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An Empirical Study of the Diathesis-Stress Theory of Disease

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One thousand, one hundred and twenty-one Jewish concentration camp survivors were compared with 367 Jewish controls who had not been in a concentration camp, and had not lost any family members in such a camp. Of interest was the mortality of camp and comparison groups, on the hypothesis that the stress of being in a camp would adversely affect inmates. It was found that former camp inmates were over twice as likely to die of cancer, coronary heart disease, or other causes as the comparison subjects of similar age and sex composition, and that severity of stress was correlated with mortality in the expected direction. Diathesis, determined by means of a special interviewer-administrated questionnaire, was found to interact synergistically with stress in producing high mortality.

KEY WORDS: stress; cancer; coronary heart disease; diathesis.

INTRODUCTION

It is well-known that stress, particularly under conditions of real or perceived lack of control, has serious biological consequences (Henry, 1992) which may lead to such diseases as cancer and coronary heart disease (CHD); the literature on stress has been surveyed by Cooper (1983, 1984), Cooper and Watson (1991), Lazarus and Folkman (1984), McGuigan, Sime, and Wallace (1984), Norfolk (1977), Perrewe (1991), and many others. The specific con-

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nection between stress, personality, and disease has been summarized by Eysenck (1991) and Temoshok and Dreher (1992). The stress level investigated has ranged from ordinary, everyday hassles to divorce and death of loved ones. Social studies in which stresses are accidental and unexplained cannot of course compare in elegance or methodological rigor with experimental designs, but as the imposition of serious stress on an experimental basis would clearly be impossible on ethical grounds, we are left with strictly observational studies where rigor is imposed by the statistical treatment.

In this study, we have looked at the long-term health effects of a type of stress probably more severe than any yet studied in this connection, namely being sent to a Hitlerian concentration camp, living there for a lengthy period of time, and having members of one's family killed. There are many written testimonies to the incredible sufferings of concentration camp inmates, and there can be no doubt about the high stress level imposed by starvation, torture, and the constant threat of death, combined with fear for the fate of loved ones. Theory would predict that such a high degree of stress would produce a high level of cancer and coronary heart disease, and our study was designed to test this hypothesis.

A proper design for such a study requires some acceptable model into which we can incorporate the concept of stress, taking into account the fact that the concept has been operationally defined in many different ways, and is extremely "fuzzy." It is widely accepted that neurotic symptoms can best be accounted for in terms of a diathesis-stress theory (Eysenck & Martin, 1987). In other words, in a meeting between an organism and a particular stress situation, both the organism and the situation make a contribution to the outcome, i.e., the occurrence or non-occurrence of a neurotic symptom, and the particular type of symptom that may result. Genetic factors are the most obvious parts of the diathesis; thus Torgersen (1979) has shown a strong genetic component in the origin of phobias, and even the *specific* phobia a patient may develop. Such studies of course require specially selected populations (e.g., twins), and are comparatively rare; usually they deal with general personality traits of a pathological nature, e.g., neuroticism (Eaves, Eysenck, & Martin, 1989).

More usually, such intermediary factors (neuroticism, depression, anxiety) are used as aspects of a person's diathesis, although of course they might equally well appear at the other end of the formula, as symptoms, the formula being:

$$\underset{\text{(diathesis)}}{D} \times \underset{\text{(stress)}}{S} = \underset{\text{(malady)}}{M}$$

The trait–state dichotomy is relevant here; neuroticism is a trait, and forms part of the diathesis; depression and anxiety may sometimes be regarded as symptoms or states, and then form part of the malady. The precise nature of the interaction between D and S ("$\times$," "$+$," or whatever) is seldom investigated in any systematic fashion.

A similar algorithm has been postulated for psychosomatic diseases, and even for such disorders as cancer and coronary heart disease, which are probably not usually thought of as psychosomatic. However, as Sir William Osler, the father of British medicine, said in 1911: "It is often more important what person has the disease, than what disease a person has." Certainly much recent work has linked cancer and coronary heart disease with psychosocial factors which, interacting with stress, constitute risk factors for these diseases (as well as for many psychosomatic disorders). The development of this research has been recorded by Eysenck (1991) and Temoshok and Dreher (1992). This suggests that the diathesis-stress theory may have a wider usefulness than originally intended.

It has formed the background of a series of investigations attempting to study the influence of psychosocial risk factors on cancer and coronary heart disease, carried out by Grossarth-Maticek (1979, 1986, 1989), summarized by Eysenck (1991). He and others (Bahnson, 1969, 1976, 1980; Bahnson & Bahnson, 1966; Kissen, 1964a,b; Kissen & Eysenck, 1962; Le Shan, 1961, 1977; Schmale & Iker, 1971) used traditional theories linking cancer with suppression of emotional expression and failure to cope properly with stress, leading to feelings of hopelessness, and helplessness, and ultimately depression. Coronary heart disease was linked by Friedman and Rosenman (1959) with Type A personality (Price, 1982), and more specifically with reactions of anger, hostility, and aggression to stress (Eysenck, 1990; Friedman & Booth-Kewley, 1987a,b).

The ambiguity of such concepts as "depression" in such a scheme as that indicated in our formula becomes clear when we look at a paper by Metalsky and Joiner (1992). In their theory, *hopelessness* "is viewed as a proximal sufficient cause of the symptoms of the depressive subtype proposed in the theory (hopelessness depression)" (p. 667). Hopelessness in turn is viewed as a common pathway for the *causes* of depression. The *diathesis*, in this formulation, consists of (1) attributional style—attribution of negative life events to stable and global causes, (2) negative inferences about the self, and (3) dire consequences of negative life events. These three cognitive diatheses are assumed to interact with negative life events and contribute to the onset of depressive symptoms. In terms of our formula: Cognitive diatheses $\times$ negative life events = hopelessness depression. Depression then causally affects the immune system, destabilizing it, possibly through such agencies as cortisol incrementation (Herbert & Cohen, 1993).

For the Grossarth-Maticek formulation, depression turns up on the other side of the equation: Hopelessness depression $\times$ stress = cancer! In other words, the *consequences* of the diathesis-stress interaction in one study can turn up in another as the diathesis itself. The confusion may arise in fact because psychologists continue to talk about *causes* when all that is demonstrated is *correlations*—and usually quite low ones. A more inclusive path analysis might go as follows: Some people are genetically predisposed, or acquire through life events the habit of using the cognitive diathesis described by Metalsky and Joiner (1992) when stressed. These reactions lead to depression of a

certain type, which in turn has been shown to be associated with an increase in cortisol level, which in turn leads to immunodepression. This immunodepression allows budding cancers to grow, thus leading to death from cancer. The theory briefly outlined here has been developed in greater detail elsewhere (Eysenck, 1991), where supportive evidence is cited; here let us only note that the actual application of the diathesis-stress theory entails difficulties in deciding just what constitutes the diathesis, whatever the malady.

It is part of the Grossarth-Maticek theory that the psychosocial diathesis predisposing a person to succumb to disease when stressed is a certain personality type which tends to react to stress by feelings of hopelessness, leading to depression (Type 1; cancer-prone), or by feelings of anger, hostility, and aggression (Type 2; heart disease-prone). Both types of reaction suggest *inefficient coping mechanisms*, such as *emotional* and *avoidance* coping (Roger, Jarvis, & Najarian, 1993), as opposed to rational and detachment coping. (Rational and detachment coping correlate .49; emotional and avoidance coping .33. The two sets correlate negatively with each other, suggesting a superfactor of appropriate and inappropriate coping.) Inappropriate coping, in the Grossarth-Maticek scheme, is part of the diathesis leading to depression, through the failure of such mechanisms.

In accordance with these considerations, Grossarth-Maticek has devised questionnaires describing several types, in particular, the cancer-prone type (Type 1) the heart disease-prone type (Type 2), as well as the *healthy* type (Type 4), which uses appropriate coping mechanisms (rational and detachment). Types 1 and 2 correlate negatively with Type 4; they also correlate *positively* with neuroticism, while Type 4 correlates *negatively* with *neuroticism* (Schmitz, 1992). This agrees well with Bolger and Schilling's (1992) finding that *neuroticism* increased reactivity to stressors, and that this reactivity accounted for twice as much of the distress difference as exposure to stressors. "These results suggest that reactions within stressful situations are more important than situation selection in explaining how neuroticism leads to distress in daily life" (p. 355).

Support for such a model comes from a recent study by Jerusalem (1993), using a longitudinal design in which stress was constituted by adaptation processes of East German migrants to West Germany. Within this longitudinal design, self-efficacy, emotional coping tendencies, and subjective illness as criteria were assessed a month after appraisal of employment status and housing conditions had been used as environmental factors. Personal resources and environmental constraints turned out to be strong predictors of stress appraisals which in turn had a significant impact on coping and health.

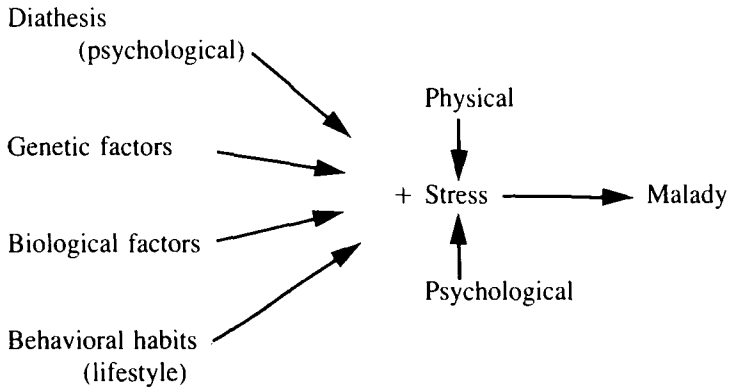
The Grossarth-Maticek system also posits another type of less interest here. Type 3 is essentially somewhat hysterical in behavior, but not necessarily linked with disease. The types are discussed in more detail elsewhere (Grossarth-Maticek, Eysenck, & Vetter, 1988), where the questionnaire used here is given in full. The results of using this and an earlier questionnaire are discussed

elsewhere (Eysenck, 1991, 1993; Grossarth-Maticek & Eysenck, 1990). It was ascertained in three prospective studies, using interviewer-administered questionnaires on apparently healthy probands in later middle-age, and following them up for periods of 15 years, that (1) Type 1 probands died predominantly of cancer, (2) Type 2 probands of coronary heart disease, while (3) probands of Types 3 and 4 had low mortality (Eysenck, 1993).

Other risk factors than psychosocial type were also studied, such as smoking, drinking, cholesterol level, blood pressure, and blood sugar. It was found that these factors were less predictive than personality type. This raised another question, namely that of the *mode of interaction* between risk factors for disease. Grossarth-Maticek (1980) found in an early study that the relationship was *synergistic*, a finding supported in later work (Grossarth-Maticek *et al.*, 1988; Eysenck, 1988, 1991; Eysenck, Grossarth-Maticek, & Everitt, 1991). This is a very important finding as the effect of *single risk factors* tended to be quite small, with the *combination* of two or more risk factors raising mortality to a much greater extent than would be done by the simple *addition* of individual effects.

It is difficult to allocate a proper place in this scheme to risk factors such as smoking, cholesterol level, or blood pressure. Genetic predisposition of course is easy; it belongs in the *diathesis* camp. But smoking, to take this as an example, does not fit in at all easily. It might be regarded as a stress factor because it might be said to reduce the efficiency of the lungs to resist cancerous development. But smoking is a habit maintained by genetic factors (Eysenck, 1980); does that not align it with the diathesis? Smoking also *reduces* tension (Eysenck, 1980, 1991); in that sense it *reduces* stress. A trifle more removed is the apparent fact that media information about smoking causing disease itself produces stress and mortality (Grossarth-Maticek & Eysenck, 1989). Clearly smoking as a risk factor does not fit into the diathesis-stress scheme at all well, and neither do most of the physical risk factors mentioned.

Perhaps an extended scheme is necessary, along these lines:



Even this is clearly too schematic; genetic factors may act directly, e.g., on the immune system or on the biological factors producing sclerosis, or it may act on psychological factors, e.g., extraversion, leading to a certain kind of lifestyle, involving smoking, drinking, promiscuity, etc. (Eysenck, 1976). It may also determine in part the psychological diathesis; Types 1 and 2 are related to neuroticism, which has a strong genetic basis (Eaves, Eysenck, & Martin, 1989). Furthermore, there is some interaction between stress and diathesis; what constitutes a stress depends to a large extent on diathesis factors. Thus, for an introvert, socializing would be a stress, but for an extravert it would be a positive reinforcement! Some factors *relieve* stress, such as sport activity, and act as negative risk factors for cancer and heart disease (Grossarth-Maticek, Eysenck, Uhlenbruck *et al.*, 1990). How are they to be accommodated? Could they be part of the diathesis? Often they are affected by stress; a person may stop playing tennis because of work stress, or take it up as a consequence of psychological advice to reduce stress. Clearly the model is insufficiently wide to take into account all relevant types of factors. Sometimes the stress effects of an activity, e.g., drinking, may be a function of the diathesis itself (Grossarth-Maticek & Eysenck, 1991); drinking to drown one's sorrows increases mortality, drinking for fun, or to celebrate, has the opposite effect.

Recent years have brought about a much more sophisticated approach to the understanding of the complexities of the diathesis-stress model, particularly the interaction of genetics and environment in this connection (Rende & Plomin, 1992). Kendler and Eaves (1986) have treated the diathesis-stress model as a form of genotype-environment interaction, and Gottesman (1991) and Dalgleish and Watts (1990) have used such models to elaborate etiological theories of schizophrenia and depression. Diathesis of course may include positive as well as negative factors, thus coping behaviors have been shown to evince genetic influence (Kendler, Kessler, Heath, Neale, & Eaves, 1991). Apparently environmental factors, such as life events, particularly when controllable, show substantial genetic influence (Plomin *et al.*, 1990). It is clear that the notion of genetic diathesis-environmental stress is grossly oversimplified, and requires considerable amplification.

In the present study, we are concerned with a very clear stress (incarceration in a Hitlerian concentration camp) and a very clear diathesis (having a high score on the Grossarth-Maticek inventory of Type 1 or 2, as opposed to Type 3 or 4. Most experimental studies have in the past used quite mild stresses, e.g., examination stress, or having to give a public speech, or undergoing the "cold pressor" test; this is of course inevitable because of ethical considerations—we cannot inflict life-type stresses on our subjects. It also means, of course, that a proper experiment is impossible because we cannot allocate subjects to treatment; we can only compare Jewish men and women who did undergo camp treatment and survived, with Jewish control subjects who did not undergo such

treatment, for whatever reason. At best we can match groups on such variables as age and sex, but of course that cannot guarantee equivalence on other, possibly more important factors. Hence, inevitably this study will be inferior methodologically to typical experimental studies, but superior to them because there can be no doubt about the severity of the stresses involved. If both types of study agree overall on results, we may conclude that the respective weaknesses may cancel out.

It might be argued that the typological assignment to high or low diathesis groups cannot be regarded as a pure measure. Answers indicative of poor coping behavior have usually been based on experience of dealing with stress; often subjects with high scores on Type 1 and 2 scales have experienced identical situations as stressful which were not so experienced by Type 4 individuals. To have to address an audience may be a stress for someone high on neuroticism, but a rewarding experience for someone low on neuroticism. It is not at all easy to make absolute distinctions, but bearing this caveat in mind we will in the rest of the paper contrast *D* (diathesis) with *S* (stress), in the sense outlined, and also look at their mode of interaction.

This rather extensive review of models and common usage of terms like stress, personality, and diathesis seemed desirable because they are often used in a very fuzzy way, and every particular type of use can be criticized with impunity. The diathesis-stress model and the risk-factor model are difficult to combine, and the suggested inclusive model is merely a first suggestion, suffering the kinds of difficulty arising from it which we have outlined. We shall use the terms diathesis and stress in a strictly operational manner, as described; we do not wish to impose a similar usage on others working in this field who might prefer a different model.

METHODOLOGY

Our particular hypotheses were as follows:

Stress.

(1) Duration of time spent in the camp would be a predictor of disease.
(2) The younger the proband at the time of incarceration, the greater would be the probability of subsequent disease.
(3) The more members of a proband's family had been killed in the camp, the greater would be the probability of subsequent disease.

Diathesis. Diathesis was established in terms of high scores for Type 1 and for Type 2, using the 4-type questionnaire already mentioned (Grossarth-Maticek *et al.*, 1988). (A person was rated as "stressed" if his or her score on

the Type 1 or Type 2 scale was higher than on either the Type 3 or Type 4 scale.)

(1) The greater the emotional vulnerability of the proband, the greater would be the probability of subsequent disease.
(2) Diathesis and stress would interact synergistically.

Our methodology was geared to testing these hypotheses as far as that was possible in a non-experimental study.

The study originated with a quite different enquiry directed to the discovery of a predictive relationship between personality, stress, and disease (Eysenck, 1991); during this study 12,362 people in the small German town of Heidelberg, aged between 32 and 66, were asked (in addition to all the questions relevant to the main purpose of the inquiry) whether they knew of any Jews in Germany, Austria, Switzerland, or Holland who had been inmates of a concentration camp and were still alive. Respondents were also asked if they knew of any suitable subjects; these too were approached. The refusal rate was very low, being just over 4%. Jews who had survived the Holocaust and had not been inmates of a concentration camp were contacted in a similar way. The total sample was made up of 1121 camp victims, and 367 who had not been camp victims.

On the basis of the interview, two groups were formed, namely an "extreme stress" group of 132 persons, and a "less extreme stress" group, also made up of 132 persons. These will be referred to as group A (extreme) and B (less extreme). The basis of allocating a person to group A was: (1) stay in concentration camp of 4 years or more, (2) more than four close members of the family (father, mother, grandfather, grandmother, siblings, aunt, uncle) murdered in the camp, (3) proband was less than 15 years of age at time of entering the camp, and (4) proband from 1945–1975 suffers chronically under severe psychological stress—inhibition, excitement, brooding, self-reproaches, guilt feelings for having survived, ideas of persecution, traumatic anxiety state. For group B, the same conditions obtained, but in weakened forms. Thus, stay in the camp was less than 4 years, between 1 and 3 family members had been lost in the camp, proband was older than 18 years at time of entering the camp, and traumatic memories were less serious and better assimilated, and successful new activities and positive reinforcements rendered the traumatic experiences less potent.

A third (comparison) group, group C, was formed of 132 Jews who had not been in a concentration camp, and had not lost any family members in such a camp. Members of group C were matched in age, sex, and profession (as near as possible) with the members of group A.

Next we considered a group (group D) of 644 persons, the members of which showed 1–3 of the 4 factors considered. This leaves a final remainder of 213 persons (group E) who did not fit into any of the groups from A to D. Of the total number contacted we failed to obtain evidence of mortality in 101 cases, in spite of repeated attempts.

We added another group (group F) of 213 persons who had not been in a camp, and who had not lost any members of their family in a camp. This group differs from group C by not being matched with the members of group A. In addition to these 213 non-camp probands, there were 22 individuals who had not been in a concentration camp, but who had suffered very bad treatment at the hands of the Nazis which might be considered almost equivalent to being in a camp. In our overall comparison (Table 5) we decided not to leave out this group, but count them as part of the no-camp group; this is the most conservative way of dealing with the problem, as it reduced the difference between camp and no-camp groups, if anything. (In actual fact, the difference made by their inclusion is minute, and does not alter any of the significance levels.) We thus have a total of 1589 persons included in the study of whom 1488 could be analyzed for mortality data.

Information was collected, and interviews carried out, by 43 specially trained students. Each interviewer contacted between 35 and 40 persons. The small number of losses from the study was made possible because the people interviewed in 1975 did not only give their address, but were also asked to give 3–5 addresses of relatives or friends or organizations where they could be traced in case they moved or died. This is an important method in social research for carrying out follow-up studies.

RESULTS

Table 1 compares groups A, B, and C. Quite clearly, group C (the control group) has the least mortality from cancer, infarct, or other causes of death, as

Table 1. Comparison of Groups A, B, and C

Group	*N*	Cancer	Heart disease	Other causes of death	Chronic illness	Health	Mean age (1992)
A: High degree of stress	132	48 (36.4%)	37 (28.0%)	27 (20.4%)	14 (10.6%)	6 (4.5%)	66.5
B: Medium degree of stress	132	16 (12.1%)	15 (11.4%)	14 (10.6%)	44 (33.3%)	43 (32.6%)	72.6
C: Comparison group	132	4 (3.0%)	4 (3.0%)	6 (4.5%)	35 (26.5%)	83 (62.8%)	67.5

Table 2. Activities and Risk Factors Injurious to Health

	Group A	Group B	Group C	Significance (chi-square)
Smoking	41	60	67	<.001
Overweight	14	49	38	<.001
Alcohol	10	16	19	NS
Pills	5	17	2	<.01[a]
Lack of exercise	49	30	12	<.001
High blood pressure	86	58	14	<.001
Diabetes	54	30	1	<.01[a]
Faulty diet	24	27	26	NS

[a]Because of small numbers of cases, the $p < .001$ significance values obtained in each instance are reported more conservatively at the $p < .01$ level.

well as having the largest proportion of healthy survivors. Ratios between groups vary widely, but always give the sequence A<B<C, where the "smaller than" sign means "less likely to survive." These data support our general hypothesis that the stress of concentration camp incarceration affects later health, and that the greater the stress, the greater the effect.

Statistical test by chi-square shows the following: The overall chi-square is 173,82, which with 8 *df* is significant at the $p < .001$ level. For cancer (435.75), infarct (30.32), other causes (225.47), chronic illness (488.23), and healthy (2965.41) the probability levels are all below .001. If the cancer and infarct figures are corrected for small numbers, p levels increase to <.01.

Table 2 shows the results of questioning members of the three groups about a number of risk factors for cancer and CHD. The major differences are that group A smokes less than the others, is less overweight, drinks less alcohol, shows the greatest lack of movement, has the highest blood pressure, and has more diabetes. It is difficult to postulate the direction of the causal arrow, and the data are presented for the sake of completeness, and not in order to support any hypothesis.

Table 3 shows the data for group D. Factors 1, 2, 3, and 4 are those given in the methodology section (longer stay than 4 years, more than four members of family murdered, 14 or less at entry, strong evidence of stress). The effectiveness of factors 1, 2, 3, and 4 is approximately equal, and each produces greater mortality than is apparent in group C (Table 1). Physical and psychological factors appear to be roughly equally important, although the possibility of synergistic action cannot be ruled out.

Table 4 shows the comparison of the control group E with a matched sample of camp inmates from group D. Again the results show a much greater risk of cancer and CHD on the part of former camp inmates, the risk ratio being 2.65 for mortality. This comparison is a successful replication of the data

Table 3. Importance of Various Factors for Mortality in Group D

Factor	*N*	Cancer	Heart disease	Other causes of death	Chronic illness	Healthy	Mean age (1992)	Significance by chi^2
1	82	7 (8.5%)	6 (7.3%)	10 (12.1%)	20 (24.3%)	39 (47.5%)	69.3	.001
2	60	5 (8.3%)	4 (6.6%)	6 (10.0%)	18 (30.0%)	27 (45.0%)	68.2	.001
3	71	3 (4.2%)	4 (5.6%)	9 (12.6%)	24 (33.8%)	31 (43.6%)	65.8	.001
4	92	9 (9.7%)	7 (7.6%)	13 (14.1%)	42 (45.6%)	21 (22.8%)	68.5	.001
Two physical factors	82	11 (13.4%)	8 (9.7%)	12 (14.6%)	21 (25.6%)	30 (36.5%)	66.7	.001
Three physical factors	89	15 (16.8%)	13 (14.6%)	17 (20.0%)	23 (25.8%)	21 (23.5%)	65.6	NS
One physical plus one psychological factor	83	13 (15.6%)	9 (10.8%)	10 (12.1%)	34 (40.9%)	17 (20.4%)	66.8	.001
Two physical plus one psychological factor	85	17 (20.0%)	14 (16.4%)	19 (22.3%)	30 (35.2%)	5 (5.8%)	67.8	.001
Totals	644	80 (12.4%)	65 (10.0%)	96 (14.9%)	212 (32.9%)	191 (29.6%)		

Table 4. Comparison of Groups D and E

	N	Cancer	Heart disease	Other causes of death	Chronic illness	Health	Mean age (1992)
Concentration camp group	213	18 (8.4%)	15 (7.0%)	20 (9.3%)	109 (51.7%)	51 (23.9%)	69.8
Comparison group	213	5 (2.3%)	6 (2.8%)	9 (4.2%)	51 (23.9%)	142 (66.6%)	70.5
Significance level		.01[a]	.05	.05	.001	.001	

[a] $p < .05$ if adjusted for small sample size.

shown in Table 1. Overall $p < .001$, and all the individual columns are significant, although if correction is made for a small sample size, cancer, and other causes both decline to $p < .05$, and infarct becomes insignificant.

We can take our two control groups together (C + F) and compare them with the combined concentration camp group, i.e., A + B + D + F. The results are shown in Table 5. The risk ratios are of course very similar, i.e., in the neighborhood of 2; in other words, having been a camp inmate increases the chances of dying from cancer, a heart infarct, or some other cause, more than

Table 5. Comparison of Combined Comparison and Concentration Camp Groups

	Cancer	Heart disease	Other causes of death	Chronic illness	Healthy
Concentration camp ($N = 1121$)	159 (14.2%)	132 (11.9%)	157 (14.0%)	380 (33.9%)	293 (26.1%)
Comparison group ($N = 367$)	22 (6.3%)	19 (5.5%)	15 (4.3%)	86 (24.9%)	225 (65.2%)
Significance level by chi^2 ($p<$)	.001	.001	.001	.001	.01

twofold. It reduces the chances of healthy living from 65.2% to 26.0%. These are very marked effects which indicate the stressful nature of the experience. Overall chi-square is 159.17, for 4 *df* ($p < .001$).

So far, our data have shown that *degree of stress* predicts mortality and incidence with some success. We would expect that personal diathesis (stress-proneness) would interact with stress to produce mortality/ill-health; there is considerable evidence for such stress-proneness (Eysenck, 1991; Temoshok & Dreher, 1992).

The overall chi-square, with 12 *df*, is 789.13, which gives a $p < .001$ value, leaving no doubt about the statistical significance of the observed differences. Comparing the concentration camp group with group C, overall chi-square is 640.88 for the concentration camp group and 44.90 for group C; both are at the $p < .001$ level, but the relationship is clearly much closer for the

Table 6. The Effect of Stress on Mortality in Comparison and Concentration Camp Groups

	Cancer	Heart	Other causes	Chronic illness	Healthy	Total
N	9 (5.7%)	8 (25.0%)	13 (08.3%)	241 (63.4%)	289 (73.8%)	560 (50.0%)
Concentration camp group with stress	159 (14.1%)	137 (11.7%)	157 (14.0%)	380 (34.8%)	392 (34.9%)	(100%)
Concentration camp group without stress	150 (94.4%)	124 (94.0%)	144 (91.7%)	139 (36.6%)	4 (1.0%)	561 (51.0%)
N	22 (6.0%)	19 (5.2%)	15 (4.1%)	86 (23.4%)	225 (61.3%)	267 (100%)
Comparison group with stress	17 (77.3%)	16 (84.2%)	5 (33.3%)	6 (170.9%)	42 (18.7%)	141 (53.0%)
Comparison group without stress	5 (22.7%)	3 (15.8%)	10 (66.6%)	25 (29.1%)	83 (36.9%)	126 (47.0%)
$p<$	.001	.001	.001	.01	.001	

concentration camp group. Comparing for stress across both groups, chi-square is 201.59 (4 *df*) giving $p < .001$, i.e., there is a greater stress effect in the concentration camp group. Comparing for no stress across both groups, chi-square is 312.52 (4 *df*) giving $p < .001$; i.e., concentration camp effects are significant even in the non-stressed group, but they are less apparent than in the stressed group.

We may finally look at specific results across both groups, i.e., concentration camp vs. group C. The chi-square p values are given in the bottom line. All the values are significant, showing that the concentration camp experience has a very marked effect on mortality from cancer, heart disease, other causes, and chronic disease, and (lack of) health, regardless of stress. On the whole, then, the analysis shows strong effects from both diathesis (stress-proneness measured by questionnaire) and external stress through being a concentration camp inmate.

We can arrange the data for total mortality in Table 7. This table suggests some degree of synergistic diathesis-stress interaction. Concentration camp experience is a strong risk factor for mortality, particularly in combination with our measure of diathesis. Diathesis is a much less powerful predictor of mortality, but is more important in combination with stress. The effect of stress alone is 36%, that of diathesis alone is 13%. Additively this would amount to 49%, but the combined effect is 61%, suggesting a synergistic effect of 12% over and above the additive effect. Of course it must be remembered that the estimate of diathesis was only obtained *after* the stress period was long over (something like 30 years before the beginning of our study); hence, many original camp victims had died in the camp, others had died between leaving the camp and the beginning of our study. We are clearly dealing with a highly selected population. Also the camp experience preceded the diathesis ascertainment, thus possibly affecting it in unknown ways. For all these reasons, the data deserve to be treated with considerable caution; the study, like most epidemiological ones, is far from a perfect experiment. Nevertheless, it seemed useful to present the data and allow readers to interpret them as far as possible. It is clear that within their obvious limits, they tend to support our hypotheses.

Table 7. Mortality as a Function of Stress and Diathesis

	No diathesis	Diathesis
No stress	18 / 126 = 14%	38 / 141 = 27%
Stress	30 / 560 = 50%	418 / 56 = 75%

SUMMARY

The data clearly show that subjects who have lived through the extremely stressful experience of being inmates of a concentration camp, and losing members of their families, have less than half the chance of avoiding cancer, coronary heart disease, or other causes of death than do comparable subjects who did not have such experiences. Inevitably, this is not an experimental study, and hence it is difficult to draw firm conclusions from the investigation. It seems likely that diathesis played an important part in mediating the higher mortality of the camp group, but the possibility must be considered that malnutrition and other forms of maltreatment may have played at least a contributory role. It is possible that malnutrition lowers the body's resistance to disease, and may also affect later measurement of diathesis. Such a possibility does not seem unreasonable. On the other hand, such factors as smoking, drinking, and overweight, which normally are considered risk factors for disease, were lower in former inmates than in comparisons. Lack of movement is a possible consequence of physical maltreatment, and may be productive of disease (Grossarth-Maticek *et al.*, 1990).

Altogether, the data make sense in terms of the model suggested, and of previous research; interpretation is inevitably to some extent subjective because of the lack of proper experimental control over allocation to group. Epidemiological studies always suffer from such departures from experimental methodology; they cannot be decisive but may lend limited support for reasonably well-established theories, at least in showing that real life experiences may mirror more limited laboratory experiments and the effects of less serious real life stresses. More than that is not claimed for these figures.

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