2%	193 7.4%
Cancer	25 16.6%	43 13.6%	58 10.9%	35 3.8%	15 2.9%	4 2.0%
CHD	45 30.0%	60 18.9%	96 17.9%	51 5.5%	14 2.7%	5 2.5%
Other causes of death	52 34.6%	79 25.0%	147 27.4%	130 14.2%	37 7.3%	7 3.6%
Still alive	28 18.6%	134 42.4%	234 43.7%	696 76.3%	436 86.8%	177 91.7%
Total mortality	122 81.3%	182 57.5%	301 56.2%	216 23.6%	66 13.1%	18 8.2%
Average age (1973)	55.7	56.1	57.8	58.3	56.9	58.8

Table 6. Degree of self-regulation and mortality in men

	Group					
	Score 1	Score 2	Score 3	Score 4	Score 5	Score 6
N	154 4.9%	509 16.3%	1221 39.2%	813 26.1%	308 9.9%	103 3.3%
Cancer	22 14.2%	63 12.3%	126 10.3%	29 3.5%	8 2.5%	2 1.9%
CHD	49 31.15%	121 23.7%	251 20.5%	48 5.9%	10 3.5%	2 1.9%
Other causes of death	51 33.1%	128 25.1%	349 28.5%	92 11.3%	15 4.8%	5 4.8%
Still alive	32 20.7%	197 38.7%	495 40.5%	644 79.2%	275 89.3%	94 91.2%
Total mortality	122 79.2%	312 61.2%	726 59.4%	169 20.7%	33 10.7%	9 8.7%
Average age (1973)	57.8	56.5	55.9	57.2	58.9	58.4

It is clear that women are significantly higher on the S–R scale ($P < 0.001$ by Mann-Whitney U-test). This agrees well with the universal tendency of women to live longer than men.

Tables 5 and 6 show, separately for women (Table 5) and men (Table 6) the interaction between degree of self-regulation and mortality from Cancer, CHD, and other causes of death. Also given are number still living and total mortality. χ^2 values were calculated for total mortality vs still living, cancer vs still living, CHD vs still living, and other causes of death vs still living; all were significant at $P < 0001$ for the sexes separately. Also given are the average ages of the S–R groups. (Ages ranged from 45 to 68 yr in 1973.) Thus for all causes of death (cancer, CHD, other) there is a very significant correlation between S–R and mortality. Figures 1 and 2 show the results diagramatically.

Table 7 shows the relationship between S–R scores and a number of risk factors in a small group of 571 persons where more detailed investigation was possible. Clearly those low on self-regulation have higher blood pressure, suffer more from diabetes, are more overweight and lacking in exercise,

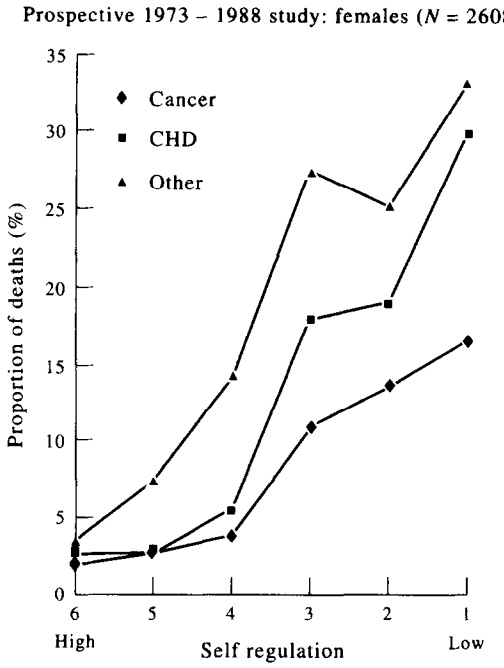

Fig. 1. Mortality and degree of self-regulation; 2608 women.

smoke more, drink more, have more accidents, have a poorer diet, are more often ill, spend more time in hospital, and report more symptoms leading to medical treatment. All these are at high levels of significance, with $P < 0.001$.

Table 8 lists smokers in relation to self-regulation for men only. There are two groups, those still alive, and those who had died. (There were too few women smokers in 1973 to make results meaningful.) Among the former, smoking is positively related to *higher degrees in self-regulation.* In those who died, smoking was more frequent in those with low self-regulation, and they smoked

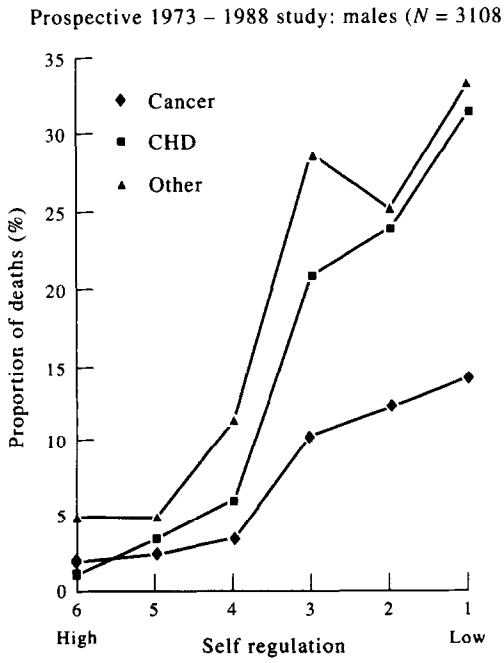

Fig. 2. Mortality and degree of self-regulation; 3108 men.

Table 7. Self-regulation as related to various physical risk factors

	Type 1	Type 2	Type 3	Type 4	Type 5	Type 6
n	304	825	175	172	810	296
Blood pressure	168/93	155/90	144/86	135/75	123/71	121/70
Diabetes	39	68	69	11	2	1
	12.6%	8.2%	3.9%	0.6%	0.2%	0.3%
Overweight	183	478	801	159	40	13
	60.0%	57.9%	45.6%	9.2%	4.9%	4.3%
Lack of exercise	194	536	961	201	62	10
	63.8%	64.9%	54.7%	11.6%	7.6%	3.3%
Number of cigarettes smoked per day	40.2	35.1	30.6	15.1	11.2	7.7
Alcohol consumed daily (g)	83.6	80.2	64.9	19.8	11.6	10
Number of accidents per year treated individ. (1970–1973)	84	155	167	90	10	1
	27.5%	18.7%	9.5%	5.2%	1.2%	0.3%
Poor nutrition	265	557	718	401	89	19
	87.1%	67.5%	40.9%	23.2%	10.9%	6.4%
Days ill per year (1970–1973)	64.7	57.2	31.5	16	18	15
Days in hospital per year (1970–1973)	22.8	20.6	10.6	4.3	2.5	1.1
Needing medicare care over 1 yr	71	125	216	99	36	8
	23.3%	15.1%	12.3%	5.7%	4.4%	2.7%
Number of symptoms leading to medical treatment (1970–1973)	14.3	12.8	11.4	4.7	2.3	1.2

more per day. These results for the relation between smoking and self-regulation may at first seem contradictory, but both are highly significant by χ^2 ($P < 0.001$). The results are in good agreement with previous studies (e.g. Friedman, Firman, Petitti, Siegelaub, Ury & Klatsky, 1983; Howard, Cunningham & Rechnitzer, 1985) which demonstrated that personality acts as a moderator of the effects of cigarette smoking on coronary risk, in the sense that smoking was having deleterious effects on health only for people with CHD-prone personality, but not on those with psychologically healthy personalities. Eysenck (1994b) has shown that this effect occurs equally for cancer, and the results in Table 8 are clearly in line with this general rule.

Data for alcohol consumption are given in Table 9. Among those alive in 1988, the relation between drinking and degree of self-regulation is reasonably linear, with low S–R scorers drinking less than

Table 8. Self-regulation and smoking—in live and dead probands

	Type 1	Type 2	Type 3	Type 4	Type 5	Type 6	Total
N	154	509	1221	813	308	103	3108
			*Group I**				
Still alive	32	197	495	644	275	94	1737
							55.9%
Smokers (*n*;%)	9	57	164	303	108	49	690
	28.1%	28.9%	33.1%	47.0%	39.3%	52.1%	39.7%
Cigarettes per day	15.3	15.6	14.7	24.6	21.7	22.0	
Total mortality	122	312	726	169	33	8	1370
							44.1%
			Group II†				
No longer living	122	312	726	169	33	8	1370
							44.1%
Smokers (*n*;%)	91	224	415	89	7	1	807
	74.5%	71.7%	57.1%	40.8%	21.2%	12.5%	59.0%
Cigarettes per day	26.9%	25.6%	24.3%	23.9%	21.3%	21.3%	
Total smokers	100	281	579	372	115	50	1497
	64.9%	55.2%	47.4%	45.7%	37.3%	48.5%	48.2;c

*χ^2 (linear) = 20.63, d.f. = 1, $P = 0.0000$.
†χ^2 (linear) = 70.59, d.f. = 1, $P = 0.0000$.

Table 9. Self-regulation and drinking—in live and dead probands

	Type 1	Type 2	Type 3	Type 4	Type 5	Type 6	Total
N	154	509	1221	813	308	103	3108
			*Group I**				
Still alive	32	197	495	644	275	94	1737 55.9%
Alcohol consumed (*n*;%)	4 12.5%	48 24.3%	51 10.3%	353 54.8%	210 76.3%	50 53.1%	718 23.1%
Daily intake (g)	21.6	23.6	39.8	48.7	42.6	44.6	
			Group II†				
No longer living	122	312	726	169	33	8.0	1370 44.1%
Alcohol consumed (*n*;%)	85 69.6%	197 63.1%	617 84.9%	17 10.0%	6 18.1%	+1 12.5%	923 67.3%
Daily intake (g)	75.8	79.4	69.6	28.3	24.2	25.3	
Total alcohol consumed (*n*;%)	89 57.7%	245 48.1%	668 54.7%	370 45.5%	216 70.1%	51 49.5%	1639 52.7%

*χ^2 (linear) = 216.44, d.f. = 1, $P = 0.0000$.
†$\chi^{(k-1)} = 160.29$, d.f. = 1, $P = 0.0000$.

high scorers. For those who died, low S–R scores clearly drank more than high S–R scorers. We again see a paradox, and again this finds an explanation in previous research that showed clearly that the effects of alcohol are dependent on personality factors; Grossarth-Maticek & Eysenck (1991a) found that alcohol consumption had a *negative valence* for health if drunk to drown one's sorrows, but not if drunk for pleasure, celebration, etc. This is an interesting feature common to smoking and drinking, showing that leaving out of account psychological factors may lead to serious misinterpretations of epidemiological data concerning the effects of cigarette and alcohol consumption. (The χ^2 results for our conclusions show $P < 0.001$ levels.)

SELF-REGULATION AND GROSSARTH-MATICEK TYPOLOGY

It is of interest to see to what extent the Grossarth-Maticek Typology (Grossarth-Maticek & Eysenck, 1990), with its six types, interacts with the self-regulation typology. It has often been objected that the Grossarth-Maticek methodology of assigning a person to one or other of the six types is faulty because: (1) it uses only a small portion of the available data, (2) it does not correct scores on one type by drawing on information regarding another type. Thus a Type 1 person with a high score on Type 4 might be expected to do better health-wise than a Type 1 person with a low Type 4 score. Profile scoring might be a better method of analysis than simply assigning a person to a given type just because he happened to score highest for that type, but from the beginning Grossarth-Maticek has used the simple typology concept, rather like Friedman and Rosenman used the Type A concept because to a medical audience this method of analysis might seem more natural and easier to follow. The fact that this simple typological approach has been very successful (Eysenck, 1991) does not mean that better methods should not be tried; it might be hoped that their use would improve predictive accuracy.

A sub-group of 3240 men and women was selected on a random basis and administered the Personality Stress Inventory (Grossarth-Maticek & Eysenck, 1990), in order to cross-validate the two inventories. Table 10 shows the major findings. Results are given separately for bad and for good self-regulation (scores of 1, 2 or 3 vs 4, 5 or 6), subdivided by subjects according to Type (1, 2, 3, 4, 5 or 6). For each of the 12 sub-divisions (2×6) are given the number and percentage of deaths from cancer, CHD (infarct) and other causes. Clearly SR is vitally important, as the percentage of mortality figures for the High and low S–R scores show. This of course merely mirrors the data in Figs 1 and 2. Within the low S–R group, clearly Type 1 has the highest cancer mortality, Type 4 the least, while for Type 2 CHD has the highest mortality, with all the other types roughly on a par. For

Table 10. Degree of self-regulation and six Grossarth-Maticek types as related to mortality

	Type 1	Type 2	Type 3	Type 4	Type 5	Type 6	Total
			Poor Self-regulation (1, 2 or 3 points)				
N	392	403	102	52	507	64	1520
Cancer	117	50	17	10	81	12	287
	29.8%	12.4%	16.6%	19.2%	15.9%	18.7%	18.8%
Infarct	51	119	19	10	69	10	278
	13.0%	29.5%	18.6%	19.2%	13.6%	15.8%	18.3%
Other causes of death	101	99	24	13	105	17	359
	25.7%	24.5%	23.5%	25.0%	20.7%	26.5%	23.6%
Average age (yr)	57.6	57.4	57.3	58.2	58.4	58.1	
Mean S–R score	2.4	2.3	2.5	3.0	2.1	2.4	
			Good Self-regulation (4, 5 or 6 points)				
N	260	204	351	477	358	70	1720
Cancer	4	4	3	2	3	+1	17
	1.5%	1.9%	0.8%	0.4%	0.8%	1.4%	1.0%
Infarct	4	5	7	2	2	1	21
	1.5%	2.4%	1.9%	0.4%	0.5%	1.4%	1.2%
Other causes of death	21	27	29	34	38	15	164
	8.0%	13.2%	8.2%	7.2%	10.6%	21.4%	9.5%
Average age (yr)	56.2	56.9	57.1	56.2	56.4	55.7	
Mean S–R score	3.8	3.9	4.1	4.7	3.8	3.9	
			Total Degree of Self-regulation				
N	652	607	453	529	865	134	3240
Cancer	121	54	20	12	84	13	304
	18.5%	8.9%	4.4%	2.2%	9.7%	9.7%	9.4%
Infarct	55	204	26	12	71	11	379
	8%4%	33.6%	5.7%	2.3%	8.2%	8.2%	11.7%
Other causes of death	122	126	53	47	143	32	523
	18.7%	20.8%	11.7%	8.9%	16.5%	23.9%	16.1%

'Other causes', there is little to choose between Types. For the good S–R scores, Type 4 does best overall, but the other Types have mortality too low to produce marked differences.

It is interesting to look at the ratios of good/bad SRI scores for each of the typologies. Going from 1 to 6, these are: 0.66; 0.51; 3.34; 9.17; 0.71; 1.09. Not unexpectedly, the 'healthy' Type 4 has much the highest ratio, followed by the fairly healthy Type 3; while the cancer-prone and CHD-prone Types 1 and 2 have much the lowest. It is apparent that the SRI measures much the same traits as does the Grossarth-Maticek Typology Type 4.

Analyses by generalized linear model shows the main effects (Typology and Self-regulation) as well as their interaction are all significant at the $P < 0.001$ level. One important consequence of these findings would seem to be that questionnaires using a *positive* wording are as useful, if not better, at indicating psychological disposition to good health, as questionnaires using a *negative* wording are in indicating psychological disposition to bad health. Most people are apparently more likely to respond truthfully to positive than to negative questions, although this point would have to be established by a specially designed experiment.

Physical risk factors for disease

To study the relationship of physical risk factors to mortality, a score was based on a specially designed questionnaire, based on known risk factors which could be obtained relatively easily. Table 11 gives the items involved and the points given for the various items. The scale has a minimum of 0 points (no positive factors, high risk), and a maximum of 24 points (many positive factors, low risk). The scale takes into account genetic factors, exercise, nutrition, alcohol, smoking and direct estimates of poor fitness—overweight, high blood pressure, high cholesterol, etc. Different numbers of points can be obtained for different items, thus blood pressure is more important than smoking or drinking. The various items were of course specified in considerable detail for the interviewers.

Table 12 shows the relationship between physical risk factor scores and (a) mortality and (b) SRI scores.

There is clearly a close relation between physical risk factors and mortality; the greater the number of positive factors, the greater the chance of survival, and the lower the risk of mortality. Conversely,

Table 11. Point scale for physical risk factors

	Points
1. A close member of the family (parents, grandparents) has reached an age of 75 yr. Add one point for each such family member. Points 0–6, respectively	0–6
2. Regular exercise	2
3. Daily activity in fresh air, irrespective of the weather	1
4. Healthy nourishment	2
5. Sufficient amount of fluid intake	1
6. Normal body weight	1
7. Little alcohol	1
8. Non-smoker	1
9. Normal blood pressure	2
10. Normal blood sugar	2
11. Normal total cholesterol	2
12. Low consumption of coffee, black tea and Coca-Cola®	1
13. No stimulant or depressant psychopharmaca.	1
14. Normal sensitivity for pain (not overly sensitive)	1

the smaller the number of positive factors, the greater the risk of mortality. Those with the most positive factors, i.e. 24 points, show a survival rate *seven times greater* than those with a score of 0 points. For those who died, probands with a score of 0 died 15 times more frequently than those with a score of 24. The relationship is significant by Mann–Whitney U-test, with $P < 0.00001$. SRI also independently predicted mortality, with $P < 0.00001$ by Mann-Whitney U-test. The regression of S–R on physical risk factors appears linear for the dead group but curvilinear for the living; only a replication can show whether this is an accidental finding, of no importance. But clearly the S–R scale measures causes of death largely *independent* of physical causes.

It will be obvious from Table 12 that physical risk factors, as expected, correlate separately with mortality. r (*bis*) between total mortality and physical risk factors is 0.36, $P < 0.001$. Using a Kruskal–Wallis ANOVA by ranks for the relationship between S–R and mortality, we obtain $H = 3520.83$, which with d.f. = 2 gives $P < 0.00001$. Carrying out the same type of analysis for physical risk factors, $H = 1118.26$, which with d.f. = 2 gives $P < 0.00001$. There is little correlation

Table 12. Mortality as related to physical risk factors and self-regulation

Positive physical factors	Still living			Mortality		
	n	%	S–R (%)	n	%	S–R (%)
0	20	0.6	4.8	315	13.9	3.1
1	21	0.6	4.9	206	9.1	3.0
2	34	1.0	4.7	170	7.5	2.8
3	47	1.4	4.6	153	6.7	3.9
4	96	2.8	4.3	104	4.6	3.1
5	78	2.3	3.9	107	4.7	3.3
6	103	3.0	3.6	101	4.4	3.3
7	124	3.6	3.7	162	7.1	3.4
8	113	3.3	3.5	103	4.5	3.5
9	272	7.9	3.8	102	4.4	3.3
10	271	7.9	3.6	100	4.4	3.4
11	294	8.5	3.7	84	3.7	3.4
12	231	6.7	3.8	75	3.3	3.3
13	186	5.4	3.6	62	2.7	3.5
14	144	4.2	3.9	51	2.2	3.6
15	169	4.9	3.7	45	1.9	3.4
16	124	3.6	3.6	37	1.6	3.3
17	116	3.4	3.8	40	1.6	3.1
18	127	3.7	3.9	35	1.5	2.9
19	131	3.8	3.4	49	2.2	2.7
20	155	4.5	3.9	51	2.2	2.5
21	163	4.7	4.0	42	1.8	2.6
22	143	4.2	4.1	31	1.4	2.1
23	136	4.0	4.5	28	1.2	2.3
24	144	4.1	4.9	21	0.9	2.4
Total	3422			2274		

Table 13. Self-regulation and genetic determinances in cancer of the breast (degree of self-regulation)

			Cancer of the breast			Healthy and living		
Group	*N*	S–R	*N*	%	S–R	*N*	%	S–R
0	544	3.6	1	0.2	3.1	316	58.1	4.6
1	349	3.5	9	2.5	3.0	205	58.7	4.7
2	138	3.6	9	6.5	2.9	64	46.4	4.9
3	54	3.9	14	25.9	3.1	21	38.9	5.0
	1085		33	3.0		606	55.6	

between S–R and physical risk factors, Spearman $\rho = -0.24$, which is significant statistically, but only accounts for less than 5% of common variance.

It is possible to pursue the search for physical risk factors a little further by looking more closely at genetic factors, implied by the first item in Table 11. This can be done by looking directly at parents and grandparents who died of the same disease as the proband. Of course this is only possible in large groups with high mortality, e.g. women with breast cancer. Table 13 shows the results for four groups of women who died of cancer of the breast.

One group had no first-degree relatives who also died of cancer of the breast, one group had one such relative, one group had two such relatives, and one group had three. There is a clear-cut regression: cancer of the breast increases with an increase in the number of relatives who died of such cancer, but there is no change in S–R, which clearly does not correlate with the genetic predisposition. Kruskal–Wallis ANOVA by rank gives $H = 58.5$, d.f. $= 2$ and $P < 0.00001$ for mortality and genetic predisposition. Breast cancer patients are clearly separated from healthy probands in respect to genetic predisposition. Looking at S–R by itself, this gives a $P < 0.00001$ for the comparison with still living probands. Clearly sufferers from cancer of the breast are strongly predisposed to develop this type of cancer by both genetic factors and by low self-regulation.

The causal nexus—an intervention study

Although clearly there are important correlations between S–R and mortality, correlations largely independent of and larger than those observed between mortality and physical risk factors, it would be dangerous to interpret these correlations as necessarily involving causality—arguing from *correlation* to *causality* is only too frequently done in epistemiology, particularly in relation to smoking (Eysenck, 1991). However, the hypothesis of a causal nexus can be given greater plausibility by intervention studies, i.e. by demonstrating that changing degree of S–R can change the risk of mortality. Previous studies have shown that *autonomy training* can change mortality risk very markedly (Grossarth-Maticek & Eysenck, 1991; Eysenck & Grossarth-Maticek, 1991), and an attempt to apply these methods in connection with the present study seemed worth-while.

The experimental and control groups used in this study are of course not included in the group that formed the samples discussed thus far. We chose 700 persons in 1974 who showed high physical risk factors (e.g. high blood pressure, high cholesterol, high cigarette and alcohol consumption, lack of exercise, etc.), as well as low degree of self-regulation (below 3, average 2.5 points). These 350 probands were divided into two groups on a chance basis, and one group was administered autonomy training, the other was left alone. The principles of autonomy training have been discussed elsewhere (Grossarth-Maticek & Eysenck, 1991a). Beginning with the use of individual and bibliographic therapy, we followed up with a course of group therapy, involving altogether about 30 hr per person. Six months after completion of the therapeutic intervention probands were again administered the SRI. (The first occasion of administration was one month before the beginning of therapy.) As a result of the changes from first to second administration, probands were divided into four groups. Group I showed an *improvement* in SRI scores, but with an average score still below 3.5. Group II showed a markedly better degree of improvement, with values well below 3.5 the first time, but a score above 3.5 the second time. (On the basis of the results shown in Figs 1 and 2, 3.5 seems to have been a good choice for making this diagnosis.)

Group III includes those probands whose scores were worse on the second occasion, and Group

IV showed a marked deterioration. Thus Group I showed an improvement of 2 points or less, and a final score below 3.5. Group II showed an improvement of 2–5 points, and a final score above 3.5. Group III showed a deterioration of 1 point or less, and Group IV one of more than 1 point. There was also a small Group V where there was no change. The average age of the treatment group was 54.6 yr, of the control group 54.8 yr, an insignificant difference. Mortality was ascertained in 1993, giving a follow-up period of 19 yr. Nine probands in each group could not be located on follow-up, thus reducing the total number analysed to $2 \times 341 = 682$. Table 14 shows the results.

Results show the following major findings.

(1) Regardless of therapy or control, mortality in the five groups is similar, being highest in Group IV, lowest in Group II (markedly worse and markedly better), with Group III and Group I showing intermediate degree of mortality. (The numbers in group 5 are too small to be very meaningful.) In other words, improvement in S–R, whether achieved spontaneously or as the result of autonomy training, is significantly related (negatively) to mortality; those who improved are less likely to die than those who got worse in degree of S–R.

(2) Overall, the group with autonomy training shows a significantly reduced mortality compared with the control group as regards mortality from all causes, as well as a higher percentage of probands who are healthy and live without any chronic disease—61.7% compared with 37.6% in the therapy and control group, respectively. This effect is clearly due to the fact that markedly improved probands are nearly eight times as frequent in the therapy group as in the control group. In the other groups the advantage of the proband who underwent therapy is small, although present even in those where S–R scores get worse.

It is interesting to note the changes in *physical function* which accompany any changes in S–R (Table 15). Measures are reported for blood pressure, cholesterol (total), cigarettes per diem, alcohol g/day, bodyweight, lack of exercise, and unhealthy nutrition. In each case, the *first* measure was taken before beginning therapy, the second after one year, i.e. six months after the second measurement of S–R. The results show that in the group with *improved* S–R, all the physical risk factors improve, while in the group with worsening S–R scores there is also a worsening of all the physical variables. The conclusion suggested by these data must be that (1) improvement in S–R is a systematic process which results not only in improvement in the physical sphere. (2) When looking for an improvement in the physical risk factors it would be advisable to try and improve the psychological risk factors, through improvement in S–R.

SUMMARY AND CONCLUSIONS

The results of this large-scale prospective study suggest that psychological factors incorporated in the concept of the *healthy personality* have a profound influence on disease and mortality. *Mens sana in corpore sano* was the health slogan of the ancients; it seems that this combination constitutes a strong correlation between body and mind, and that changes in the psychological sphere produce changes in the physical sphere also. That of course is the main assertion of psychosomatic theory, and this study adds to the large literature supporting it. Psychological risk factors exert a largely independent influence on mortality, and can be influenced, modulated and changed decisively by autonomy training, a kind of behaviour therapy stressing management technique.

The personality of probands incorporates their sensitivity to stress, their coping behaviours, and their general outlook on life; self-regulation is in many ways the opposite to neuroticism, constituting a flexible autonomous, functional way of solving problems and getting over difficulties, while neuroticism is linked with inappropriate emotional responses, rigidity, and inability to cope with stress, leading to feelings of helplessness, hopelessness and finally depression. [In the case of cancer, we are dealing with a tendency to *suppression of emotion* and *denial*; hence for the cancer-prone person low neuroticism scores may be predictive of cancer (Kissen & Eysenck, 1962). This denial factor may cause confusion; thus Kreitler and Kreitler (1991) found health oriented people scoring *low* on negative emotions, like anxiety and fear, but *high* on neuroticism.] It would seem that preventive medicine should pay attention to psychological factors that have been shown to be vitally important to survival, as well as modifiable by autonomy training which, particularly when administered in the

Table 14. The preventive effects of autonomy training on mortality

Therapy group (with autonomy training)							Control group					
Ca	CHD	Other causes of death	Alive	Alive, ill	*N*		*N*	Ca	CHD	Other causes of death	Alive	Alive, ill
5	10	10	44	12	81	Improving	196	17	29	30	88	32
6.1%	12.3%	12.3%	54.3%	14.8%				8.6%	14.7%	15,3%	44.8%	16.3%
8	9	10	157	15	199	Markedly Improved	25	1	1	2	19	2
4.0%	4.5%	5.0%	78.8%	7.5%				4.0%	4.0%	8.0%	76.0%	8.0%
3	7	11	6	8	35	Worse	80	7	16	21	15	21
8.5%	20.0%	31.4%	17.1%	22.8%				8.7%	20.0%	26.3%	18.7%	26.3%
3	5	9	1	1	19	Markedly worse	21	4	8	7	1	1
15.7%	26.3%	47.3%	5.2%	5.2%				19.0%	38.0%	33.3%	4.7%	4.7%
1	1	2	+2	1	7	No change	19	2	3	4	5	5
14.2%	14.2%	28.5%	28.5%	14.2%				10.5%	15.7%	21.0%	26.3%	26.3%
20	32	42	210	37	341	Total	341	31	57	64	128	61
5.8%	9.4%	12.3%	61.7%	10.8%				9.1%	16.7%	18.8%	37.6%	17.9%
					9	Not investigated	9					
					54.6	Mean age (yr)	54.8					

Table 15. Physiological and behavioural effects of autonomy training

Therapy group								Control group							
Blood pressure	Cholesterol	Cigarettes per day	Alcohol per g	Body weight	Lack of exercise	Healthy nutrition	*n* (= 341)	*n* (= 341)	Blood pressure	Cholesterol	Cigarettes per day	Alcohol per g	Body weight	Lack of exercise	Healthy nutrition	
							Group 1									
163/119	276	28.9	86.3	+17	49	49	81	196	162/120	281	30.1	82.6	+20	88	18	1
152/96	259	26.3	70.1	+13	13	49			159/110	264	25.9	75.3	+16	59	39	2
							Group 2									
162/120	291	31.6	90.6	+15	102	17	199	25	163/120	290	28.6	88.3	+14	13	3	1
148/90	255	23.6	42.3	+7	11	113			149/90	278	25.1	50.2	+8	6	16	2
							Group 3									
163/117	287	24.6	90.4	+14	20	17	35	80	168/119	276	23.6	86.2	+13	35	42	1
165/118	289	27.7	95.3	+15	26	18		169/123	285	28.1	89.2	+12	50	39	2	
							Group 4									
161/112	288	20.8	85.3	+13	10	13	19	21	170/114	267	25.3	84.6	+10	12	15	1
169/115	305	30.6	97.2	+16	11	14			170/114	267	25.3	84.6	+10	12	15	2
							Group 5									
164/99	273	25.3	70.7	+12	2	3	7	19	162/98	276	26.7	68.5	+13	6	5	1
163/110	273	26.1	69.4	+9	2	3			162/99	278	26.2	68.7	+14	5	4	2

form of group therapy, is very cost effective. Sole attention to smoking and other similar physical factors is counter-productive when it leads to the neglect of important psychological risk factors.

Recognition of the psychological involvement in physical disease has been hindered by philosophical problems introduced by Descartes and the wholly erroneous notion of *body* and *mind* as totally separate substances. There is no evidence for, and much evidence against, this view, and just as physicists had to adopt the fundamental notion of a space-time continuum, so psychologists and physicians will have to return to the Hippocratic notion of a body-mind continuum. As Sir William Osler (1906), the father of English medicine, used to say: "It is very often much more important what person has the disease than what disease the person has." (pp. 258–259.)

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