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Personality and Disease: Correlations of Multiple Trait Scores with Various Illnesses

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Abstract

Correlations between personality measures and self-reported data on health status were examined in a sample of 5133 men and women, aged between 40 and 65. A wider range of diseases was studied than is typical. Small but theoretically meaningful correlations with personality were found for some diseases. Personality syndromes of *Emotional Lability*, *Type A Behaviour*, *Behavioural Control*, *Locus of Control over Diseases* and *Psychoticism* were distinguished factorially. Emotional Lability appeared to be the most robust predictor of general disease vulnerability. Some small but significant associations between specific illnesses and Type A and Behavioural Control were also found.

Keywords

cross-sectional studies, disease-prone personality, health-surveys, psychological risk-factors, self-reported health status

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Personality as a predictor of disease

OVER THE past years, intensive research has been directed towards identifying personality risk factors that may prove useful in predicting the development and possible course of chronic disease(s). The search for psychological variables that predict disease continues since traditional medical risk factors such as smoking, nutritional habits, family history and lack of exercise prove not to be sufficient in explaining the onset of most chronic diseases. Research has focused especially on cancer and coronary heart diseases (CHD) because they are the most life-threatening diseases in the western world. In cancer research, some psychological risk factors such as variables like difficulties in expressing emotions, helplessness and hopelessness, motivation for perfectionism and additional personality variables were grouped together as Type C (= cancer) (Temoshok, 1987). However, studies showed that Type C is more closely correlated with the progression of cancer, rather than with its onset (Sanderman & Ranchor, 1997; Temoshok, 1987). Another important risk factor linked to cancer is depression, although meta-analyses have suggested both a positive association (McGee, Williams, & Elwood, 1994), and no relationship (Wulsin, Vaillant, & Wells, 1999).

The research examining the role that psychological factors play in the onset of CHD has focused on the Type A Behaviour Pattern (TABP), which is defined as being time-urgent, competitive and hostile (i.e. Rosenman, 1996). However, as a recent meta-analysis (Myrtek, 1995) showed, the practical relevance of this concept is questionable since the average strength of the correlations between TABP and disease reached only $r = .009$ ($p < .05$, $N = 46,789$ from 16 studies). As a result, current research focuses on specific components of the TABP concept, i.e. aggression, hostility, anger, time urgency, exaggerated social control (Wright, 1988). Friedman and DiMatteo (1989), for example, reported that the mean correlation between anger/hostility/aggression and health outcomes was $r = .14$ (2.0% explained variance, respectively). Other studies have confirmed a significant, but modest, role for hostility: a meta-analysis (Miller, Smith, Turner, Guijarro, & Hallett, 1996) shows that self-reported hostility traits explain 3.2 per cent of the variance ($r = .18$, respectively) in CHD.

Although the emphasis remains on cancer and CHD research, correlations between other diseases and psychological factors have also been examined. For example, positive correlations were found between diseases of the stomach and neuroticism, as well as introversion (Robertson, Ray, Diamond, & Edward, 1989). Nevertheless, a notorious problem found in the research of chronic, organic diseases is the inconsistency of results reported across various studies. Further, the reported effect sizes, if an effect was reported at all, tended to be weak. Most studies may be criticized for examining the relationship between a single disease and only a few, selected personality variables (Friedman & Booth-Kewley, 1987).

In order to gain a broader understanding of the relationship between personality and disease, Friedman and Booth-Kewley (1987) recommended comparing simultaneously various diseases and multiple personality variables. In this way, the convergent and discriminant validity of the personality concepts as an explanation for the diseases can be tested and the construct validity of the selected personality characteristics can be assured. Through the simultaneous examination of various diseases and multiple personality variables it can be tested whether certain traits correlate specifically with certain diseases or whether they are correlated with diseases in general. If a set of personality characteristics that describe a 'proneness' for a particular disease, e.g. heart disease, also predict a 'proneness' for another disease, e.g. cancer, then, conceptually, it makes little sense to differentiate between a cancer-prone personality and a heart disease-prone personality. Neuroticism (Kirmayer, Robbins, & Paris, 1994), Hostility (Miller et al., 1996) and Type A Behaviour (Rimé, Ucros, Bestgen, & Jeanjean, 1989) have been discussed as marker variables of the disease-prone personality. However, it is difficult to single out any one of these personality traits as a key marker for disease-proneness. First, measures of these traits are correlated (Carmody, Crossen, & Wiens, 1989), so that it is difficult to attribute the source of risk unequivocally. Second, most work has focused on a small number of diseases

conditions only, especially cancer, CHD and psychosomatic disorders (in the case of neuroticism).

To date, the Heidelberg study has focused primarily on testing hypotheses derived from the personality theory of Grossarth-Maticek (see Grossarth-Maticek, Eysenck, & Vetter, 1988), regarding the prediction of heart disease and cancer on the basis of two newly defined personality types. According to Grossarth-Maticek (1989), Type 1 personalities ('Inhibition of self-centred expression') are more susceptible to cancer and Type 2 personalities ('Barriers in the self-centred expression') to coronary heart disease. Characteristics of the remaining personality types are as follows: Type 3 personalities are more susceptible to psychopathic behaviour, Type 4 to health and autonomy, Type 5 to depression and Type 6 to antisocial tendencies, criminal behaviour and greater inclination to become addicted to drugs (for more details, see Grossarth-Maticek, 1989; Grossarth-Maticek et al., 1988). In several large-scale studies (for an overview see Eysenck, 1991), the scales assessing Type 1 and 2 (see Grossarth-Maticek & Eysenck, 1990) showed a high validity in predicting death rates that were consistent with the hypotheses. However, a thorough examination of these studies reveals a number of theoretical inconsistencies, methodological inadequacies and obscurities of data analyses. These problems are discussed in detail in a Special Issue of *Psychological Inquiry* (e.g. Amelang, 1991; Cooper & Faragher, 1991; Derogatis, 1991; Fox, 1991; Grunberg & Singer, 1991; Schüler & Fox, 1991; Suinn, 1991; Temoshok, 1991; van der Ploeg, 1991). The present authors aimed to investigate empirically the replicability of the findings using similar (but not identical) methods to those of Grossarth-Maticek. To date, the first wave of data has been collected. The second wave of data collection, including objective medical information, began in the spring of 2002. The cross-sectional results described so far provide little support for the theoretical concepts of Grossarth-Maticek, whereas neuroticism and related theoretical constructs seem to be the most effective psychological predictors of both coronary heart disease and cancer (Amelang, 1997; Amelang, Schmidt-Rathjens, & Feldt, 1998).

Methodological issues

There are two methodological issues that may threaten the validity of results in this field: use of a cross-sectional design, and use of self-reports. First, statistically meaningful correlations between personality characteristics and disease do not allow a causal meaning to be placed on the observed relationship. A causal interpretation is generally difficult due to the number of possible interpretations (Suls & Rittenhouse, 1990). For example, if a cancer group scores higher on depression scales than a healthy group, this finding could reflect both cause and reaction to the illness. In addition, an unidentified third variable could play a role in a significant correlation. It is possible that depression correlates with unhealthy behaviours (e.g. smoking, poor nutrition) contributing to disease, and thereby relates only indirectly to aetiology. A further explanation could be that both depression and cancer relate to a common biological cause or genetic disposition. Correlations between personality variables and disease may also be mediated by other diseases. For instance, a stroke may be a consequence of hypertension, which in turn might be caused by Type A Behaviour Pattern. It is realistic to suppose that the relationship between personality and health factors reflects multiple and possibly complex causal relationships. However, given the neglect of multi-factor studies in this area, it is important to ascertain the nature of cross-sectional relationships before probing further into questions of causality.

A second issue is that research on personality and health often relies on self-reports, whose validity is open to question. In fact, medical researchers are increasingly addressing the validity of self-reports of illness using health records or evidence from medical examination as criteria (e.g. Bush, Miller, Golden, & Hale, 1989; Heliövaara, Aromaa, Klaukka, Knekt, Joukamaa, & Impivaara, 1993). Broadly, such studies confirm that self-reports are a suitable measure for diseases (Kobasa, Maddi, & Courington, 1981). Robinson, Young, Roos and Gelskey (1997) have recently shown that the specificity of self-reported diseases are generally high, i.e. healthy subjects seldom report having a disease. In contrast, sensitivity varied substantially for different diseases. Similar findings have been

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obtained by other researchers (Bush et al., 1989; Haapanen, Miilunpalo, Pasanen, Oja, & Vuori, 1997; Heliövaara et al., 1993). All these authors conclude that self-report is often valid, but that self-reports are most accurate for diseases that are conceptually clear, severe and persistent. Conversely, there are various factors that may lower validity of self-report. Mental illnesses may carry a social stigma, leading to denial of disease. Some diseases, such as lower back disorders and bronchitis, may not be seen as sufficiently severe or unusual to report. Furthermore, although medical records are seen as 'gold standard' in research of this type (Robinson et al., 1997), inaccuracies in such records may lower agreement with self-report (Ferraro & Su, 2000). Hospital records may be incomplete, inaccurate or even hard to read accurately, leading to a general under-reporting bias, especially when minor illnesses are comorbid with a major disease (Powell, Lim, & Heller, 2001). Consistent with these concerns, evidence shows that self-reports are a better predictor than medical records of disability measured 15 years later (Ferraro & Su, 2000). Heistaro, Jousilahti, Lahelma, Vartiainen and Puska (2001) found that initial self-rating of health was a good predictor of mortality in a 23-year prospective study.

Even if self-reports are generally acceptably valid, personality may still be associated with memory and reporting biases that influence associations between personality and reported health (see Stone, Turkkan, Bachrach, Jobe, Kurtzman, & Caine, 2000). In particular, subjects high in Neuroticism are said to be prone to complain about physical symptoms (Stone & Costa, 1990), so that apparent associations between personality and disease may be an artefact of response biases. However, it is unwise to dismiss links between personality and self-reported disease as being solely a consequence of methodological artefact. In some areas of research, such as the link between hostile personality and CHD (see Whiteman, Deary, & Fowkes, 2000 for a review), personality effects on health are substantiated by prospective studies of objective pathology. Admittedly, it has proved difficult to link neuroticism to objective heart disease (as opposed to pain and subjective symptoms: Adler & Matthews, 1994). At the same time, there is some evidence that neuroticism may be linked to cardiovascular reactivity, hypertension and other physiological processes that may promote illness (Byrne, 1992; Friedman, 2000). Furthermore, self-reported complaints may be a consequence of psychosomatic diseases that are genuine disorders but whose physical basis is uncertain. Diseases of this kind associated with neuroticism include non-ulcer dyspepsia, irritable bowel syndrome and chronic fatigue (Matthews & Deary, 2003). Thus, it is likely that correlations between personality traits and disease may reflect a variety of causal processes, perhaps varying from trait to trait, or from illness to illness. The multi-causal nature of associations highlights the need for research investigating multiple traits and diseases.

Aims of the research

This article aims to broaden the spectrum of analyses for personality and disease, following the recommendation of Friedman and Booth-Kewley (1987). Use of a self-reported cross-sectional database has some clear limitations. However, these limitations are offset by: (1) increasing evidence for the validity of self-reports from medical research; (2) the likelihood that personality correlates of disease may relate to both physical and psychosomatic illness; and (3) the presentation of these findings as a part of a wider investigation that will eventually provide mortality data. In addition, the present study was concerned primarily with well-known chronic diseases that have clear diagnostic criteria and are easily communicated to the patient, the class of disease for which self-reports appear to be most valid (Haapanen et al., 1997). Specifically, this study addresses the following questions:

- What is the appropriate psychometric framework for operationalizing the multiple aspects of personality that may relate to diseases?
- How do personality traits correlate with a spectrum of self-reported major disease(s)?
- Are personality–disease correlations specific for a particular disease, e.g. cancer, CHD or diabetes? Alternatively, do personality–disease correlations reflect a general vulnerability to disease?

Method

Participants

The participant sample was drawn from the city of Heidelberg, Germany, and several neighbouring communities. The names and addresses of residents of both sexes between the ages of 40 and 65 (only a few participants were of an age outside this range) were made available to us by the respective town halls. The names and addresses of residents were selected on a random basis for the city of Heidelberg; for the smaller communities, the resident lists contained all those in the targeted age range. Participants were assured that personal data would be coded and handled anonymously; they were paid for participation with approximately 15 Euros.

Recruitment of participants

From the resident lists, all participants were contacted who were listed in the telephone book, and whose names were not obviously foreign (to avoid potential communication difficulties which we had initially experienced). Having a telephone was a criterion of eligibility because telephone contact was used to request participation. Later, telephone contact permitted clarification of minor administrative problems (e.g. missing bank account numbers for remitting the honorarium).

The following procedures have been used for data collection: one procedure involved sending a letter to residents describing our study and then requesting participation over the telephone. When consent was given, participants were invited to our university department or to a community hall where data were collected in small groups (approximately 20 participants per group) ($n = 2047$). Another procedure was to mail the questionnaire to the participants after consent was given over the telephone ($n = 2306$). A further procedure was to send the questionnaire and a letter of explanation without first asking whether or not residents would participate. A subsample ($n = 780$) was recruited in this manner. This procedure, however, has been discontinued due to a low return rate for the postage paid. For more details concerning recruitment of participants see Amelang, Schmidt-Rathjens and Matthews (1996). No substantial differences of statistical parameters (means and correlations) between the different methods of questionnaire administration were found (see also Amelang, Schmidt-Rathjens, & Yousfi, 2001).

Response rate

If residents said that they were unwilling to participate, their names were removed from the list. Even though telephone contact was an essential organizational tool, it was impossible to reach all potential participants in this manner (either the residents were not at home when we called, or they had moved or had died, or we had only indirect contact by leaving a message with a family member or on an answering machine). In part, this may be due to the fact that the resident lists were obtained during the early preparation stages of this study. In this way we tried to get contact with more than 15,000 residents. We have had direct communication with 10,250 (i.e. residents from whom we had a direct 'yes' or 'no' answer about participation). Finally 5133 persons participated in this study. The average response rate was calculated to be approximately 55 per cent, i.e. the number of participants related to the number of persons with whom we had personally communicated ($n = 10,250$). This value may be close to the upper limit for a study of this kind because it was necessary to mention the possibility of later disease (and even death) in the introductory remarks, and (for legal reasons) to request the participants' written agreement to collect data on cause of death from the respective local health centers. These remarks may have been threatening to a substantial proportion of individuals and may have discouraged participation. Gender had no apparent effect on the willingness to participate. The mean age for the male participants was 53.8 (SD = 7.2, due to missing values $n = 2434$) and for the female participants 53.0 (SD = 7.2, due to missing values $n = 2652$).[1]

A sample of 1316 residents, who had refused participation in the study were asked to provide their age, sex, education level and marital status over the phone. These results showed that the individuals refusing participation were on average 55.5 years old (SD = 7.1), 2 years older than participants. Moreover, the non-participants were more likely to be employed (61 per cent compared to 34 per cent participants) and

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were less educated (63 per cent of the non-participants had only completed the Grammar school, which is equivalent to approximately 9 years of primary education, compared to 48 per cent for the participants). Results indicate small differences in family status between the participants and non-participants, i.e. non-participants were more likely to be married or widowed than participants. These differences suggest that the participants were not representative of the sample of contacted residents.

Questionnaire materials

Measurement of personality Participants completed a battery of scales measuring personality factors. Scales were selected if they had been *empirically* demonstrated to be correlated with diseases, or if there was a *theoretical* rationale for linking the personality trait to diseases. Specifically, the following scales were used:

- R(evised)-Scales: Due to the controversy surrounding the claims made by Grossarth-Maticek (e.g. 1989) and colleagues that certain personality types may be strongly susceptible to cancer and coronary heart disease and due to the fact that these diseases are the leading causes of death in western societies, it seemed appropriate to include items assessing the Personality Types 1 and 2 proposed by Grossarth-Maticek in this battery of scales. However, the original scales from Grossarth-Maticek contain repetitive items and showed insufficient item–scale correlations so we revised the six Grossarth-Maticek scales. These scales, labelled R(evised)-Scales, were internally consistent in each of the samples used for scale construction and, more importantly, were highly intercorrelated with the original scales. Details regarding the construction of the R(evised)-Scales can be found elsewhere (see Amelang & Schmidt-Rathjens, 1992, 1993; Schmidt-Rathjens & Amelang, 1993). Because the other scales of the Grossarth-Maticek typology seemed also to be relevant for the differentiation between health and illness (e.g. Type 4 'Health/autonomy', Type 5 'Rationalism') they were also included.
- Scales associated with coronary heart disease and cancer: In addition to Type 1 and Type 2 personalities proposed by Grossarth-Maticek, other personality factors have played an important role in recent research. In correspondence with current research, we selected scales that focus on specific components of the TABP concept including the factors Time Urgency and Perpetual Activation (TUPA), Anger Expression (Anger In, Anger Out, Anger Control), Aggression, Irritability, Jealousy and Exaggerated Social Control. For Cancer, Anger In and Depression have been shown to be important personality variables.
- Scales with protective qualities: Research surrounding Antonovsky's theory of health (Antonovsky, 1979, 1987, 1993) has focused on why individuals remain healthy despite the stresses experienced every day. Based on Antonovsky's theory of health, we included the following protective factors in our battery of scales: Sense of Coherence, Optimism, Locus of Control Over Diseases and Social Support.
- Scales of general importance in personality research: As representatives of relatively broad factors in personality research, Neuroticism, Extraversion and Psychoticism were also included in the questionnaire.

Table 1 shows the list of personality scales utilized for the present study.

Measurement of disease(s) With regard to present and past state of health, participants indicated whether they had (or have had) any of the diseases or risk factors listed in the first column of Table 3. With the list of presented diseases we intended: (1) to broaden the number of diseases to incorporate other important bodily organs in addition to the heart; and (2) to assess other diseases that have received more research attention in recent years due to increasing frequency of these diseases in western societies, e.g. asthma and diabetes. Reported diseases were subjective reports and were not confirmed by a medical practitioner.

The questionnaire also collected information on living habits such as smoking, exercise, sleeping habits and educational level. Some of these variables were already dealt with elsewhere

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Table 1. List of personality scales utilized for the present study. For each of the scales Cronbach-α is indicated for the total sample (N = 5.133)

A revised version of the six scales used by Grossarth-Maticek and co-authors for measuring their six personality types (Amelang & Schmidt-Rathjens, 1992)

R-Scale:

1. *Inhibition of self-centred expression*; disposition for cancer: 28 items (α = .90)
2. *Barriers of self-centred expression*; disposition for CHD: 25 items (α = .92)
3. *Psychopathology*: 20 items (α = .83)
4. *Health/autonomy*: 31 items (α = .88)
5. *Rationalism*: 23 items (α = .91)
6. *Antisocial tendencies*: 13 items (α = .76).

Optimism (Life Orientation Test, LOT; Scheier & Carver, 1985): 8 items (α = .55)
Questionnaire for measuring the locus of control over diseases (FEGK; Ferring & Filipp, 1989): 16 items concerning the *Internal Locus of Control* and 13 concerning the *External Locus of Control* (α = .79, .81, respectively)
Time Urgency and Perpetual Activation Scale (TUPA; Wright, McCurdy, & Rogoll, 1992): 13 items (α = .65)
State-Trait-Anger Expression-Inventory (STAXI; Schwenkmezger, Hodapp, & Spielberger, 1992): 24 trait-items measuring *Anger In*, *Anger Out* and *Anger Control* (α = .82, .83, .81, respectively)
Social Support-Scale (SOZU K–22; Fydrich, Sommer, Menzel, & Höll, 1987): 22 items (α = .91)
Depression Scale (von Zerssen, 1976): 16 items (α = .85)
Sense of Coherence Scale (SOC-HD; Schmidt-Rathjens, Benz, Van Damme, Feldt, & Amelang, 1997): 19 items (α = .80)
Hostility (Dimension '*Aggression*', Saltz-Epstein-Questionnaire: 8 items; Dimensions '*Irritability*' and '*Jealousy*', Buss-Durkee-Hostility-Inventory: 11 and 8 items respectively; from Kornadt, 1982) (α = .62, .66, .63, respectively)
Psychoticism (Baumann & Dittrich, 1976): 20 items (α = .60)
Eysenck-Personality-Inventory (EPI; *Extraversion*, *Neuroticism* and *Social Desirability* Eggert, 1974): 57 items (α = .69, .85 and .57 respectively)
Exaggerated Social Control (Way of Life Scale, WOLS; Wright, von Bussmann, Freidman, Khoury, Owens, & Paris, 1990): 21 items (α = .76)

(Amelang, 1997; Amelang et al., 1996); for the sake of clarity and due to limited space these variables will be not be analysed in the present article. Since not all participants provided all of the information requested, missing values were replaced with the mean for continuous variables and with the mode for dichotic variables. A total of 3806 subjects have complete data. Forty-seven subjects did not report their age. On average, each participant had .71 (= 5.1%) missing values with respect to the diseases. Seventy-six per cent of the participants had no missing data in the list of diseases, 89 per cent had no more than 1 missing value.

Analyses for aggregation of personality and health measures

Factor analysis of personality variables In order to obtain variables with a higher degree of reliability and to reduce redundancy between scales, orthogonal factor analyses were run on the personality scales listed in Table 1. Principal component analysis was used as the method of factor extraction. (These results and those from the following paragraphs will be reported in the Results section.)

Aggregation of diseases We expected that different diseases of the same organ could be combined, because the different disease items in our questionnaire that refer to the same organ differed rather in the severity and progression than in the kind of disease (e.g. asthma and chronic bronchitis, gastric ulcers and chronic gastritis, myocardial infarction and angina pectoris). A further advantage is that aggregation may increase the validity of self-reports, in that people may become confused between related conditions (e.g. infarction vs. other heart disease: Paganini-Hill & Chao, 1993).

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Tetrachoric correlations were calculated in order to confirm the validity of aggregating related diseases of the same organ.

Association of aggregated personality measures and diseases

Point-biserial correlations As a first test of the relationships between the disease categories and personality variables, point-biserial correlations were calculated (Categories: symptom vs. no symptom). Regression estimates of the individual factor scores from the factor analysis of the trait variables were used as personality measures. Although the intercorrelations between aggregated disease categories were expected to be small, inter-category correlations of any size make it difficult to interpret the results of the point-biserial correlations since the strength of the effect could be influenced by confounding variables. Therefore, semi-partial correlations (= part correlations) were calculated to test whether the particular disease categories do in fact relate to some of the variance of the personality factors independent from the rest of the disease variables, i.e. to test incremental validity of particular disease categories. If the semi-partial correlation is substantially lower than the point-biserial correlation, then it might be that the personality factor and the disease are only indirectly related, i.e. there are other diseases, age or gender that mediate the correlation. Moreover, we calculated partial Eta-Square for the joint effect of all disease on the personality factors whereas the effect of age and gender was statistically controlled. Partial Eta-Square indicates the proportion that all diseases jointly explain from the variance in the personality factor that remains after the effects of age and gender were partialled out (Cohen, 1973).

Mean differences Correlations, or, more exactly, squared correlations, specify the effect strength as the part of the variance in the personality variable that can be explained by the disease. When a disease is seldom reported, then the frequency distribution of the 'yes/no' answers is skewed, which means that even when the statistical relationship between disease and personality variables is high, the computed correlations and consequently the explained variance are low. Differences between individuals with and without a particular disease should be calculated using a measure of effect size that is independent of the prevalence rate like mean differences. Mean differences are reasonably reported in units of the pooled standard deviations within the groups reporting a disease and not reporting a disease ($d=(M_{ill} - M_{healthy})/SD_{within\ groups}$, see Cohen, 1988). The formulas for both measures of effect size (d and r) cohere in the following manner:

$$r = d/\sqrt{d^2 + 1/(p(1-p))} \text{ or } d = r/\sqrt{p(1-p)(1-r^2)}$$

where p is the prevalence rate and r is the point-biserial correlation.

The semi-partial correlations can also be expressed as mean differences when least-square means (estimated population marginal means) are calculated within the framework of the general linear model (Searle, Speed, & Milliken, 1980). The least-square means are not the actual means; instead they are the means that the theoretical model predicts for uncorrelated regressors. Consequently, they reflect the mean difference (in the personality factors) between subjects who are identical with regard to the other diseases, age and gender. Contrary to the observed mean differences, the least-square mean differences could not be attributed to confounding regressors. In order to achieve a measure of effect size, which is comparable with d, we calculate the difference between the least-square means in units of the standard deviation of the residual of the corresponding linear model. The statistical significance of the least-square mean differences is identical to the significance of the corresponding semi-partial point-biserial correlation. The level of significance for the differences in the sample mean values is equivalent to the level of significance for the basic point-biserial correlation. Hence, the choice of correlations (Pearson/semi-partial) or mean differences (ordinary/least-square) has no effect on the evaluation of the statistical significance of an effect. However, the choice of the measure of effect size may lead to different evaluations of the practical relevance of an effect if there are differences in the prevalence rate. Whereas the magnitude of a correlation is directly related to its statistical significance, the relationship (of the magnitude)

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of a mean difference to its statistical significance depends on the prevalence. Even high mean differences are difficult to discriminate from sampling error if the prevalence is very low.[2]

Results

Factor analysis of personality variables

Formal criteria (scree-test, slope of Eigenvalues 7.70 – 2.52 – 2.26 – 1.26 – 1.08 – .91 – .84 – .79 – .73 – .68 . . .), and consideration of the content of factor loadings, suggested the extraction of five factors. The varimax-rotated structure is shown in Table 2.

The structure of the rotated varimax loadings is quite clear. Neuroticism, Depression and both R-Scales 1 and 2 (Inhibition of self-centred expression, Barriers of self-centred expression, respectively) load positively on Factor 1; Sense of Coherence (SOC-HD), Optimism and R-Scale 4 (Health/Autonomy) load negatively on Factor 1. Due to the pattern of loadings this factor was labelled Emotional Lability. R-Scales

3 and 6 (Psychopathology and Antisocial Tendencies, respectively), Time Urgency and Perpetual Activation Scale (TUPA), Exaggerated Social Control, Anger Out and Extraversion load positively on Factor 2. This group of loadings contains the major characteristics of Type A Behaviour. R-Scale 5 (Rationalism), Social Desirability and Anger Control load positively on Factor 3; whereas Anger Out, Irritability and Aggression load negatively on this factor. These loadings describe Behavioural Control. The highest loadings on Factor 4 are those for internal and external Locus of Control over Diseases. Therefore, Factor 4 received the label Locus of Control over Diseases. Psychoticism loads positively on Factor 5 while Social Support loads negatively on this factor. The two highest loadings define this factor, which was labelled Psychoticism. Despite the fact that the factor labels we chose refer mainly to well-known personality traits, they are more than just surrogates of these original scales. In contrast to these established personality scales, the reported factors are uncorrelated, which

Table 2. Rotated varimax loadings on 5 factors for the total sample (N = 5133). Numbers in bold indicate loadings higher than .45

Scales	Factor 1	Factor 2	Factor 3	Factor 4	Factor 5	h^2
R-Scale 1 (Inhibition)	**.81**	.28	.15	.00	–.05	.75
R-Scale 2 (Barriers)	**.81**	.39	–.05	–.09	.00	.82
SOC-HD	**–.76**	.01	.17	.34	–.20	.77
Depression	**.76**	.18	–.03	–.06	.08	.63
Neuroticism	**.76**	.23	–.27	.05	–.05	.70
R-Scale 4 (Health/Autonomy)	**–.70**	.04	.31	.34	–.25	.77
Anger In	**.68**	.07	.08	.29	.05	.56
Optimism	**–.64**	.00	.05	.30	–.22	.56
Jealousy	**.54**	.32	–.11	.09	.28	.49
Irritability	**.53**	.29	**–.50**	.05	–.04	.62
R-Scale 3 (Psychopathology)	**.51**	**.69**	.02	–.05	.08	.74
R-Scale 6 (Antisocial Tendencies)	.31	**.70**	–.08	–.14	.16	.64
TUPA	.20	**.69**	.07	.10	.08	.54
Exaggerated Social Control	–.02	**.54**	–.19	.22	.09	.38
Extraversion	–.39	**.49**	–.29	–.02	–.30	.57
R-Scale 5 (Rationalism)	.15	.34	**.70**	.19	.04	.67
Social Desirability	–.09	–.08	**.69**	–.09	–.02	.50
Anger Control	–.12	–.25	**.58**	.42	.08	.60
Anger Out	.25	**.48**	**–.51**	–.07	–.10	.56
Aggression	.21	.41	**–.47**	.12	.17	.47
Internal LOC over diseases	–.04	.18	.06	**.75**	–.10	.61
External LOC over diseases	.29	.44	.39	**–.47**	–.05	.66
Psychoticism	.04	.17	.05	–.02	**.80**	.67
Social Support	**–.46**	–.03	.04	.13	**–.54**	.53
Variance explained	7.69	2.51	2.27	1.26	1.08	

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prohibits redundancy and facilitates the interpretation of correlations to disease. Nevertheless, it should be noted that the factorial structure remained essentially the same after application of an oblique rotation.[3]

Correlations and aggregation of the diseases

The frequencies and the tetrachoric correlations of the diseases are reported in Table 3.

As expected, different diseases of a particular organ were correlated substantially. Four aggregated disease categories were defined by multiple diseases: pulmonary disease (asthma, bronchitis), stomach disease (duodenal ulcer and gastritis), liver (hepatitis, cirrhosis) and CHD (angina pectoris, myocardial infarction, cardiomyopathy). The tetrachoric correlations between the diseases within each category range from 0.68–0.82, and are shown in bold in Table 3. Five of the remaining diseases—cancer, gallstone, goiter/thyroid, urinary/renal and indigestion—showed no correlations exceeding .4 with other diseases, and were treated as 5 separate disease categories. Stroke and diabetes were also treated as 2 more separate disease categories, although they were moderately correlated (r = .4–.6) with diseases related to CHD, and with each other. The risk factors of stroke and CHD overlap widely (Taylor, 1995), but acquiring stroke participants is rare (here: $n = 78$) due to the high mortality rate. In addition, many individuals after suffering from stroke may be unable to participate in a study of this kind. Finally, three risk factors for cardiovascular disease (hypertension, adiposity and high cholesterol levels) were only moderately correlated with each other, and with CHD diseases, and were analysed as separate categories. Thus, diseases were analysed using four multi-disease categories, seven single-disease categories and three cardiovascular risk factors.

Results of point-biserial correlations and mean differences

The point-biserial and semi-partial correlations between disease(s) and personality factors are found in Table 4.

The respective effect sizes of the mean differences in personality associated with different disease groups can be found in Fig. 1.

As shown in Table 4, the uncorrected correlations between *all* of the disease categories and the personality factor *Emotional Lability* were highly significant. Even after the effects of age and gender were statistically controlled, the diseases jointly explain 8 per cent of the remaining variance in *Emotional Lability*. The highest correlations were obtained for indigestion, stomach and heart diseases. Nevertheless, the largest mean differences were found for stroke. However, the correlation between stroke and *Emotional Lability* is influenced by other diseases, age and gender as indicated by the statistical insignificance of the semi-partial correlation and the least-square mean difference. Nevertheless, the least-square mean difference for stroke is higher than the statistically significant least-square mean difference of pulmonary diseases. The inconsistency between the statistical insignificance and the relative high least-square mean difference associated with stroke is a consequence of its relatively low prevalence. The semi-partial correlations (and the least-square mean differences) for cancer, diabetes, gall, liver thyroid and urinary/kidney diseases indicate that these diseases do also not contribute to the explanation of *Emotional Lability* variance when controlling for other diseases, age and gender. In other words, there is no correlation between these illnesses and *Emotional Lability* for subjects that do not differ with regard to other diseases, age and gender.

Stroke showed the largest mean differences with respect to *Type A Behaviour*, too. This effect remains statistically significant even after controlling for other diseases, age and gender. Again, the low magnitude of the corresponding semi-partial correlation and significance level is a consequence of the low prevalence of stroke. Notable mean differences were also found for diabetes, adiposity, heart and pulmonary diseases. Adiposity and heart diseases showed the highest correlation coefficient. With the exception of cancer, thyroid and urinary diseases all other diseases correlated significantly with *Type A Behaviour*. However, liver, gall and stomach diseases, hypertension and indigestion do not contribute incrementally to the explanation of the variance.

With the exception of cancer, liver, pulmonary and thyroid diseases all other

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Table 3. Tetrachoric correlations and frequencies of the disease items

	N	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19
Angina pectoris	537	1	.	.	.	.	.	.	.	.	.	.	.	.	.	.	.	.	.	.
Myocardial infarction	167	**.82**	1	.	.	.	.	.	.	.	.	.	.	.	.	.	.	.	.	.
Cardiomyopathy	395	**.73**	**.71**	1	.	.	.	.	.	.	.	.	.	.	.	.	.	.	.	.
Diabetes	292	.41	.52	.43	1	.	.	.	.	.	.	.	.	.	.	.	.	.	.	.
Stroke	78	.57	.59	.62	.50	1	.	.	.	.	.	.	.	.	.	.	.	.	.	.
Hypertension	1378	.34	.35	.32	.38	.42	1	.	.	.	.	.	.	.	.	.	.	.	.	.
Adiposity	1097	.30	.35	.28	.45	.36	.50	1	.	.	.	.	.	.	.	.	.	.	.	.
High cholesterol levels	2092	.32	.35	.25	.32	.40	.39	.36	1	.	.	.	.	.	.	.	.	.	.	.
Asthma	335	.31	.24	.36	.27	.32	.21	.28	.16	1	.	.	.	.	.	.	.	.	.	.
Chronic bronchitis	634	.31	.16	.30	.25	.28	.19	.23	.23	**.68**	1	.	.	.	.	.	.	.	.	.
Hepatitis	742	.36	.30	.33	.36	.39	.27	.30	.28	.34	.27	1	.	.	.	.	.	.	.	.
Cirrhosis of the liver	121	.37	.36	.41	.43	.51	.22	.33	.30	.52	.41	**.82**	1	.	.	.	.	.	.	.
Gallbladder stone	664	.22	.14	.21	.18	.27	.19	.28	.13	.13	.14	.32	.39	1	.	.	.	.	.	.
Gastric/duodenal ulcer	705	.25	.23	.21	.15	.24	.09	.12	.17	.29	.26	.29	.38	.25	1	.	.	.	.	.
Chronic gastritis	1819	.23	.10	.13	.07	.14	.03	.05	.15	.18	.23	.28	.26	.22	**.76**	1	.	.	.	.
Goiter/thyroid complication	772	.12	.05	.16	.01	.17	.07	.08	.03	.13	.07	.13	.21	.19	.15	.16	1	.	.	.
Urinary/renal calculus	1189	.17	.09	.12	.05	.15	.08	.08	.06	.08	.10	.17	.16	.20	.11	.22	.19	1	.	.
Indigestion/constipation	1451	.13	.08	.13	.08	.15	.04	.12	.11	.14	.16	.22	.21	.28	.26	.34	.23	.31	1	.
Carcinoma	284	.19	.14	.17	.14	.03	.01	.02	.05	.10	.11	.14	.22	.15	.17	.12	.13	.13	.18	1

Table 4. Correlations of diseases, age and gender with personality. Point-biserial for semi-partial (= part) correlations (all other diseases, age and gender controlled) are reported

	N	Emotional Lability		Type A Behaviour		Behavioural Control		LOC over Diseases		Psychoticism	
		bivariate	part	bivariate	part	bivariate	part	bivariate	part	bivariate	part
		correlation		correlation		correlation		correlation		correlation	
CHD	758	.15***	.08***	.11***	.05**	.09***	.03*	.02	.03*	.05***	.04**
Cancer	284	.05***	.01	.02	.00	.00	−.01	−.03	−.02	−.02	−.01
Liver	758	.09***	.02	.07***	.01	.00	−.03*	.02	.01	.06***	.04**
Stomach	1918	.16***	.10***	.05**	.02	−.08***	−.08***	.02	.01	−.03	−.04**
Pulmonary	784	.09***	.04**	.10***	.06***	−.01	−.02	.00	−.02	.07***	.06***
Diabetes	292	.05**	−.01	.10***	.05**	.06***	.01	−.02	−.03*	.01	−.01
Stroke	78	.06***	.02	.08***	.04*	.04**	.01	−.02	−.03*	.04*	.02
Gall	664	.09***	.00	.04**	.00	.06***	.04**	−.02	.00	−.03	.00
Goiter/Thyroid	772	.08***	.01	.01	.01	−.02	.00	−.01	.02	−.06***	−.03
Urinary/Kidney	1189	.09***	.02	.02	.01	−.04**	−.02	.01	.01	−.03*	−.02
Indigestion	1451	.21***	.13***	.03*	.01	−.04**	−.02	−.01	.01	−.02	.03
Hypertension	1378	.10***	.03*	.07***	.01	.10***	.02	.00	−.02	.01	.00
Adiposity	1097	.12***	.06***	.12***	.07***	.06***	.01	.03	.04**	.00	−.02
High cholesterol	2092	.13***	.07***	.09***	.03*	.07***	−.00	.01	.00	.01	−.02
Gender (f)		.13***	.12***	−.07***	−.05***	−.09***	−.06***	−.13***	−.13***	−.20***	−.18***
Age		.09***	.03**	.10***	.05***	.32***	.29***	−.03*	−.03*	−.01	−.01
Partial Eta^2		.08***		.03***		.02***		.01*		.01***	
Multiple R		.33***		.21***		.35***		.15***		.22***	

$*$ $p < .05$; $**$ $p < .01$; $***$ $p < .0007$ (This corresponds to a Bonferroni-corrected p-value of .05 for all 70 correlations between diseases and the personality factors. Correlations with higher p-values should be interpreted with caution)

The values in the columns labelled 'part' are semi-partial correlations controlled for the relationship of all the other diseases, age and gender with the personality variables in question. Positive correlations of gender implicate higher value for females on the respective factor. The power of the test with significance level of .0007 is 99.9 per cent for bivariate and point-biserial correlations of $rho = .09$. The power for detecting an effect of $rho = .05$ is 95 per cent when testing with a significance level of 5 per cent

Partial Eta-Square indicates the proportion of the variance of the respective personality factor that all diseases jointly explain from the variance which remains after gender and age have been controlled statistically

Multiple R has to be squared to get the explained variance of the respective model with all predictors
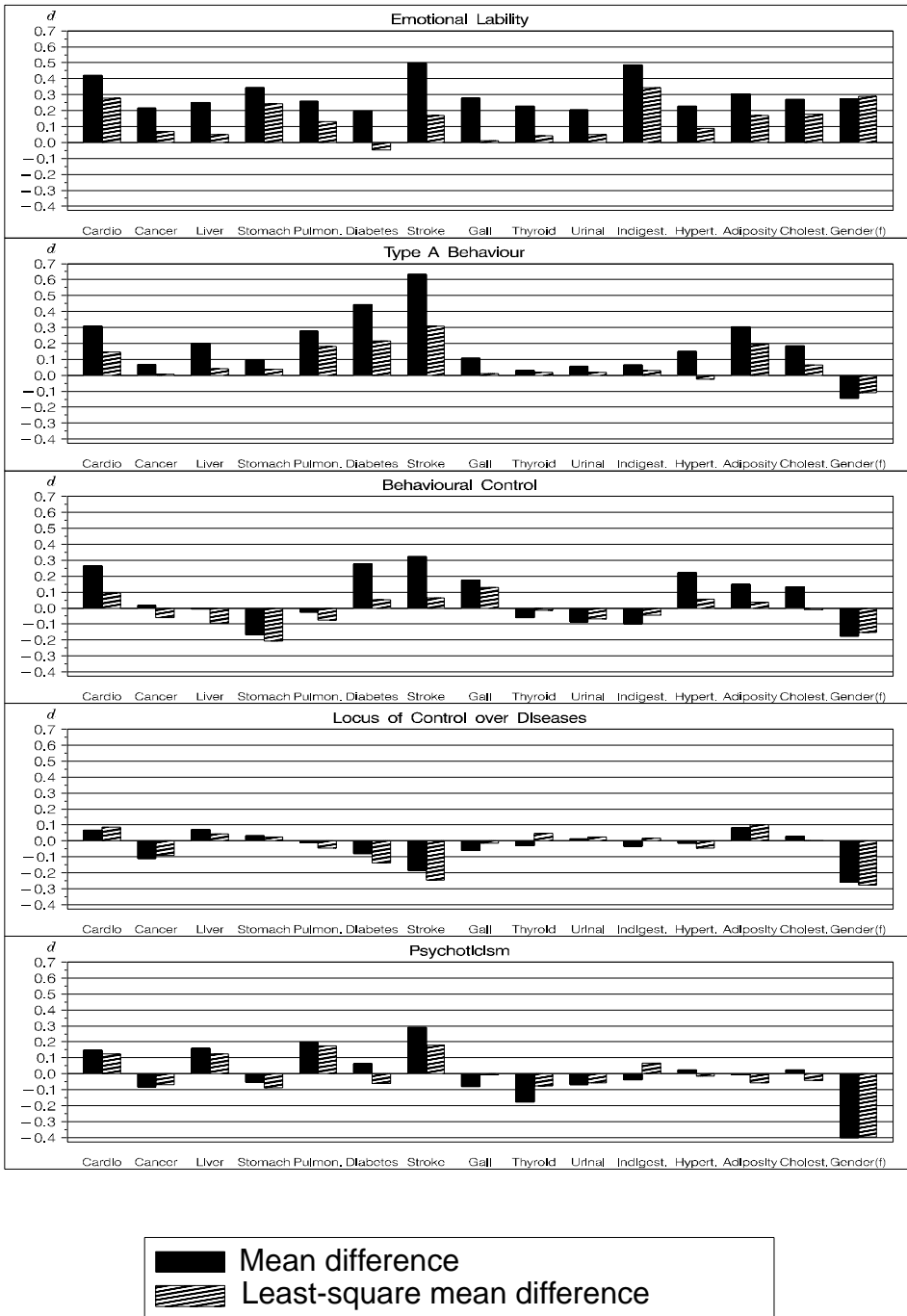
Figure 1. Mean differences and least-square mean differences (*d*) associated with diseases and gender. All subjects without the disease in question served as reference group. Gender differences are reported for females with respect to males.

diseases correlated significantly with *Behavioural Control*. Notable mean differences were found for stroke, diabetes, hypertension and heart disease. However, stroke and diabetes did not contribute incrementally to the explanation of variance in *Behavioural Control*. The significant correlation between *Behavioural Control* and indigestion, hypertension, high cholesterol and adiposity decrease also to non-significant levels after controlling for other diseases, age and gender. Solely, diseases of the stomach, liver, gall and heart remain statistically significant when other diseases, age and gender are controlled.

Analyses run on the personality factor *Locus of Control over Diseases* failed to show any significant, interpretable correlations with disease(s).[4] The corresponding multiple correlation relating personality to the disease and demographic predictors is only significant through the correlations with age and gender.

With regard to *Psychoticism*, small but statistically highly significant correlations were obtained for pulmonary, liver, thyroid and heart diseases. The size of the respective mean differences is practically meaningless. Although Stroke shows the highest mean difference, this difference is no longer significant after controlling for other diseases, age and gender. Only the effects of liver, pulmonary and heart disease might be interpretable because at least the correlations seem to be of reasonable size and they remain significant with other diseases controlled. The statistical relation of thyroid disease to *Psychoticism* might be mediated by age, gender or other diseases.

Discussion

The present study demonstrates various small but significant associations between personality and self-reported health status. As previously discussed, the cross-sectional design and use of self-report necessitates caution in interpreting results. Subject to these limitations, the results reported have several implications. These include: (1) the choice of psychometric framework for investigating links between personality and illness; (2) the nature of associations between personality and disease; and (3) the extent to which personality factors are disease-specific or general predictors of ill-health. In the

sections that follow we discuss these topics with reference to specific methodological issues where relevant.

Choice of psychometric framework

The findings of this study address one of the major areas of uncertainty in health psychology: the appropriate choice of personality constructs for use in health studies. Investigators have variously used: (1) traits designed from the outset to map onto specific diseases, such as the Grossarth-Maticek Types 1 to 6; (2) broad personality 'superfactors' such as Neuroticism; and (3) narrower but still general traits such as Type A, optimism–pessimism and the Spielberger anger scales. The present data show that factor analysis helps to resolve these difficulties. The first factor here was aligned with neuroticism. It absorbed much of the reliable variance both in the R-Scales, and in some purportedly distinct personality constructs, including Optimism, Anger In, Depression and Sense of Coherence. These data are consistent with the view that a variety of health-related traits relate primarily to negative affectivity or neuroticism (Kirmayer et al., 1994; Matthews & Deary, 2003). However, the factor analysis also identified dimensions specific to the health context. Factor 2, for example, corresponds to the established Type A construct, in blending constructs such as time urgency, expressed anger and extraversion. Factor 3 resembles the so-called Type C (cancer-prone) personality (Temoshok, 1987), characterized by excessive rationality, and suppression of negative emotions. In contrast to typical Type A and Type C constructs (see Deary, MacLullich, & Mardon, 1991; Sanderman & Ranchor, 1997), Factors 2 and 3 showed no substantial correlations with the Neuroticism scale (see the factor loadings of Neuroticism in Table 2). Factors 4 and 5 were rather specific, relating to locus of control and psychoticism, respectively.

Current research tends to use a varied collection of personality measures, many of which are confounded with neuroticism (Sanderman & Ranchor, 1997). The findings here suggest a different research strategy. The first step is to check for variance in health that may be attributed to the general emotional lability factor, followed by an assessment of the correlates of

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more specialized factors. Hostility, for example, is often discussed as characteristic of the disease-prone personality, but it may be useful to distinguish this trait from general emotional lability, as a more specific aspect of TABP (see Carmody, Crossen, & Wiens, 1989). Our factor of Emotional Lability may pick up some of the variance associated with hostility, as indicated by the loadings of Irritability and Jealousy. Type A Behaviour emerged as a separate factor including some hostile aspects (as did the negative pole of *Behavioural Control*).

Correlations between personality traits and the disease spectrum

In this study, we have attempted a comprehensive assessment of personality traits relevant to health. Through factor analyses, the assessed characteristics were grouped into five orthogonal dimensions, and correlated with multiple diseases. For each of these dimensions, the most important results will be summarized, and their relationship with previous findings will then be discussed.

First, it must be stressed that all the reported empirical relations between diseases and personality are rather low in magnitude. Hence, the widespread opinion (see Eysenck, 1991) that personality is one of the major causes for disease is certainly not supported by the present results. However, there were some effects that could not be interpreted as sampling error, even following a conservative approach that controlled for effects of other diseases, age and gender, and for inflation of alpha error across all 70 predictor-criterion correlations. The significant correlations or mean differences were of small effect size, according to the conventions of Cohen (1988). Nevertheless, these small associations are of some interest. The effect sizes were larger or comparable to those established by meta-analyses of the Type A and hostility constructs (Miller et al., 1996; Myrtek, 1995); traits that are a major focus for health psychology. Whiteman et al.'s review (2000) concluded that personality variables are associated with relative risk factors for CHD varying from 1.2 to 2.0. This corresponds roughly to range from $d =$ .2 and $d = .7$. Even a small effect size may thus correspond to a substantial number of cases within a regional or national population. The

practical relevance of these small effects should be considered with regard to the large humanitarian and monetary costs of the diseases.

Furthermore, despite small effect sizes, reducing Type A Behaviour has been seen as an appropriate target for clinical intervention (Bracke & Thoresen, 1996). Evidence supports the utility of psychosocial interventions that attenuate stress-related behaviours, including Type A Behaviours, in reducing mortality in cardiac patients (Cardiac Rehabilitation Guideline Panel, 1995; Schneiderman, Antoni, Saab, & Ironson, 2001). If it is worthwhile addressing Type A personality as a risk factor for CHD, then it may also be worthwhile to investigate interventions directed towards other disease risks associated with personality, as identified in this study and others. In reference to the biopsychosocial model (Taylor, 1995) the aetiological onset of diseases is a multi-factorial process that includes not only psychological factors but also physical, hereditary, social and numerous other factors. Neglecting even weak risk factors prohibits the development of models that explain how the various factors interact in the development of diseases and may hinder progress in this field of study.

According to our results, *Emotional Lability* is correlated with all of the diseases surveyed, a finding that is consistent with previous studies suggesting that neuroticism operates as a general risk factor (Kirmayer et al., 1994). The largest effects were found for CHD, stroke and indigestion. The first two of these are aspects of illness for which self-reports appear to be generally valid (Haapanen et al., 1997; Heliövaara et al., 1993). The association between neuroticism and indigestion should perhaps be treated with caution, given that such illnesses may have a psychogenic component. For example, irritable bowel syndrome is known to be linked to neuroticism (Kellner, 1991).

The incidence of CHD was more strongly linked to *Emotional Lability* than to *Type A Behaviour*, further supporting the importance of distinguishing these two personality constructs in health research. However, the hypothesis that *Emotional Lability* and cancer are correlated was not strongly supported by our results, consistent with other recent work (McKenna, Zevon, Corn, & Rounds, 1999). Although individuals reporting cancer scored

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higher in *Emotional Lability*, no differences were found after controlling for the effects of other diseases, age and gender. Presence of cancer is generally accurately self-reported (Colditz, Martin, Stampfer, Willett, Sampson, Rosner, Hennekens, & Speizer, 1986), so it is unlikely that the null results reflect reporting bias. These findings contradict the view of Grossarth-Maticek and Eysenck (1990) that cancer is highly predictable from personality.

In addition, *Type A Behaviour Pattern* (TABP) correlated with most of the diseases. The highest correlations were found for CHD, stroke, pulmonary diseases, adiposity and diabetes. Theoretically, TABP is most often associated with CHD, but, here, its correlations with the other disease conditions listed were of similar or larger magnitude to the correlation with CHD. The results found here suggest that the TABP concept may be a relevant factor in other illnesses, independent of any neuroticism/emotional lability effect, despite the fact that the reported correlations are rather low. TABP is said to be associated with a tendency to deny physical symptoms (Carver, Coleman, & Glass, 1976; Weidner & Matthews, 1978), whereas the opposite is true for Emotional Lability. This might have contributed to the differences in effect size.

The results for the personality factor *Behavioural Control* are noteworthy because they contradict the accepted stereotypes that individuals who are unable to express their emotions tend to suffer from diseases of the stomach, while irritable individuals tend to suffer from diseases of the heart. Here, *Behavioural Control* correlates positively with diseases of the heart, and negatively with diseases of the stomach. Both effects seem to be independent of other diseases, age and gender. The relationship between personality and gastric disturbance has been rather neglected, although specific conditions such as ulcers and dyspepsia seem to be generally linked to anxiety and stress (e.g. Haug, Svebak, Hausken, Wilhelmsen, Berstad, & Ursin, 1994). Possibly, behavioural control has some benefits in reducing the impact of stress on gastric functioning. However, accuracy of reporting stomach diseases may be limited by the poorly defined nature of some of these conditions (Westbrook, McIntosh, Rushworth, Berry, & Duggan, 1998).

Behavioural Control might be expected to correlate with cancer-proneness on the basis of its associations with rationalism, anger suppression and social desirability. But in fact, there seems to be no empirical relationship between cancer and *Behavioural Control*. The high values of *Behavioural Control* for people with stroke may be attributed to correlations with other diseases, age and gender. But the low prevalence of stroke in our sample does not permit conclusive interpretations.

Locus of Control and *Psychoticism* were rather weakly related to disease: *Locus of Control* related most strongly to reduced stroke risk, and *Psychoticism* to pulmonary illness and stroke.

The largest mean differences with respect to any of the personality factors were found for stroke, however, only when the effects from other diseases, age and gender are not controlled. These results are difficult to interpret due to the low prevalence rate of stroke in this study. The power for uncovering weak mean differences ($d = .2$) for stroke with a significance level of .05 was only 42 per cent (whereas the respective power for the more frequent heart diseases was more than 99.9 per cent). Hence, the insignificance of the incremental effect of stroke for many diseases might be attributed to the low statistical power of the respective tests. The use of effect strengths, which are independent from the prevalence rate of the diseases like mean differences and odds ratios insure the comparability of the effects from various diseases. However, different test powers are calculated for the same effect strength coefficient if there are prevalence differences.

The interpretation of the personality correlates for stroke remains problematic. Due to the similarities with the aetiology of heart disease, stroke may be caused through the same personality characteristics, i.e. *Emotional Lability*, *Type A Behaviour* and *Behavioural Control*.[5] On the other hand, stroke causes brain tissue damage, reduction in the quality of life and other secondary health problems. Hence, large changes in personality variables and the onset of other diseases as a consequence of the initial disease could be expected particularly for stroke patients. In the entire subject sample, only seven stroke patients reported suffering from stroke alone.

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Personality traits as specific and general predictors of disease

Sanderman and Ranchor (1997) distinguish the *specificity* approach—specific traits link to specific diseases—from the *generality* approach—personality factors influence general susceptibility to disease. The present data provide some support for both approaches, but the strongest feature of the data was the fairly general association between *Emotional Lability* and disease-proneness. The association is consistent with several alternative causal hypotheses. First, personality traits that correlated with Emotional Lability (e.g. Depression, Anxiety) might be associated with immune system impairments (for instance a lower sIgA-baseline, secretion rate; see Hennig, Pössel, & Netter, 1996), which lead to general vulnerability to disease. One of the salient factors of emotional lability or neuroticism is the experience of stress, i.e. self-ratings on Neuroticism may reflect the subject's experience of being frequently stressed (Hennig & Netter, 1997, p. 154). Therefore, the immune impairments may be the result of exposure to stress, although the evidence in this area is conflicting (Koh, 1998; Netter, Müller, Hennig, & Rohrmann, 1999). Another possible mediator is increased sympathetic nervous system activity, which has been linked to abnormalities in glucose metabolism and risk of CHD (Schneiderman et al., 2001). Second, more neurotic individuals may be especially 'complaint-prone': neuroticism is more reliably associated with subjective symptoms than with objective pathology (Stone & Costa, 1990). Despite the empirical evidence that this tendency seems not to distort survey data over diseases that require medical diagnosis (Kobasa et al., 1981; Robinson et al., 1997), there is still scope for biasing of self-report data, and the Stone and Costa (1990) hypothesis merits serious attention. Third, emotional lability may be a secondary consequence of distress provoked by the illness: prospective data are required to distinguish this hypothesis from the possibility that personality is a causal influence on illness. Fourth, emotional lability may have indirect effects mediated by 'health behaviours' that promote wellness or disease. For example, neurotic individuals are more likely to smoke, and depressed individuals may be neglectful of their health. However, empirical studies show that the links between negative affect and health behaviours are complex, and moderate anxiety and depression may even have beneficial effects such as care-seeking (Mayne, 1999).

Factors 2–5 showed rather more specific patterns of association with disease status, but as previously described, these patterns did not fully support findings from previous studies. For example, the Type A factor related not just to CHD, but various other diseases also, and the diseases associated with *Behavioural Control* did not include cancer. Again, associations may reflect causal effects of illness on personality as much as the reverse possibility. For example, chronic gastric disturbance might elicit feelings of irritability and expressed anger, leading to lowered *Behavioural Control* scores. The correlations of the Factors 2–5 with disease are very low, but this may be attributed to the low prevalence for some of the diseases.

In conclusion, we have noted that the cross-sectional design of the study imposes constraints on interpretation of the data. Nevertheless, the present data have some general implications for research on personality and health. First, it is important to use personality constructs with a strong psychometric basis, and to discriminate the influence of general emotionality from more specific factors. Second, despite the fact that the reported effect sizes were low, the data reaffirm that various personality factors appear to play some role in physical illness, and extend existing data by demonstrating multiple links between personality traits and diseases. Third, different personality traits may exert multiple general and illness-specific effects on health, but further work is required to verify the reliability of the new findings, and to differentiate possible causal explanations.

Notes

1. These numbers differ slightly from former publications. In preparation of the follow-up study our list of names and addresses was checked. By doing that we found 13 males and 20 females whose gender was coded erroneously.
2. The same is true for prevalence rates that exceed 50 per cent markedly.
3. When extracting only three factors, Psychoticism and the constructs Internal and External Locus of Control have small communalities, but the remaining three factors showed nearly the same structure.

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In contrast, the five factors solution presented in Table 2 showed satisfactorily high communalities for all variables.

4. Semi-partial correlations of diabetes, stroke, adiposity and heart diseases are hardly interpretable because of the non-significant Pearson correlations and the low levels of statistical significance and effect size.

5. Additionally, some of the main biological risk factors (diseases of Factor 12) of vascular disease (like stroke and CHD) have been included as predictors. If the relation of personality is partly mediated through these precursor diseases high semi-partial correlations cannot be expected.

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