Age and Sex Differences in Genetic and Environmental Factors for Self-Rated Health: A Twin Study

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**Objectives.** Self-rated health has been shown to be a predictor for future health status and mortality. The purpose of this study was to investigate age-group and sex differences in genetic and environmental sources of variation for self-rated health.

**Methods.** A sample of twins from the Swedish Twin Registry participated in a computer-assisted telephone interview with assessment of self-rated health. Structural equation model analyses on 1,243 complete twin pairs provided estimates of genetic and environmental components of variance.

**Results.** Individual differences primarily reflected individual specific environmental influences at all ages. The increase in total variance across age groups was primarily due to genetic influences in the age groups 45–74 years and greater environmental influences in the oldest age group (>74). No significant sex differences were found in variance components.

**Discussion.** Genetic variance in the two middle age groups (45–74) could reflect genetic susceptibility to age-dependent illnesses not yet expressed in the youngest group. The findings suggest that it might be more fruitful to explore the origins of individual differences for self-rated health in the context of an individual’s age and birth cohort rather than in the context of sex.

Self-rated health is reported to predict functional ability (Idler & Kasl, 1995) and survival (Idler & Benyamin, 1997; Idler & Kasl, 1991) and is often a more powerful predictor of death than medical diagnoses (Mossey & Shapiro, 1982). Therefore, self-rated health is increasingly used as an outcome variable in health services research (Vang, 1997) and is of importance for policy makers and physicians. Considering the increase in use of self-rated health measures, it is important to understand why individuals differ in their health perception.

The concept of self-rated health implies a global assessment of one’s own health, summarizing the way in which different aspects of health, physical as well as mental, are combined within one’s perceptual framework (Bjorner et al., 1996; Brook et al., 1979; Liang, 1986). Although not specifically designed to address questions of age differences, many studies report worse self-rated health in older age groups, and a concomitant increase in individual differences (Bjorner et al., 1996; Earles, Connor, Smith, & Park, 1997; Roberts, 1999; Undén & Elofsson, 1998). An increase in total variance with age has been demonstrated for many health-related variables in cross-sectional studies (Nelson & Dannefer, 1992) and is consistent with life-span developmental theories (Baltes, Reese, & Lipsett, 1980). Relatively little is known about the causes of these individual differences across the life span, a topic that deserves further attention and is the focus of this study.

Previous studies have demonstrated that genetic factors are of moderate importance for individual differences in self-rated health (Christensen, Holm, McGue, Corder, & Vaupel, 1999; Harris, Pedersen, McClearn, Plomin, & Nesselroade, 1992). This may not be surprising given that disease status is to some extent genetically influenced, and epidemiological studies confirm a substantial genetic susceptibility to death (Iachine et al., 1998; Marenberg, Risch, Berkman, Foderus, & deFaire, 1994; Yashin, Iachine, & Harris, 1999). Thus, we would expect genetic influences for self-rated health. Nevertheless, it is notable that the increase in variance in self-rated health from late adulthood onward is for the most part attributable to increases in environmental variance (Harris, Pedersen, McClearn, et al., 1992). Furthermore, genetic and environmental factors mediate the association between psychosocial factors and self-rated health, although the sources of the covariation are dependent on the age of the sample (Harris, Pedersen, Stacey, McClearn, Plomin, & Nesselroade, 1992). Genetic factors mediate the association in one cohort, but less so in another. Although it is apparent that the role of genetic and environmental influences on individual differences in self-rated health varies with age group, most studies focus predominantly on older subjects (i.e., those over 50 years of age) and have relatively few or no younger subjects.

Even less well understood are sex differences in self-rated health, despite reports of mean differences (Bjorner et al., 1996; Murray, Dunn, & Tarnopolsky, 1982; Undén & Elofsson, 1998). Finding mean differences does not necessarily imply variance differences between the sexes, although one twin study (Lichtenstein & Pedersen, 1995) has suggested...
sex differences in the relative importance of genetic effects. They found that genetic variance was not significant for males. Inclusion of opposite-sexed twins is essential for evaluating whether different sets of genes and/or different environments are operating in the two sexes or whether their relative importance differs. To our knowledge, there are no reports that include opposite-sexed twins and specifically test whether different genes are of importance for individual differences in self-rated health in the two sexes.

Even though self-rated health is increasingly accepted as an important measure of health, there are several uncertainties as to why people differ in their health perception, the extent of age differences across the life span, and whether there are different determinants in men than in women. Therefore the aim of this study was to evaluate age-group and sex differences in the relative importance of genetic and environmental factors for self-rated health using like- and opposite-sexed twins aged 17–91 years.

Methods

Participants

A random sample of 850 pairs of twins from the Swedish Twin Registry, which comprises in principle all twin births in Sweden since 1886 (Cederlöf & Lorich, 1978; Pedersen et al., 1996), was selected for screening of health status. During 1996 and 1997, approximately equal numbers of pairs for each birth year between ages 17 to 85 were contacted for a telephone interview (Screening Across the Lifespan Twin study—pilot [SALT—pilot]). During 1998 the health screening was expanded with twins aged 65 years or older, regardless of the survival status of the co-twin (SALT). Of the additional 3,500 individuals contacted before October 1998, 2,650 participated. In total (SALT-pilot & SALT), the response frequency was 79%. The total sample of respondents consists of 1,262 complete pairs, including 343 pairs of monozygotic (MZ) twins, 438 pairs of like-sexed dizygotic (DZ) twins, 462 pairs of opposite-sexed (OS) twins, and 19 like-sexed twin pairs of undetermined zygosity. The twin analyses were based on the 1,243 pairs of known zygosity in which both responded. Mean age of the sample was 60.5 years (SD = 18.01). The male portion of the sample was 44%.

Data Collection

Data collection was performed with a computer-assisted telephone interview including a number of items asked to all twins regarding different diseases and symptoms. Efforts were made to interview members of a pair within a month of each other to minimize the risk of biasing the results by differential age effects. The Ethics Committee of the Karolinska Institutet and the Institutional Review Board of the University of Southern California approved the study.

Zygosity determination.—In order to establish the zygosity of the twin pairs, we asked the question, “During childhood, were you and your twin partner as alike as two peas in a pod or not more alike than siblings in general?” If both members of a pair responded “alike as two peas in a pod,” they were classified as MZ; if both responded “not alike,” they were classified as DZ. If the twins did not agree we used the answer to the question, “How often did strangers have difficulty distinguishing between you and your twin partner when you were children?” If both members of a pair responded “almost always or always” or “often,” they were classified as MZ. If both responded “seldom” or “almost never or never,” they were classified as DZ. The zygosity classification was confirmed using 13 DNA markers in a subset (n = 199 pairs) of the sample, and proved correct in 99% of the pairs.

Measures

Three items developed for Duke University’s Older Americans Resource Survey (OARS, 1978) were included: (1) “How would you rate your general health status?”, with response alternatives: excellent, very good, good, fair, poor; (2) “How would you rate your health status compared to 5 years ago?”, with response alternatives: better, almost the same, poorer; and (3) “Do you think your health status prevents you from doing things you would like to do?”, with response alternatives: Not at all, partially, to a great extent. Items were standardized separately (M = 0, SD = 1) and then summed. To achieve a positive scale, 10 points were added to the sum. A high score indicates a more positive health rating. Cronbach’s coefficient alpha was 0.67. Thus, the inclusion of three items in the definition of self-rated health resulted in modest but adequate reliability. In a principal components analysis of the three items, general health status had the highest loading (0.85) and health status compared to 5 years ago had the lowest loading (0.66), suggesting that these three items might tap different dimensions of self-rated health. Given these psychometric properties, we decided to analyze both the summed health scale, henceforth called “self-rated health,” and item number 1 “How would you rate your general health status?” separately. This non-comparative question is the most frequently used measure in studies investigating self-rated health (Björner et al., 1996).

Analytical Procedure

Genetic and environmental sources of individual differences.—The aim of quantitative genetic analysis is to determine the extent to which genetic and environmental influences are important for variation in a trait, in this case self-rated health. If additive genetic influences (A) are important for self-rated health, then MZ twins should be significantly more similar than DZ twins, as MZ twins share an identical genetic makeup whereas DZ twins share on average 50% of their segregating genes. Shared environmental influences (C) refer to nongenetic influences that contribute to similarity within pairs of twins regardless of zygosity, such as shared family environment, uterine environment, and contact throughout life. Nonshared environmental influences (E) are those individual specific influences (e.g., accidents, illnesses, different experiences or occupations) that make family members different from one another, including measurement error. Thus, the total phenotypic variance (Vp) can be described in terms of the following components of variance:

\[
Vp = Va + Vc + Ve,
\]
SELF-RATED HEALTH IN TWINS

where \( V_a \) denotes the variance associated with genetic influences, \( V_c \) the variance associated with shared environmental influences, and \( V_e \) the variance associated with nonshared environmental influences. Heritability is the proportion of phenotypic variance due to genetic influences. Intrapair similarity across the twin types is based on the following expectations:

\[
\begin{align*}
\text{cov}(MZ) &= V_a + V_c \\
\text{cov}(DZ) &= \frac{1}{2} V_a + V_c. 
\end{align*}
\]

The path diagram in Figure 1 illustrates the relationship between the measured phenotype in two members of a pair and the latent factors (A, C, E). Structural equation modeling is commonly employed in twin studies to provide maximum-likelihood estimates of percentages of total variance (\( a^2 \), \( c^2 \), and \( e^2 \); Neale & Cardon, 1992). The significance of parameters was evaluated through nested model comparisons using Mx (Neale, 1995). Akaike’s Information Criterion (AIC), reflecting both the goodness of fit of the model and its parsimony, was computed (\( \chi^2 - 2df \)) and the model with the lowest (i.e., largest negative) AIC value is said to fit best (Neale & Cardon, 1992).

The effects of age were adjusted for within sex and age groups through regression techniques (McGue & Bouchard, 1984). However, partialing out age does not preclude differences in the relative influence of genetic and environmental effects between age groups. Thus, analyses were performed separately for each age group and compared across age groups.

Sex differences.—Studies of like-sexed twins enable one to evaluate whether there are (a) sex differences in the total variance and (b) sex differences in the relative importance of genetic and environmental influences. Including opposite-sexed pairs provides the opportunity to test whether different genes and different environments are operating in the two sexes (Neale & Cardon, 1992). Lower intraclass correlations for the opposite-sexed twins than for the like-sexed DZ twins suggest a sex-specific effect, that is, that different genes or environments are operating in men and women. In order to obtain parameter estimates for \( a^2 \), \( c^2 \), and \( e^2 \) and a parameter, \( R_g \), which indicates whether genetic effects are the same or different in males and females, we used all five twin groups (MZ female, MZ male, DZ female, DZ male, OS) simultaneously, and a series of models was tested (Neale & Cardon, 1992).

Age differences.—In a second series of analyses, we tested for differences in genetic and environmental effects between the age groups. A model where parameters (\( a^2 \), \( c^2 \), and \( e^2 \)) are constrained to be equal across all groups was compared to a model in which the parameters are estimated for each group separately. In the next model, variance components for the groups are all constrained to be equal to a scalar multiple across groups. As a result, the standardized variance components (e.g., heritability estimates) are equal across age groups, but total variance may differ across age groups.

RESULTS

Descriptive Statistics

A fundamental assumption of quantitative genetic analyses based on twin data is that variances are equal for MZ and DZ twins. Analyses of variance (ANOVAs) indicated that there were no differences in means and variances between MZ and DZ twins for self-rated health or for the general health item. Descriptive statistics for the sample divided into four age groups (17–44, 45–64, 65–74, and 75–91) are presented by sex in Table 1. Consistent with expectations, variance increased across age groups for both self-rated health and the general health item. Compared to the younger participants, the older twins tended to rate their health less positively (\( F = 34.03, 3 \text{ df}, p = .0001 \) for self-rated health and \( F = 68.75, 3 \text{ df}, p = .0001 \) for the general health item). Contrary to expectations, no significant sex differences were found in means or variances.

Table 1. Number of Included Swedish Twins (n), Mean Values (\( M \)), and Standard Deviations (SD) for Self-Rated Health\(^a\) and for One General Health Item\(^b\) by Age Group and Sex

<table>
<thead>
<tr>
<th>Groups</th>
<th>Sex</th>
<th>( n )</th>
<th>( M )</th>
<th>SD</th>
</tr>
</thead>
<tbody>
<tr>
<td>Self-Rated Health(^a)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>17–44 years</td>
<td>Men</td>
<td>285</td>
<td>10.63</td>
<td>1.72</td>
</tr>
<tr>
<td></td>
<td>Women</td>
<td>285</td>
<td>10.71</td>
<td>1.63</td>
</tr>
<tr>
<td>45–64 years</td>
<td>Men</td>
<td>176</td>
<td>9.70</td>
<td>2.39</td>
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<tr>
<td></td>
<td>Women</td>
<td>218</td>
<td>9.86</td>
<td>2.38</td>
</tr>
<tr>
<td>65–74 years</td>
<td>Men</td>
<td>447</td>
<td>10.42</td>
<td>2.20</td>
</tr>
<tr>
<td></td>
<td>Women</td>
<td>557</td>
<td>10.19</td>
<td>2.40</td>
</tr>
<tr>
<td>75–91 years</td>
<td>Men</td>
<td>190</td>
<td>9.37</td>
<td>2.59</td>
</tr>
<tr>
<td></td>
<td>Women</td>
<td>366</td>
<td>9.23</td>
<td>2.53</td>
</tr>
<tr>
<td>General Health Item(^b)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>17–44 years</td>
<td>Men</td>
<td>285</td>
<td>0.31</td>
<td>0.78</td>
</tr>
<tr>
<td></td>
<td>Women</td>
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<td>0.27</td>
<td>0.78</td>
</tr>
<tr>
<td>45–64 years</td>
<td>Men</td>
<td>176</td>
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<td>1.00</td>
</tr>
<tr>
<td></td>
<td>Women</td>
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<td>0.94</td>
</tr>
<tr>
<td>65–74 years</td>
<td>Men</td>
<td>447</td>
<td>0.12</td>
<td>1.00</td>
</tr>
<tr>
<td></td>
<td>Women</td>
<td>557</td>
<td>-0.02</td>
<td>1.05</td>
</tr>
<tr>
<td>75–91 years</td>
<td>Men</td>
<td>190</td>
<td>-0.17</td>
<td>1.08</td>
</tr>
<tr>
<td></td>
<td>Women</td>
<td>366</td>
<td>-0.25</td>
<td>1.09</td>
</tr>
</tbody>
</table>

\(^a\)Self-rated health = sum of 3 items, “How would you rate your general health status?”, “How would you rate your health status compared to 5 years ago?”, and “Do you think your health status preveins you from doing things you would like to do?” 10 was added to achieve positive values on the scale.

\(^b\)One item = “How would you rate your general health status?”
Intraclass Correlations

Intraclass correlations by zygosity, sex, and age group are presented in Table 2. Equal or greater DZ than MZ correlations suggest that only environmental effects are contributing to variation among twins aged 17-44 years and the oldest group, aged 75-91 years. However, both MZ and DZ correlations are relatively small in these two age groups. Hence, nonshared environmental influences appear to be of primary importance in these groups. The greater MZ than DZ correlations for age groups 45-64 and 65-74 years suggest that additive genetic effects contribute to variability in these groups. The patterns of intraclass correlations for self-rated health and the general health item are similar. The correlations of opposite-sexed twins do not suggest that there are sex differences in the youngest groups, but possibly in the older groups. Lower intraclass correlations for the opposite-sexed twins than for the DZ twins suggests a sex-specific effect.

Quantitative Genetic Analyses

Results of the full model (including $a^2$, $c^2$, $e^2$, and $R_g$) and the most “parsimonious” models, henceforth referred to as “best-fitting” models (with the lowest AIC), are presented in Table 3 for self-rated health. In the full model for the youngest age group (17-44), additive genetic effects ($a^2$) account for 8% of the total variance, shared environmental effects ($c^2$) for 0%, and nonshared environmental effects ($e^2$) for 92%. The genetic correlation ($R_g$) between the opposite-sexed twins was estimated as 0, suggesting sex differences. The full model reflects the intraclass correlations for self-rated health but does not have the best fit. The best-fitting model included only shared ($c^2$) and nonshared environmental effects ($e^2$). Shared environmental effects accounted for 2% of the variation, and nonshared environmental effects accounted for 98% of the variation. For this and all other age groups, models constraining estimates ($a^2$, $c^2$, $e^2$) to be equal across sexes and the genetic correlation ($R_g$) fixed to 0.5 do not fit worse than those models allowing parameter estimates to differ in men and women. These results indicate a lack of sex differences in genetic and environmental influences on self-rated health.

Nonshared environmental influences are of greatest importance for all age groups. Genetic variation is significant for middle-aged twins (45-64 years, 44%) and the group aged 65-74 (40%). Shared environmental effects, but not genetic variance, account for 21% of the variation in the oldest age group.

Results of the full and best-fitting models for the general health item are presented in Table 4. The full model reflects the intraclass correlations but does not have the best fit, in a manner similar to the composite self-rated health scale. Again, there is no evidence of sex differences in the etiology of the general health item; parameter estimates are similar to that for self-rated health, and nonshared environmental influences were the most important source of individual differences in all groups. Genetic variance was significant in the two middle age groups, and shared environmental variance was significant in the oldest group.

In the second series of analyses we tested for differences based on age-group membership for self-rated health. The chi-square difference test of the model that constrained parameters ($a^2$, $c^2$, $e^2$) to be equal across all age groups compared to the model in which parameters are estimated freely for each group showed a significant effect of age group ($\chi^2$ difference = 105.51, 9 df, $p = .001$). Even when a scalar effect was added, the variance components were significantly different across the age groups, indicating that the relative importance of genetic and environmental effects differs by age group ($\chi^2$ difference = 27.67, 3 df, $p = .001$).

Discussion

The results of this study provide additional insight into the nature of individual differences in self-rated health across a broader span of ages than previously reported. In
accordance with most previous studies, there was an increase in variance for self-rated health across age groups (Bjørner & Søndergaard Kristensen, 1999; Bjørner et al., 1996; Undén & Elofsson, 1998), consistent with life-span developmental expectations of increasing variance with increasing age (Baltes et al., 1980; Nelson & Dannefer, 1992). Individual differences are predominantly due to individual-specific, nonshared environmental variance, although genetic variance also contributes to total variance in age groups 45–75 years of age. Unlike previous reports (Bjørner et al., 1996; Undén & Elofsson, 1998), we found no significant sex differences in means or variance components.

**Sex Differences**

Given the associations between functional abilities and self-rated health (Bjørner & Søndergaard Kristensen, 1999; Bjørner et al., 1996), and sex differences in functional problems and performance measures (Merrill, Seeman, Kasl, & Berkman, 1997), one could have expected to find sex differences in self-rated health. Furthermore, previous analyses of the associations between self-rated health and psychosocial factors suggest that there is less genetic variance for self-rated health in men than in women (Lichtenstein & Pedersen, 1995). The intraclass correlations reported here suggested a tendency toward sex differences in genetic and environmental influences in the older age groups, but no significant differences were found.

**Age Differences**

The increase in variance for self-rated health across age groups is first attributable to an increase in genetic variance and finally to nonshared environmental effects (shared environmental rather than genetic effects also contribute to the variance in the oldest group). Harris, Pedersen, McClearn, and colleagues (1992) reported a very similar pattern of results in another Swedish sample, even though younger age groups were poorly represented in their sample. There is a remarkable similarity in the components of variation in the oldest group in that and the present study. Although it is impossible to disentangle whether the age-group differences represent age-related changes or cohort differences with cross-sectional data, the results indicate that age-related changes are operating because subjects in the oldest age group in our study were born before 1928 whereas the oldest subjects in the Harris study were born in 1914 or earlier. Thus, twins 70 years old and older in both studies, regardless of birth year, show greater environmental variance and no genetic variance. Nevertheless, longitudinal data are necessary to distinguish cohort effects from aging effects and will also allow testing of whether the same environmental influences are operating at all ages or whether new influences come into play (Eaves, Long, & Heath, 1986).

**Environmental Effects**

Individual differences in self-rated health are primarily due to nonshared environmental effects regardless of age

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### Table 3. Proportions of Variance Explained by Genetic and Environmental Variance by Age Group for Self-Rated Health, Based on 1,243 Pairs of Swedish Twins

<table>
<thead>
<tr>
<th>Age Groups</th>
<th>Men</th>
<th>Women</th>
<th>Fit of Model</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>a²</td>
<td>c²</td>
<td>e²</td>
<td>Rg</td>
</tr>
<tr>
<td>17-44 Years</td>
<td>0.08</td>
<td>0.00</td>
<td>0.92</td>
<td>0</td>
</tr>
<tr>
<td></td>
<td>(0.00-0.35)</td>
<td>(0.00-0.24)</td>
<td>(0.65-1.0)</td>
<td>(0.00-0.5)</td>
</tr>
<tr>
<td>45-64 Years</td>
<td>0.35</td>
<td>0.11</td>
<td>0.54</td>
<td>0</td>
</tr>
<tr>
<td></td>
<td>(0.00-0.71)</td>
<td>(0.00-0.52)</td>
<td>(0.29-0.85)</td>
<td>(0.00-0.5)</td>
</tr>
<tr>
<td>65-74 Years</td>
<td>0.44</td>
<td>0.00</td>
<td>0.56</td>
<td>0</td>
</tr>
<tr>
<td></td>
<td>(0.23-0.60)</td>
<td>(0.40-0.76)</td>
<td>(0.00-0.5)</td>
<td>(0.00-0.5)</td>
</tr>
<tr>
<td>75-91 Years</td>
<td>0.40</td>
<td>0.08</td>
<td>0.92</td>
<td>0</td>
</tr>
<tr>
<td></td>
<td>(0.00-0.36)</td>
<td>(0.00-0.31)</td>
<td>(0.62-1.0)</td>
<td>(0.00-0.5)</td>
</tr>
</tbody>
</table>

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*S Self-rated health = sum of 3 items, “How would you rate your general health status?”, “How would you rate your health status compared to 5 years ago?”, and “Do you think your health status prevents you from doing things you would like to do?”

**Full model; a² = additive genetic, c² = shared environment, e² = nonshared environment, Rg = genetic correlation.**

**Akaike’s Information Criterion (AIC) was computed (χ² - 2df), and the model with the lowest (i.e., largest negative) value of this index is said to fit best by AIC (Neale & Cardon, 1992).**

**For all age groups, in the best fitting models (i.e., the most parsimonious according to AIC), estimates for men and women are equal and Rg is fixed to 0.5.**
group. A recent Danish study of twins 75 years of age and older (Christensen et al., 1999) found genetic variance but concluded that individual specific environmental influences are the most important source of individual differences in self-rated health in elderly persons. These effects reflect (apart from random and measurement error) individual specific experiences (e.g., accidents, social relationships, socio-economic status, work experiences, leisure activities, physical exercise) that may differ in their importance at different ages. For example, accidents and work experiences may in the youngest group. Many studies have shown that self-reports of health status correlate with more objective health measures (Liang, 1986; Schulz et al., 1994), and subjects also frequently mention absence or presence of illness as a component of their self-ratings (Idler, Hudson, & Leonthal, 1999). Analysis of age differences for a chronic illness scale showed a cross-sectional increase in genetic effects until the age of 70 similar to that found for self-rated health (Harris, Pedersen, McClearn, et al., 1992). Further studies are necessary to reveal whether symptoms of chronic illnesses mediate a major part of the genetic effects in the groups aged 45–64 and 65–74.

It is unclear whether the present patterns, with a lack of or reduction in genetic variance in the oldest age group, represent longitudinal changes or age-group effects due to loss of genetic variance for specific diseases. Genetic variance for an “objective” measure of chronic illnesses decreased longitudinally for men but was stable for women over a 30-year time span, from 50+ to 80+ years of age (Pedersen, Stensfossen, Berg, Johansson, & McClearn, 1999). Thus, the present results may not simply reflect loss of genetic variance due to selective survival. The lack of genetic influences on self-rated health in the oldest group may also reflect individual specific coping styles. Older individuals may learn to live with their illnesses over the years, and by the age of 80 they may no continue to include chronic diseases in their own health evaluation.

### Genetic Effects

What does the genetic variance reflect? The presence of genetic effects in the groups aged 45–64 and 65–74 could reflect genetic susceptibility to age-dependent illnesses such as diabetes or heart disease that have not yet been expressed in the youngest group. Many studies have shown that self-reports of health status correlate with more objective health measures (Liang, 1986; Schulz et al., 1994), and subjects also frequently mention absence or presence of illness as a component of their self-ratings (Idler, Hudson, & Leonthal, 1999). Analysis of age differences for a chronic illness scale showed a cross-sectional increase in genetic effects until the age of 70 similar to that found for self-rated health (Harris, Pedersen, McClearn, et al., 1992). Further studies are necessary to reveal whether symptoms of chronic illnesses mediate a major part of the genetic effects in the groups aged 45–64 and 65–74.

### Composite Measure Versus Single Item

We found no differences in variance components between self-rated health, including three items, and the general health item, suggesting that the essence of self-rated health does not appear to differ when measured by one single general item or a summed scale. A study on construct validity and functioning of a five-item general health scale (Bjorner & Søndergaard Kristensen, 1999) suggested some degree of

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### Table 4. Proportions of Variance Explained by Genetic and Environmental Variance by Age Group

<table>
<thead>
<tr>
<th>Age Groups</th>
<th>Parameter Estimate</th>
<th>Fit of Model</th>
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<tr>
<td></td>
<td>$a^2$</td>
<td>$c^2$</td>
</tr>
<tr>
<td>17–44 years</td>
<td>Full model</td>
<td>.00</td>
</tr>
<tr>
<td></td>
<td>Best fit1</td>
<td>.10</td>
</tr>
<tr>
<td>45–64 years</td>
<td>Full model</td>
<td>.47</td>
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<tr>
<td></td>
<td>Best fit1</td>
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<tr>
<td>65–74 years</td>
<td>Full model</td>
<td>.42</td>
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<td></td>
<td>Best fit1</td>
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<tr>
<td>75–91 years</td>
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</tr>
</tbody>
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*One item, “How would you rate your general health status?”

*Full model; $a^2$ = additive genetic, $c^2$ = shared environment, $e^2$ = nonshared environment, $R_g$ = genetic correlation.

Akaike’s Information Criterion (AIC) was computed ($x^2 – 2df$), and the model with the lowest (i.e., largest negative) value of this index is said to fit best by AIC (Neale & Cardon, 1992).

*For all age groups, in the best fitting models (i.e., the most parsimonious according to AIC), estimates for men and women are equal and $R_g$ is fixed to 0.5.
heterogeneity, but the items measuring current health had the highest loading. The authors conclude that the scale had good concurrent validity as judged from associations with physical, mental, and functional problems. We too found that the general health item had the highest loading on a first principal component. We doubt that the age-group differences are a function of the use of a composite scale versus a single item. Nevertheless, the meaning of self-rated health may differ by age group, and our findings may reflect shifts in the subjects' basis for health evaluation.

Limitations

Twin studies are sometimes criticized for the assumption that environmental similarity of MZ twins equals that of DZ twins. If MZ twins are treated more similarly than DZ twins, greater similarity in MZ twins for self-rated health could in part reflect this differential treatment. However, studies of the equal environment assumption for a wide variety of characteristics have shown that differential treatment is of little or no importance for twin similarity (Kendler, Gardner, & Charles, 1998; Loehlin & Nichols, 1976; Scarr & Carter-Salzmann, 1979).

Model fitting, which estimates the magnitude of the heritable and environmental components for self-rated health, cannot reveal how these anonymous variance components act and interact with each other. We do not know, for example, if specific genes for chronic diseases could be expressed differentially depending on environmental factors.

The size of our sample might give limited power to detect sex differences in the variance components. However, power calculations at a significance level of .05 and a heritability of 40%, as reported for the age groups 45–64 and 65–74, reveal that we have 80% and >95% power, respectively, to detect whether different genes are operating in men than in women. As there is no significant genetic variation in the youngest and oldest age groups, it is meaningless to test for a significant difference in genetic effects for men and women. Strengths in our study are the inclusion of opposite-sexed twins as well as all adult ages. The findings presented here, based solely on cross-sectional data, can be seen as a first step in identifying factors involved in the self-evaluation of one's own health as well as a step toward examining individual differences in self-rated health across the life span.

Conclusion

The purpose of this study was to explore age-group and sex differences in genetic and environmental sources of variation for self-rated health. We found age-group differences in variance components, but no significant sex differences. We conclude that individual specific environmental factors are of greatest importance for individual differences in self-rated health at all ages. Genetic factors are also of importance in older ages (45–74), when chronic diseases start to be more common. After the age of 75, environmental factors are again the sole source of variation. These findings, based on cross-sectional data, suggest that it might be more fruitful to explore the origins of individual differences in self-rated health in the context of an individual's age and birth cohort rather than in the context of sex.

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