Happiness and Health: Environmental and Genetic Contributions to the Relationship Between Subjective Well-Being, Perceived Health, and Somatic Illness

Espen Røysamb
Norwegian Institute of Public Health and University of Oslo

Kristian Tambs and Ted Reichborn-Kjennerud
Norwegian Institute of Public Health

Michael C. Neale
Virginia Institute of Psychiatric and Behavioral Genetics

Jennifer R. Harris
Norwegian Institute of Public Health

The aim was to identify genetic and environmental influences on the covariances between subjective well-being (SWB), perceived health, and somatic illness. Analyses were based on 6,576 Norwegian twins aged 18–31. Heritabilities ranged from .24 to .66. SWB correlated .50 with perceived health, −.25 with musculoskeletal pain, and −.07 with allergy. Common genetic factors accounted for 45%–60% of associations. SWB and perceived health was to a high extent influenced by the same genes ($r_g = .72$ and .82 for males and females, respectively). For SWB and musculoskeletal pain, $r_g = −.29$ and −.42 for males and females, respectively. Effects were partly sex specific. Environmental factors shared by twins did not affect the covariances. Results support a differentiated view of SWB–health relations, and imply that both genes and environment play important roles in the associations between well-being and health.

Are healthy people happier, and if so, why? As research on subjective well-being (SWB) has flourished in recent years, the relationship between well-being and physical health has received increased attention (e.g., Diener, Suh, Lucas, & Smith, 1999; Kahneman, Diener, & Schwarz, 1999; Kwan, Bond, & Singelis, 1997; Veenhoven, 1996). Physical health is seen as an important source of human well-being, and a number of studies have reported empirical relations between SWB and different health dimensions (Brief, Butcher, George, & Link, 1993; Feist, Bodner, Jacobs, & Miles, 1995; Larson, 1978; Watten, Vassend, Myhrer, & Syversen, 1997; Woodruff & Conway, 1992). However, much uncertainty remains regarding the precise nature of the SWB–health relationship. Recently, Diener et al. (1999) called for more methodologically sophisticated designs to identify the nature of the relations between SWB and different correlates. The present study set out to disentangle the underlying genetic and environmental factors influencing the association between well-being and health among young adults by analyzing data from a population-based twin registry.

Genetic Factors, Well-Being, and Health

Twin studies have demonstrated that genetic factors account for substantial amounts of individual variation in well-being and health conditions. Heritability estimates for SWB typically range from .25 to .55 (Bergeman, Plomin, Pedersen, & McClearn, 1991; Harris, Pedersen, Stacey, McClearn, & Nesselroade, 1992; Lykken & Tellegen, 1996; Røysamb, Harris, Magnus, Vittersø, & Tambs, 2002). Of interest, a recent observational study yielded similar estimates for SWB among chimpanzees (Weiss, King, & Enns, 2002). Corresponding heritabilities have been reported for personality traits such as Neuroticism and Extraversion (e.g., Loehlin & Martin, 2001) and mental health problems (Kendler, 1993).

Likewise, substantial genetic effects have been reported for a number of somatic disorders and health-related conditions. For example, genetic factors have been found to account for a considerable amount of the variance in self-rated health (Christensen, Holm, McGuie, Corder, & Vaupel, 1999; Harris et al., 1992; Kendler, Myers, & Neale, 2000), atopic disorders (Harris, Magnus, Samuelsen, & Tambs, 1997; Huss & Huss, 2000; Ono, 2000), back/neck pain (Reichborn-Kjennerud et al., 2002), cardiovascular...
disorders (Turner, Cardon, & Hewitt, 1995), and total mortality (Yashin, Iachine, & Harris, 1999).

Despite the mounting evidence that genes play an important role in the etiology of well-being, personality, mental health, and physical illness, genetic and environmental influences on the covariance between SWB and physical health have been largely unexplored. A literature search in PsycINFO revealed a total of 882 articles (1980–2002) including both the terms “life satisfaction/well-being” and “physical health/physical illness/self-rated health.” However, when “twin/heritability” was included as criterion, three articles remained (Harris et al., 1992; Johansson et al., 2001; Lichtenstein, Gatz, Pedersen, Berg, & McClearn, 1996). Only one of these studies (Harris et al., 1992) investigated explicitly the genetic and environmental contribution to the interrelation between SWB and health. Among people older than 65 both genetic and environmental contributions substantially contributed to the relationship between perceived health and life satisfaction. However, in the younger group the association could be entirely accounted for by environmental factors. These findings raise questions regarding possibly divergent mechanisms in different age groups.

Thus, the current state of knowledge includes solid evidence for both environmental and genetic influences on well-being and health variables, and evidence for empirical relationships between well-being and health, yet very limited knowledge on the way and degree to which genetic and environmental factors operate in generating these relationships. A potential finding of a substantial effect of common genetic factors in SWB and health-related conditions would be important for moving the field ahead in terms of identifying the specific physiological and behavioral mechanisms through which the association develops.

Previous studies have reported partially sex-specific predictors and mechanisms involved in generating and sustaining SWB. For example, in the environmental realm, the effect of life events on SWB seems to be different for men and women (Forest, 1996; French, Gekoski, & Knox, 1995), and sex differences have been reported for the association between marital satisfaction and relationship awareness (Acitelli, 1992). In the genetic sphere, Roysamb et al. (2002) found evidence that partly different genetic factors influence SWB in men and women, and Eaves, Heath, Neale, Hewitt, and Martin (1998) reported sex differences in the magnitude of genetic effects for neuroticism. However, little is known about the ways in which possible sex differences develop. By investigating the interrelations between SWB and physical health from a genetically informed perspective it is possible to shed new light on the mechanisms involved.

Dimensions of Health
Clearly, physical health is a multidimensional construct, comprising a number of different health-related conditions. An important distinction is that between perceived global health and specific somatic problems (Vassend, 1994; Watten et al., 1997). Perceived or self-rated health has typically been regarded as a sum of a person’s medical health status and its functional consequences (Manderbacka, Lahelma, & Martikainen, 1998; Moum, 1992). However, Brief et al. (1993) reported evidence of perceived health as partially influenced by personality factors. Moreover, whereas previous studies typically reported relatively strong relations between SWB and perceived health, the associations seem weaker and less stable when external or “objective” measures of specific disorders are used (Diener et al., 1997; Watten et al., 1997). The consistent finding of a relatively strong relation between SWB and perceived health suggests that there might be common factors—genetic and environmental—that influence these two phenomena independently of actual health status. Theoretically, the relation between SWB and perceived health can be partially explained by a top-down perspective (Feist et al., 1995). In this framework SWB represents a general tendency to hold a positive, or not so positive, life view, which also influences the perception of different aspects of life such as health. To the extent that there is a substantial genetic influence on the disposition to evaluate one’s life positively, it is reasonable to hypothesize that this genetic factor will influence perceived health as well.

Musculoskeletal pain is a major health problem in the industrialized world, often starting at a young age and occurring with increasing prevalence during young adulthood (Andersson, 1997; Linton, 2000). Typically, musculoskeletal pain comprises pain in the back, neck, shoulder, and head. In a recent population study, 25% of adult individuals reported neck or back pain to be substantial, and 19% had taken a sick leave because of spinal pain during the last year (Linton, Hellsing, & Halden, 1998). An estimated 5%–10% of adults have a disabling level of neck pain (Croft et al., 2001). In a meta-analysis, Linton (2000) concluded that there was a clear link between distress, anxiety, mood, emotions, and the onset of both acute and chronic pain. Common genetic factors contributing to the relationship between back/neck pain and anxiety/depression have been reported (Reichborn-Kjennerud et al., 2002), and the serotonin and noradrenaline systems of the brain appear to be involved in both psychological distress and pain sensitivity (Atkinson et al., 1999; Besson, 1999; Nemeroff, 1998).

Allergic disorders such as asthma, allergy, and hay fever represent another illness category that has a relatively high prevalence among young adults, and clinical studies have linked allergic symptoms to a variety of psychological symptoms (e.g., Bell, Jansoski, & King, 1991; Marshall, 1993). Moreover, allergies tend to cluster in families; genetic factors seem to play a major role in the disposition to develop an allergic disorder (Harris et al., 1997), and common genes influencing both depression and allergy have been suggested (Wamboldt et al., 2000). There is evidence that certain immune mediators, notably cytokines, can trigger symptoms similar to depression, such as concentration and sleep problems, and lethargy (e.g., Bell, Miller & Schwartz, 1992). Presumably, allergic reactions would release cytokines that might then lead to depressive symptoms (Wamboldt et al., 2000). To the extent that such problems contribute to reduced well-being, there is reason to expect genetic influences on a potential association between allergic disorders and low levels of SWB.

Twin Analysis
Data collected from identical and fraternal twins provide not only estimates of heritability and environmental effects on single variables but, more interestingly, estimates of the effects of genes and environment on the bi- and multivariate associations between different variables (Neale & Cardon, 1992). By means of twin data, four general sources of variance and covariance can be
estimated: Additive genetic factors (A), nonadditive genetic factors (D for “dominance”), common environment (C), and non-shared environment (E). Additive genetic variance comprises the total summative effect of multiple genes. Nonadditive genetic variance refers to intralocus genetic interactions (dominance) or to interlocus interaction (epistasis). Both forms of nonadditive effects represent interaction in the sense that the expression of one genetic variant depends on the presence of other genetic variants. Heritability comprises both additive and nonadditive genetic effects, and refers to the part of the total variance that is due to genetic factors. Common, or shared, environment comprises all environmental factors that contribute to twin similarity, that is, all events that are effectively common. Nonshared, or unique, environment refers to the part of environmental influences that contributes to twin dissimilarity, including random error variance. It should be noted that a certain environmental event is not common or nonshared, per se, but might be either depending on its effect on twin similarity/dissimilarity (e.g., Neale & Cardon, 1992; Plomin, DeFries, McClearn, & Rutter, 1997). With data from fraternal twins of opposite sex it is possible to investigate whether the genetic and environmental factors differ in magnitude across gender, and to what extent the factors contributing to twin similarity are the same for men and women.

To summarize the aims of the present study: First, we wanted to identify the phenotypic associations between SWB, general perceived health, musculoskeletal pain, and allergic disorders among young adults. A second aim was to estimate heritabilities for well-being and health measures, and to identify possible sex differences. Third, a major aim was to estimate the effects of genetic and environmental factors on the interrelations between SWB and the health measures. That is, to what extent is the genetic influence on well-being associated with the genetic influence on the different health measures? Likewise, to what extent do the environmental influences correlate across phenotypes? It was hypothesized that genes would contribute substantially to the observed associations.

Method

Sample

The data were collected in Norway in 1998–1999. On the basis of the Norwegian Birth Registry, comprising all births in Norway since January 1, 1967, a total of 12,700 like- and unlike-sexed twins aged 18–31 were mailed a questionnaire. Responses were obtained from 8,045 subjects, comprising 3,334 complete pairs and 1,377 singletons, representing an individual response rate of 63% and a pairwise response rate of 53%. This data collection was a follow-up and expansion of a corresponding survey in 1992, in which a total of 5,864 twins aged 18–25 participated. The present study is based on data from 6,668 twins (full pairs), of which 59% also participated in the 1992 survey. The number of complete pairs was: 526 monozygotic (MZ) males, 397 dizygotic (DZ) males, 777 MZ females, 655 DZ females, and 979 DZ pairs of unlike sex (DZU). Of the total sample of 3,334 complete pairs, a total of 3,288 pairs (98.6%) had valid responses on all variables and constituted the final sample on which the analyses were performed. Zygosity was determined on the basis of seven analyses were performed. Zygosity was determined on the basis of seven

analyses were performed. Zygosity was determined on the basis of seven
sample of 3,334 complete pairs, a total of 3,288 pairs (98.6%) had valid
526 monozygotic (MZ) males, 397 dizygotic (DZ) males, 777 MZ females,
also participated in the 1992 survey. The number of complete pairs was:

present study is based on data from 6,668 twins (full pairs), of which 59% 
in 1992, in which a total of 5,864 twins aged 18
individual response rate of 63% and a pairwise response rate of 53%. This
comprising 3,334 complete pairs and 1,377 singletons, representing an

measurement plays a role, and DZ correlations less than half the MZ correla-
tions higher than half the MZ correlations indicates that common envi-
ronment plays a role, and DZ correlations less than half the MZ correla-

Measurement

The SWB index (Moum, Ess, Sarfes, & Tambs, 1990; Raysamb et
al., 2002) comprises four items, for example, “When you think about your
life at present, would you say you are mostly satisfied with your life, or
mostly dissatisfied?” (six response categories) and “Are you usually happy
or dejected?” (five response categories). Because of differences in response
categories (ranging from four to six), all items were transformed into 0–10
scales to provide a common metric and equal weighting for each item, and
a descriptively eloquent scaling of the mean-score index (transformation
algorithm: X = [Y − 1] × 10/[Z − 1], where X is new score, Y is original
score, and Z is number of response categories). A high score indicates high
level of well-being. Cronbach’s a for the index was .71.

Perceived health was measured with the following item: “What is your
health like, at present?” (measured on a scale from 1 = poor to 4 = very
good). This single item measure of perceived health, and highly similar
versions, has been used in several studies and has been shown to have
acceptable psychometric properties (e.g., Bardage, Isacson, & Pedersen,
2001; Krause & Jay, 1994).

Musculoskeletal pain was measured by four items referring to having
experienced typical symptoms: back pain, neck and shoulder pain, head-
ache, and muscular pain (e.g., Andersson, 1997; Linton, 2000). Endorse-
ment was coded 1 and nonendorsement was coded 0. The four items were
summed to a total index of musculoskeletal pain, with a Cronbach’s a of
.79.

Allergy was measured using five items inquiring about typical disorders:
asthma, hay fever, hives, atopic eczema, and other eczema (e.g., Bell et al.,
1991; Kaplan & Mascie, 1989). Endorsement was coded 1 and nonen-
forcement was coded 0. The five items were summed to a total allergy
index, with a Cronbach’s a of .67. The somatic illness items referred to
having experienced the symptoms without any specific time limit. How-
ever, respondents were allowed to report that they no longer experienced
the symptoms, and such a response was coded as nonendorsement. It
should be noted that because of the dichotomous nature of the items in
allergy and musculoskeletal pain, the alphas were based on polychoric
correlations.

Analyses

Structural equation modeling by means of Mx (Neale, Boker, Xie, &
Maes, 1999) was used to test alternative models and to estimate the genetic
and environmental influences on phenotypic variances and covariances.
These models are widely used to analyze twin data and are described in
detail elsewhere (e.g., Kendler, 1993; Neale & Cardon, 1992).

Briefly, estimation of the relative contribution of different latent factors
allows us to determine (a) to what extent variation in the phenotypic
measures are familial, and (b) the relative contribution of genetic and
common environmental effects to familial aggregation. Expected covari-
ance matrices for MZ and DZ twins are based on an additive genetic
co-twin correlation of 1.0 for MZ and 0.5 for DZ twins, a nonadditive
genetic correlation of 1.0 for MZ and 0.25 for DZ twins, and a common
environment correlation of 1.0 for both MZ and DZ twins. The nonshared
environment factor (E) is by definition not contributing to twin similarity.
These specific co-twin correlations are imposed as fixed parameters in the
different zygosity groups during the modeling analyses.

The observed pattern of MZ versus DZ correlations indicates the degree
to which the different factors (A, C, D, E) are present. Given that all factors
except E can contribute to twin similarity, an MZ correlation below unity
implies the presence of nonshared environmental effects. Because MZ
twins share all their segregating genes and DZ twins on average share 50%
of their segregating genes, a higher MZ than DZ correlation implies the
presence of genetic effects. Moreover, a preliminary estimate of heritability
is provided by the formula 2x(MZcorr − DZcorr). A pattern of DZ corre-
lations higher than half the MZ correlations indicates that common envi-
ronment plays a role, and DZ correlations less than half the MZ correla-
tions suggests that the genetic effect might be partly nonadditive (see, e.g., Neale & Cardon, 1992; Plomin et al., 1997).

By means of multivariate modeling, genetic and environmental influences on the covariances between variables can be estimated. That is, the degree to which the same or different genetic and environmental factors influence different observed variables can be estimated (see, e.g., Neale & Cardon, 1992). Moreover, sex-specific effects may be investigated when data from both same-sex and opposite-sex twins are available (Neale & Cardon, 1992). Under the quantitative sex-limitation model, the same factors contribute to variation in the phenotype under study; however, the magnitude of the genetic and environmental effects is allowed to vary across sexes. The qualitative sex-limitation model allows for sex differences in the sources of variance, as well as in the magnitude of estimates for men and women.

For the present modeling procedures the data were summarized into twin–co-twin correlation matrices for each of the five Zygosity × Sex groups. Because of the categorical nature of the health measures, polychoric correlations were calculated using PRELIS (Jöreskog & Sörbom, 1996). Under the liability-threshold model, an underlying continuous liability is assumed. To avoid potential problems involved in the calculation of asymptotic covariance matrices for polyserial correlations (i.e., correlations between categorical and continuous variables), the measure of SWB was polythemedized into categories so as to yield true polychoric correlations (see, e.g., Kendler et al., 2000).

Multivariate models may be parameterized in different ways. A Cholesky decomposition was used (Loehlin, 1996), which parameterizes one set of genetic and environmental factors (e.g., A1 and E1) influencing all the observed variables, a second set of factors (e.g., A2 and E2) influencing all but the first observed variable, and so on, until a final set of factors (e.g., Aq and Eq) influences the last variable in the chosen order. When correctly constrained, the order of variables does not influence the total fit of the model, nor the parameter estimates (Neale, Roysamb, & Jacobson, 2003).

Furthermore, as recommended by Loehlin (1996), the Cholesky parameterization was translated into a correlated factor model. This model explicates genetic and environmental correlations, that is, correlations between the genetic factors influencing each phenotype, and correlations between the environmental factors (Loehlin, 1996; Neale & Cardon, 1992). Finally we also tested an independent factor model, corresponding to a confirmatory factor analysis with both a genetic and an environmental factor structure, to separate the variances of the variables into common and specific parts.

A series of models was tested, beginning with a relaxed model and in a stepwise manner testing alternative constraints. Model fit was evaluated by the chi-square statistic, in addition to the root-mean-square error of approximation (RMSEA) and Akaike’s information criterion (AIC). To test for the significance of specific parameters the chi-square-difference test for nested models was used (see, e.g., Bollen, 1989; Neale & Cardon, 1992).

Results

Means and Prevalences

The mean score on the SWB index (0–10 scale) was 7.15 (SD = 1.61). Men scored slightly but significantly (p < .01) higher than women, with scores of 7.33 and 7.02, respectively. There were no gender differences in variances. For perceived health (1–4 scale) the mean score was 3.35 (SD = 0.63). In total, 43.1% reported to have very good health, 49.2% reported having good health, 7.0% had not quite good health, and 0.7% reported having poor health. Men reported slightly, and significantly (p < .01), better health than women.

The prevalence of musculoskeletal pain ranged from 4.0% (muscular pain) to 18.9% (back pain). For the total index, 67.8% reported no symptoms, 21.3% reported one symptom, 7.7% reported two symptoms, 2.8% reported three symptoms, and 0.5% reported all four types of pain. Women reported more symptoms than men (p < .01). For allergic disorders, prevalences of single items ranged from 6.0% (atopic eczema) to 13.9% (hay fever). For the total index, 64.7% reported no symptoms, 25.6% reported one symptom, 7.4% reported two symptoms, 1.8% reported three symptoms, and 0.4% reported four or five symptoms. Women reported slightly more symptoms than men (p < .01).

Although significant gender differences were observed for all indices, the differences were modest. Gender explained less than 1% of the variance in SWB, perceived health, and allergy, and 2.9% of the variance in musculoskeletal pain. Age accounted for less than 0.5% of the variance for all the measures.

Correlations Across Variables and Between Twins

Table 1 shows the phenotypic correlations between all measures. SWB correlated rather strongly with perceived health, and weaker, but still substantially, with musculoskeletal pain. In contrast, the association between SWB and allergy was virtually negligible. To test the unique relation between SWB and perceived health, independent of the specific somatic disorders, a partial correlation was calculated, yielding r = .40 (p < .01). Likewise, the partial correlation between SWB and musculoskeletal pain was −.14 (p < .01) after controlling for the effect of perceived health. The corresponding correlation between SWB and allergy was reduced to a nonsignificant r = −.02.

Table 2 shows co-twin correlations (i.e., correlations between twin1 and twin2 for each measure) and cross-twin cross-trait (i.e., trait1–twin1 vs. trait2–twin2) correlations. Co-twin correlations were generally higher among MZ twins than among DZ twins, though varying across phenotypes. Thus, genetic variance is indicated for all variables. The correlations among DZ twins of same sex were higher than those of twins of unlike sex, thus indicating potential sex-specific effects.

The cross-twin cross-trait correlations were, as expected, generally lower than the same-trait correlations. However, the same pattern was found with greater MZ than DZ correlations. This suggests that genetic factors contribute to the associations between the measures. Allergy yielded the highest co-twin MZ correlations, yet, there were virtually no cross-relations with SWB.

Univariate Models

To identify the factors involved in generating the observed pattern of correlations, a number of theoretical models were tested. The first phase involved the testing of alternative univariate models, that is, separate analyses for each of the phenotypes. The model fitting strategy involved starting with the least restrictive model; a model comprising sex-specific factors for A, C, and E factors, followed by alternative models with specific constraints.

1 It could be argued that musculoskeletal pain and allergy only represent two of a number of relevant disorders. Therefore an additional analysis was conducted, in which a broad spectrum of 15 single health problems, typically with low prevalences (e.g., diabetes, epilepsy) were controlled for. The partial correlation between SWB and perceived health remained at a similar level (r = .39, p < .01).
These alternatives involved constraining effects to zero (e.g., no C effect) and including nonadditive genetic effects (i.e., a D factor). With regard to sex-specific effects, the first set of models allowed for qualitative sex limitation; next the constraint of identical genetic factors across sex was imposed (i.e., quantitative sex limitation). Finally, the constraint of identical magnitudes of effects for males and females was tested (i.e., no sex limitation). For each phenotype four models (ACE/ADE/AE/CE) within each of the three sex-limitation categories were tested. Models were compared by the nested model chi-square test and AIC.

The best-fitting models were as follows: For SWB, the model comprised A (additive genetic) and E (nonshared environment) effects, and there was evidence of qualitative sex limitation, $\chi^2(2, N = 3,288) = 5.06$ (AIC = 1.06, RMSEA = 0.00). As a comparison, a model containing only environmental effects (C and E), with quantitative sex limitation, yielded much worse fit, $\chi^2(3, N = 3,288) = 58.84$ (AIC = 52.83, RMSEA = 0.00), thus clearly showing the presence of genetic factors in SWB. For perceived health, again only A and E effects were significant, and there were no significant sex effects, $\chi^2(4, N = 3,288) = 4.38$ (AIC = 3.62, RMSEA = 0.00). Similarly, for musculoskeletal pain the best-fitting model included A and E factors, and no sex effects, $\chi^2(4, N = 3,288) = 3.73$ (AIC = 4.25, RMSEA = 0.00). Finally, the best-fitting model for allergy included A, C, and E factors and quantitative sex limitation, $\chi^2(1, N = 3,288) = 0.37$ (AIC = 1.63, RMSEA = 0.00).

Multivariate Models
The univariate modeling results were useful in selecting the multivariate models to be tested in the next phase of analysis. In addition to addressing the question of covariance sources, the multivariate models are more powerful in providing precise estimates of the latent effects on each of the phenotypes. Whereas the univariate models are based solely on the cross-twin same-trait correlations, the multivariate models represent a full information approach in which both the cross-twin same-trait and the cross-twin cross-trait correlations in each zygosity group are taken into account in the estimation process.

Because allergy was virtually unrelated to SWB, we chose to omit this variable from the multivariate analyses. Moreover, on the basis of the univariate findings, only A and E factors were included. Given evidence of sex limitation for SWB, different types of sex effects were specified and tested. The first model, which allowed for qualitative sex limitation, yielded good fit, $\chi^2(60, N = 3,288) = 85.92$ (AIC = −34.08, RMSEA = 0.00). The second model imposed the constraint of same genetic factors for men and women, yet allowed for different effect sizes (quantitative sex limitation), and yielded reduced fit, $\chi^2(63, N = 3,288) = 96.83$ (AIC = −29.17, RMSEA = 0.00); $\Delta \chi^2(3, N = 3,288) = 10.91$, $p < .05$. Third, a model with no sex effects yielded a further decrease in fit, $\chi^2(66, N = 3,288) = 108.29$ (AIC = −23.71, RMSEA = 0.00); $\Delta \chi^2(3, N = 3,288) = 11.46$, $p < .01$.

Allowing for qualitative sex limitation (model 1) for the genetic factors implies that the genetic correlations between phenotypes may vary between genders; however, the environmental correlation matrix was constrained to be invariant across sex. A fourth model was tested in which the E structure was also allowed to vary, but this model did not fit significantly better, $\Delta \chi^2(3, N = 3,288) = 2.72$, ns. In summary, the first model, which included qualitative sex limitation for the genetic factors, was best able to account for the observed correlation structure.

Figure 1 depicts the best fitting model—in the form of a correlated factor model. Heritabilities are obtained by squaring the effects from the latent A and E factors. Thus, heritability estimates for SWB were .44 (confidence interval [CI] = .39, .50) for women and .44 (CI = .38, .50) for men. Correspondingly, heritability for perceived health was .27 (CI = .22, .33) for women and .38 (CI = .28, .45) for men. Finally, heritability for musculoskeletal pain was .31 (CI = .25, .38) for women and .24 (CI = .17, .31) for men. Heritability estimates for allergy (based on the univariate models) were .49 (CI = .31, .64) for females and .66 (CI = .59, .72) for...
males. In addition, among females common environment accounted for .20 (CI .06, .36) of the variance in allergy.

The two-headed arrows in Figure 1 represent genetic and environmental correlations. It should be noted that the genetic correlations are roughly twice the size of the environmental correlations, and in particular the genetic correlations between SWB and perceived health were high.

The cross-sex genetic correlations (not shown in Figure 1) were .69 (CI = .40, .96) for SWB, .84 (CI = .45, .96) for perceived health, and .95 (CI = .59, .99) for musculoskeletal pain. Thus, whereas the univariate models only yielded significant sex effects for SWB, the full model, with an integration of all information, revealed small but significant sex differences for all measures. It should be noted, however, that the upper bound of the confidence intervals were close to unity, and that only for SWB was the female–male genetic correlation substantially lower than unity. Although the cross-sex genetic correlation was lowest for SWB, the total heritability was equal for men and women.² Thus, partially different genetic factors seem to be operating, yet their total effect is similar in magnitude.

The correlations as depicted in Figure 1 reflect the degree to which the genetic, or environmental, factor influencing one phenotype is associated with the corresponding factor influencing another phenotype. Moreover, to complement the picture, the genetic and environmental contributions to the total estimated phenotypic correlations were also calculated. Figure 2 depicts the estimated phenotypic correlations partitioned into contributions from genetic and environmental factors. Genetic factors accounted for 45%–60% of the correlations. Thus, it should be noted that whereas the genetic correlations (i.e., correlations between

² It should be noted that heritability estimates for the SWB index, based on a univariate model, have previously been reported in Røysamb et al. (2002). Those estimates were based on the data set collected in 1992, and included 59% of the responders who participated in the 1998–1999 collection. The previous heritability estimates (i.e., based on roughly half the current sample, and data collected 6 years earlier) were .46 (males) and .54 (females).
genetic factors) were in general substantially higher than the environmental correlations, the total contribution of genetic variance to the phenotypic correlations was roughly equal to the contribution of the environmental factors.

Having focused on the bivariate relations so far, the final analysis addressed the issue of different contributions to the total variance of the three measures. That is, when taking all three measures into account simultaneously, what amount of variance in each is due to common and specific genetic and environmental factors? On the basis of the best fitting model, previously described above, an independent factor model was tested. This corresponds to a factor analysis with the additional feature of distinguishing between genetic and environmental factors. Figure 3 shows the partitioned variance of SWB, perceived health, and musculoskeletal pain.

For SWB, roughly half (males) or two thirds (females) of the genetic variance was accounted for by a factor common to all three measures. That is, when taking all three measures into account simultaneously, what amount of variance in each is due to common and specific genetic and environmental factors? On the basis of the best fitting model, previously described above, an independent factor model was tested. This corresponds to a factor analysis with the additional feature of distinguishing between genetic and environmental factors. Figure 3 shows the partitioned variance of SWB, perceived health, and musculoskeletal pain.

For SWB, roughly half (males) or two thirds (females) of the genetic variance was accounted for by a factor common to all three measures, yet there was also substantial genetic variance that was unrelated to the other two measures. In contrast, most of the environmental variance was specific for SWB, only a small portion was accounted for by a common factor. All of the genetic variance in perceived health was accounted for by the common factor, and the environmental variance was explained by roughly equal amounts of common and specific variance. Finally, for musculoskeletal pain the larger part of both the genetic and environmental variance was specific.

Discussion

The study set out to shed new light on the relationship between SWB, perceived health, and somatic illness. Whereas several previous studies have shown empirically that such relations exist, the nature of these associations has remained unclear. Our main aim was to contribute to this field by disentangling the SWB–health associations into genetic and environmental effects.

With regard to our opening question asking whether healthy people are happier, our findings suggest that, among young adults, (a) people who perceive their health to be good are also happier, (b) people without musculoskeletal pain are a little happier, and (c) people with an allergic disorder are just about as happy as those without. Thus, our findings support a divergent perspective on the SWB–health relation where some, but not all, dimensions of physical health are associated with well-being.

The finding of a rather strong association between well-being and general perceived health is congruent with results from several previous studies (Brief et al., 1993; Larson, 1978). Perceived health appeared to be more strongly related to well-being than to somatic disorders such as musculoskeletal pain and allergy. Substantial heritabilities were found for both well-being and perceived health, which accords with previous studies (Christensen et al., 1999; Lykken & Tellegen, 1996; Røysamb et al., 2002). Moreover, and more importantly, the genes that influence well-being are to a high extent the same genes that influence perceived health. Harris et al. (1992) reported similar findings among elderly people, but no
common genetic effect among the younger population. The present results show that a mechanism previously documented only in the elderly population, also seems to be present among young adults.

At the neurophysiological level, this finding might reflect common mechanisms involving the neurotransmitters serotonin and norepinephrine. Several lines of research have linked depressive symptoms with dysfunctional transmitter systems (see, e.g., Berenbaum, Raghavan, Le, Vernon, & Gomez, 1999), and Jang et al. (2001) found evidence of a serotonin transporter gene (5-HTTLPR) to be involved in the genetic influence on neuroticism. Thus, given that both depression and neuroticism are associated with low levels of well-being (Diener et al., 1999; DeNeve & Cooper, 1998), there is reason to believe that some similar transmitter mechanisms are involved in the way a common set of genes affect well-being and perceived health.

At a psychological level, the strong genetic correlation lends support to the notion of a top-down process. That is, SWB is conceived of as a general tendency to hold a positive life view, which also affects the perception of different aspects of life such as health (Feist et al., 1995; Headey, Veenhoven, & Wearing, 1991). Given that this tendency is partly constituted by genetic factors, then the same genes appear to influence evaluations of health. Within this framework our results imply that the process of a general tendency to a positive life view transforming into positive evaluations in subdomains of life is highly influenced by genetic dispositions.

Thus, one important implication of our findings concerns the very nature of perceived health. In contrast to the traditional perspective that perceived health reflects a cognitive summation of medical health status (e.g., Manderbacka et al., 1998), the present results testify to the notion of perceived health as strongly associated with general outlook tendencies. Perceptions of health appear substantially colored by a person’s view of life as good—or not so good—and this coloring process is to a certain extent driven by common genes.

However, this does not imply that perceived health is unrelated to somatic illness. Of interest, Bardage et al. (2001) showed that low self-rated health uniquely predicted later mortality, and in a similar vein Danner, Snowdon, and Friesen (2001) found that positive emotionality in young adulthood predicted longevity decades later. In conjunction with our findings, we may speculate that present perceived health is more about general well-being than about current health status, but nevertheless that there are long-term effects of perceived health and well-being on physical health—possibly through both immune system functioning and behavioral pathways.

With regard to the association between well-being and musculoskeletal pain, again there was evidence of genetic factors playing an important role. Our findings are in accord with reports of genetic contributions to relations between psychological problems and back/neck pain (Reichborn-Kjennerud et al., 2002). Possibly, some of the same neurophysiological mechanisms as previously

---

**Figure 3.** Genetic and environmental factor analysis of subjective well-being, perceived health, and musculoskeletal pain. Total variance for each phenotype is partitioned into variance accounted for by a common (for all three measures) genetic factor, specific (for each measure) genetic factors, a common environmental factor, and specific environmental factors. SWB = subjective well-being.
discussed are implicated, that is, a genetic liability to dysfunctional serotonin and norepinephrine systems might be involved in the relationship.

In contrast to the associations previously discussed, the relation between allergic disorders and well-being was negligible. This is in discord with some previous findings involving psychological distress, but it should be noted that previous reports partly involve small effect sizes (e.g., Wamboldt et al., 2000). Moreover, given the chronic nature of atopic disorders, our findings might reflect the long-term adaptation to a situation that initially appears difficult. Thus, we cannot reject the possibility that onset of allergic disorders has an immediate impact on SWB, nor the possibility that current psychological distress has an impact on allergic symptoms. However, in the long-term perspective, having an atopic disorder seems unrelated to well-being. To some extent the divergent associations for musculoskeletal pain and allergy are remarkable, and indicate that whereas the neurophysiological systems involved in musculoskeletal pain are partly involved in well-being, those involved in allergic disorders are largely unrelated to well-being.

Our findings suggest that the environmental factors affecting SWB to a high extent are specific for well-being, in the sense that they are different from the environmental factors influencing the health measures. Thus, researchers looking for environmental factors that affect SWB among young adults would probably do best to explore outside the health domain. This does not rule out the possibility that a certain health-related intervention might also influence SWB, but among the naturally occurring variation in environmental sources of physical health the effects on SWB appear relatively small.

The pattern of genetic and environmental effects was similar for men and women. However, there was also evidence of sex-specific effects. In particular there was evidence for partly different sets of genetic factors affecting well-being in women and men. One tentative explanation is that the process of genetic expression reflects an interplay between genetic dispositions and cultural value systems. Given that partly different ideals, norms, and values prevail for women and men (Larsen & Cutler, 1996), and that well-being to some extent is related to the match between “actual self” and the culturally prescribed “ideal self” (Mallard, Lance, & Michalos, 1997; Markus & Nurius, 1986; Pavot & Diener, 1993), it makes sense that differing sets of genes indirectly influence well-being. That is, a set of genes that predispose for certain physical or psychological characteristics that are highly valued for women, but not for men, might contribute to well-being only among women. The current design and data do not provide for empirical testing of such a model. Yet, the notion of genetic expression as moderated by cultural values and norms is an important direction for future research.

In addition to the limitations and caveats already mentioned, some other issues should be raised. One ostensible weakness concerns the measures used. Self-reported measures of somatic disorders do not necessarily correspond perfectly to external or “objective” measures. However, the observed correlational structure, including only moderate associations between the somatic illness variables and perceived health, testifies to the divergent validity of the measures. Another issue concerns our summing of specific illness items assumed to reflect a single underlying liability dimension. Future research should also look into the configuration of symptoms, and their underlying causes. Moreover, the application of measures with only modest reliabilities could be seen as challenging. However, our main focus here is to understand the nature of the covariances, and whereas random error contributes to variance, such error does not contribute to covariances.

Another issue concerns the generalizability of the findings. Heritability estimates are relative to the environmental heterogeneity; hence the results are not necessarily applicable across time, culture, and age. Although a random and representative sample of Norwegian twins is likely to be fairly representative of corresponding populations in other Western countries, studies in older—or younger—age groups, as well as in other cultures should be encouraged.

In conclusion, among young adults, SWB is substantially related to perceived health, moderately and negatively related to musculoskeletal pain, and virtually unrelated to allergic disorders. We found evidence for a considerable genetic contribution to the observed associations. In particular, the genetic variance in well-being and perceived health is highly overlapping. The environmentally caused variance in well-being seemed to be rather independent of the environmental factors influencing health. In the present era of molecular genetics, we believe it is also important to develop “molecular environmentics.” That is, future studies should attempt to disentangle specific environmental causes of well-being while controlling for genetic effects, and to address the complex interplay between genes and environment. Our findings suggest that for young adults the important loci of environmental causes might not primarily be linked to the health arena. Finally, future research into the diverse relations between human well-being and health should take into account the presence of both environmental and genetic factors inherent in these associations.

References


Received April 15, 2002
Revision received March 21, 2003
Accepted March 27, 2003