Original Research Report

Longitudinal Twin Study of Subjective Health: Differences in Genetic and Environmental Components of Variance Across Age and Sex

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Abstract

Objective: The current analysis examines sex differences in longitudinal changes in genetic and environmental influences on three measures of subjective health (SH).

Method: Sample includes 7,372 twins (mean intake age = 73.22) with up to 8 waves of measurement (mean = 3.1). Three SH items were included: general self-rated health (SRH), health compared to age peers (COMP), and impact of health on activities (ACT) which previous research shows capture different frames of reference.

Results: Latent growth curve modeling indicated significant differences across gender and frame of reference in trajectories of change with age and in genetic and environmental contributions to change. Men have higher mean scores on all three SH measures, indicating better SH, but there were no sex differences in pattern of change with age. Accelerating declines with age were found for SRH and ACT, whereas COMP improved with age. Results indicated more genetic variance for women than men, but declining genetic variance for both after age 70. Increasing shared environmental variance with increasing age was also found for both sexes.

Discussion: As aging triggers a re-evaluation of the meaning of “good health,” physical aspects of health may become less important and shared cultural conceptions of health may become more relevant. This change in conceptions of good health may reflect both aging and the change in composition of the elderly population as a result of selective survival.

Keywords: Frame of reference, Latent growth curve model, Question type, Self-rated health

Research often demonstrates that subjective health (SH) predicts morbidity and mortality, independent of many measures of objective health (Benyamini, 2011; Idler & Benyamini, 1997; Latham & Peek, 2013; McFadden et al., 2009), although there are exceptions (Fried et al., 1998). The utility of what is often a single item (eg, rate your overall health) has long been accepted and incorporated into major international projects (Euro-REVES, 2002; World Health Organization, 1996). Given its predictive value, research has shifted to identifying factors that contribute to SH (Arnadottir, Gunnarsdottir, Stenlund, & Lundin-Olsson, 2011; Bailis, Segall, & Chipperfield, 2003; Darviri et al.,...
Finally, as predicted by Jylhä’s (2009) emphasis on the role of conceptualizations of “good health,” different SH items could result in different appraisals of the factors that constitute self-perception of health. For instance, the general self-rated health item (how you rate your overall health) has a nonspecific frame of reference, whereas items that were specifically self-comparative (compare your current health with previous health) or age-comparative (compare your health to others your age) appear to shift the person’s perspective-taking about health. Results indicate that age differences, sex differences, and even the variables that predict SH vary by question type (Dening et al., 1998; McCullough & Laurenceau, 2004; Sargent-Cox et al., 2008; Sargent-Cox, Anstey, & Luszcz, 2010; Seitsamo & Klockars, 1997).

If the combinations of factors that contribute to SH vary by age, sex, and question type, then the genetic and environmental contributions to variance in SH should also vary by age, sex, and question type. Therefore, the twin method provides a means for testing expectations generated by the current understanding of SH. Studies of adult twins in Australia, Denmark, Finland, Sweden, and the United States have reported heritability estimates for SH primarily in the range of 25%–30%, with evidence for modest increases in heritability with age (for a review, see Franz et al., 2017). A recent cross-sectional twin analysis that included 12,900 individuals aged 25–102 from the Interplay of Genes and Environment across Multiple Studies consortium (IGEMS; Pedersen et al., 2013), which is also the basis for the present study, provided a more nuanced understanding of genetic and environmental influences on SH (Franz et al., 2017). Results indicated that heritability varied significantly by age, sex, and question type. For the general self-rated health variable, genetic variance increased with age for men, but was more stable for women. Genetic variance for a SH measure focused on activities peaked in midlife for men, but increased in late life for women. No age or sex differences in genetic or environmental variance were evident for the age-comparative item.

The goal of the current analysis is to expand on the results of Franz and colleagues (2017) using longitudinal twin data from the IGEMS consortium to examine within-person change in conceptions of SH, in genetic and environmental components of variance over age, and between genders and question types. To the extent that the predictors of SH differ across age, sex, and question type, we expect the genetic and environmental components of variance to differ as well. Shared cultural concepts of health (Jylhä, 2009) will be reflected in shared environmental variance, and may become more relevant as aging triggers a re-evaluation of the meaning of “good health.” The meaning of “good health,” and thus the role of shared environmental variance, may differ between men and women and be differentially elicited by question type. We expect that age changes in genetic and environmental influences on SH will reflect age changes in genetic and environmental influences on physical health (Finkel,
Gerritsen, Reynolds, Dahl, & Pedersen, 2014). However, given that the relationship between physical and SH declines with age (Pinquart, 2001), the genetic variance for SH change with age, particularly for items that are more self-comparative versus age-comparative. Longitudinal twin data will allow for direct assessment of within-person changes in genetic and environmental components of variance.

Method

Participants

IGEMS is an international consortium of twin studies from the Nordic countries, the United States, and Australia covering the adult life span (Pedersen et al., 2013). Three of the IGEMS studies included measures of SH and the three or more longitudinal measurement waves required to support latent growth curve modeling: Swedish Adoption/Twin Study of Aging (SATSA; Finkel & Pedersen, 2004), Origins of Variance Among the Oldest Old (OCTO-Twin; McClearn et al., 1997), and Longitudinal Study of Aging Danish twins (LSADT; Christensen, Holm, McGue, Corder, & Vaupel, 1999). Previous analyses indicate that these samples are representative of their age peers within each country for health variables at intake (Christensen & McGue, 2012; Pedersen, Steffensson, Berg, Johansson, & McClearn, 1999; Svedberg, Bardage, Sandin, & Pedersen, 2006). The sample sizes and age ranges from the three studies are presented in Table 1: a total of 7,372 individuals contributed relevant data to the current study. Age ranged from 26 to 102 years, with a mean age at intake of 73.22 (SD = 11.9); 78% of the sample was aged 70–90 years. Mean interval between measurement waves ranged from 2.01 years (OCTO) to 3.71 years (SATSA). In all three studies and in the overall sample, women were significantly older than men on average; however, there were no significant sex differences in number of waves of participation (mean = 3.15, SD = 1.9). Moreover, using an age-based growth curve model takes any sex differences in age into account.

Measures

Three different types of questions were used to assess SH in the three studies. The most common question used to assess SH is the general self-rated health item (SRH): “how would you rate your overall health?” Two studies (SATSA and OCTO) recorded answers on a three-point scale, whereas LSADT used a five-point response scale. These studies also included an age-comparative item (COMP): “compared to others your age, how would you rate your overall health?” for which all three studies used a three-point response scale. Finally, participants indicated how their health affected their daily activities (ACT). Two studies used the question “do you think your health condition is preventing you from doing things you would like to do?” with a three-point response scale. In LSADT, the item was phrased “do you feel well enough to do what you like?” and used a five-point response scale.

Although the SH questions administered across the studies were similar or identical, the response scales varied. To examine and reconcile differences among these putatively similar measures, we engaged in a harmonization process, collecting new data on all combinations of questions and answer schemes used in all of the IGEMS studies from an independent international sample of 1,065 participants aged 30–98 years (Gatz et al., 2015). The harmonization sample allowed us to verify that similarly worded questions correlated substantially, regardless of exact wording or response scales. Average correlations across response scales were .77 for SRH, .78 for ACT, and .63 for COMP. Comparison of three types of harmonization methods indicated that the optimal approach involved standardizing scores within each sample and converted to T-scores (mean = 50, SD = 10). For all measures, high scores indicated better SH.

Table 1. Sample Demographics

<table>
<thead>
<tr>
<th>Variables</th>
<th>SATSAa</th>
<th>OCTOb</th>
<th>LSADTC</th>
<th>Total sample</th>
</tr>
</thead>
<tbody>
<tr>
<td>Number of individuals</td>
<td>1,939</td>
<td>702</td>
<td>4,731</td>
<td>7,372</td>
</tr>
<tr>
<td>Number of pairs MZ/DZd</td>
<td>319/631</td>
<td>141/190</td>
<td>754/1,355</td>
<td>1,215/2,176</td>
</tr>
<tr>
<td>Maximum number of waves</td>
<td>8</td>
<td>4</td>
<td>6</td>
<td>8</td>
</tr>
<tr>
<td>Mean number of waves (SD)</td>
<td>4.11 (2.28)</td>
<td>2.86 (1.18)</td>
<td>2.72 (1.51)</td>
<td>3.15 (1.86)</td>
</tr>
<tr>
<td>Mean wave interval (SD)</td>
<td>3.71 (1.37)</td>
<td>2.01 (0.04)</td>
<td>2.01 (0.06)</td>
<td>2.95 (0.66)</td>
</tr>
<tr>
<td>% Female</td>
<td>57.89%</td>
<td>66.67%</td>
<td>58.93%</td>
<td>59.09%</td>
</tr>
<tr>
<td>Age range</td>
<td>26–93</td>
<td>79–98</td>
<td>70–102</td>
<td>26–102</td>
</tr>
<tr>
<td>Mean age (SD) Men</td>
<td>58.39 (13.64)</td>
<td>83.14 (2.92)</td>
<td>76.55 (5.39)</td>
<td>72.05 (12.0)</td>
</tr>
<tr>
<td>Mean age (SD) Women</td>
<td>61.36 (14.16)</td>
<td>83.80 (3.28)</td>
<td>77.76 (5.77)</td>
<td>74.01 (11.72)</td>
</tr>
</tbody>
</table>

Note: aSwedish Adoption/Twin Study of Aging bOrigins of Variance Among the Oldest Old cLongitudinal Study of Aging Danish Twins dMZ = Monozygotic twins; DZ = Dizygotic twins. *Difference in mean age between men and women is significant at p < .01
Statistical Method

Due to the range in age at intake, an age-based biometric latent growth curve model (LGCM) was used to examine genetic and environmental contributions to changes in SH over age (Neale & McArdle, 2000). The LGCM provides estimation of fixed effects, that is, fixed population parameters as estimated by the average growth model of the entire sample, and random effects, that is, individual variation in growth model parameters. The intercept is evaluated at the centering age; given the mean intake age of 73.2 and the mode age of 75, the centering age was set at age 75. The age-based quadratic latent growth curve model is presented in Supplementary Figure 1. Observed data are indicated by y0 through y8. Group mean intercept (M0) and rates of change are estimated (M1 and M2) and residual variances (u0 through u8) are set equal across waves. The paths from the latent slope factors to the observed scores are the age basis coefficients, B(t) and B(t)^2. The age basis serves as a marker for the age of the subject at each time of measurement, adjusted for the centering age. Therefore, age basis coefficients are defined as an individual’s observed age at each measurement occasion minus the centering age (75 years).

Using twin data, the random effects, or variance, in latent growth curve parameters can be divided into three separate components: additive genetic effects (A), shared environmental effects that serve to make members of twin pairs more similar to each other (C), and nonshared environmental effects unique to each individual and error associated with age-specific residuals (E). For simplicity, the model in Supplementary Figure 1 includes only the additive genetic effects for the intercept (A0) and slopes (A1 and A2). Genetic influences on correlations among intercepts and slopes are captured by the paths from A0 to L and Q and from A1 to Q. In total then, there are six genetic parameters (paths) estimated by the model. Shared environment and nonshared environment were also included in the model, for a total of 18 biometric parameters.

By fitting structural models to the observed monozygotic (MZ) twin and dizygotic (DZ) twin covariance matrices, we can estimate the proportion of phenotypic variance accounted for by the variance in genetic factors, shared environment factors, and nonshared environment factors. Separate parameters were estimated for men and women and then equivalence of parameter estimates was tested across sex. Biometric latent growth curve models were fit with the structural equation modeling program Mx version 1.66b (Neale, Boker, Xie, & Maes, 2003). The raw maximum likelihood estimation procedure was used throughout. We tested nested models using a likelihood ratio test (ie, subtracting the −2 log likelihoods of the models being compared).

Results

Model Comparisons

In the first set of models, sex differences in the biometric latent growth curve model were tested, as reported in the top of Table 2. First, the full model with all parameters estimated separately for men and women was fit to the data. In model 2, all model parameters were equated across sex: 3 growth curve parameters (intercept, linear change, and quadratic change) and 18 biometric parameters (paths for A, C, and E). The likelihood ratio test indicated a significant change in model fit for SRH (LRT = 148,255–148,197 = 58, df = 44–123 = 21, p < .01) and COMP (LRT = 58, df = 21, p < .01), but failed to achieve significance for ACT (LRT = 32, df = 21, p = .06). Thus model fitting indicated sex differences in the models for SRH and COMP. In model 3, only the three growth parameters were equated across sex; a significant change in model fit occurred only for SRH, although the comparison was marginally significant for ACT (LRT = 7, df = 3, p = .07). The biometric parameters were equated across sex in model 4, and significant changes in model fit resulted for SRH and COMP, but not ACT.

Latent Growth Curve Parameters

In the second phase of model fitting, additional models were tested to identify the nature of the sex differences in change trajectories; results are presented in the middle of Table 2. Sex differences in each growth curve parameter were tested independently in models 5, 6, and 7. Comparing model fit statistics to model 1 indicated significant sex differences in intercept, only. Thus, men and women differ in mean SH at age 75, but there are no significant sex differences in either linear or accelerating rates of change, regardless of question type. Change trajectories estimated by the growth curve model are presented in Figure 1; growth curve parameter estimates are reported in Table 3. Trajectories are presented from age 35 to 90 years because coverage before 35 and after 90 is sparse. For each SH variable, men report significantly higher (more positive) SH at age 75; the difference in means is about 1 point for SRH and ACT and half a point for COMP. Even though there are no sex differences in rates of change with age, there are striking differences across question type. Both SRH and ACT show significant and accelerating rates of decline with age, with a steeper rate of decline evident for ACT. In contrast, the COMP variable demonstrates significant but slightly decelerating increases with age.

Genetic and Environmental Parameters

In the third phase of model fitting, sex differences in individual biometric parameters were investigated; results are presented in the bottom section of Table 2. Sex differences in each component of variance were tested independently: in model 8 genetic variance (A) was equated across sexes, in model 9 shared environmental variance (C) was equated and in model 10 nonshared environmental variance (E) was equated. For SRH, only model 10 resulted in a significant change in model fit, indicating sex differences in nonshared variance. Additional models were tested that dropped either all genetic variance for men (6 parameters) or all genetic variance for women. Dropping genetic variance for men
did not result in a significant change in model fit (LRT = 1, df = 6, ns), but it did result in a significant change in model fit for women (LRT = 15, df = 6, p < .05). Thus, models indicate significant genetic variance for SRH in women, but not in men. Longitudinal changes in components of variance as estimated by the biometric LGCM are presented in Figure 2; parameter estimates are reported in Supplementary Table 1. As shown in the top panels of Figure 2, total variance increases in SRH with age resulted from increases in genetic variance up to age 65 and then from increases in shared environmental variance. For both men and women, genetic variance declined after age 65, although genetic variance in men was not significant. The middle panels of Figure 2 show that the age changes in variance components were fairly similar for ACT; however, model comparisons reported in Table 3 indicate that sex differences in variance components did not achieve significance.

Longitudinal age changes in mean SRH and ACT were fairly similar; therefore, it is not surprising that the longitudinal age changes in variance components were also similar. Results for mean COMP indicate that the age-comparative variable taps a different formulation of SH, a conclusion that is also supported by the longitudinal age changes in variance components. Model fit results presented in Table 3 indicated significant sex differences in all three components of variance. Age changes in variance shown in the bottom panels of Figure 2 highlight the sex differences. Total variance was generally stable for men, with a peak in midlife, whereas it generally increased across the life span for women. Estimates of genetic variance were zero for men across the life span, whereas significant genetic variance was indicated for women, peaking in midlife and decreasing thereafter. Both men and women demonstrated increasing shared environmental variance with increasing age.

**Discussion**

The primary goal of the current study was to analyze mechanisms underlying longitudinal changes in three

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**Table 2.** Results of Comparing All Models to the Full Model (model 1)

<table>
<thead>
<tr>
<th>Model</th>
<th>Parameters</th>
<th>SRH</th>
<th>ACT</th>
<th>COMP</th>
</tr>
</thead>
<tbody>
<tr>
<td>Initial Model Testing</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>1. Full Model</td>
<td>44</td>
<td>148,197*</td>
<td>145,505b</td>
<td>147,919c</td>
</tr>
<tr>
<td>2. Equate all across sex</td>
<td>23</td>
<td>148,253**</td>
<td>145,537</td>
<td>147,977**</td>
</tr>
<tr>
<td>3. Equate LGCM across sex</td>
<td>41</td>
<td>148,209**</td>
<td>145,512</td>
<td>147,922</td>
</tr>
<tr>
<td>4. Equate biometric across sex</td>
<td>26</td>
<td>148,244**</td>
<td>145,528</td>
<td>147,972**</td>
</tr>
<tr>
<td>Follow-up testing of LGCM</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>5. Equate I across sex</td>
<td>43</td>
<td>148,203*</td>
<td>145,513**</td>
<td>147,925*</td>
</tr>
<tr>
<td>6. Equate L across sex</td>
<td>43</td>
<td>148,198</td>
<td>145,505</td>
<td>147,919</td>
</tr>
<tr>
<td>7. Equate Q across sex</td>
<td>43</td>
<td>148,199</td>
<td>145,506</td>
<td>147,922</td>
</tr>
<tr>
<td>Follow-up testing of biometric</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>8. Equate A across sex</td>
<td>38</td>
<td>148,205</td>
<td>145,506</td>
<td>147,932*</td>
</tr>
<tr>
<td>9. Equate C across sex</td>
<td>38</td>
<td>148,202</td>
<td>145,506</td>
<td>147,934*</td>
</tr>
<tr>
<td>10. Equate E across sex</td>
<td>38</td>
<td>148,211*</td>
<td>145,514</td>
<td>147,947**</td>
</tr>
</tbody>
</table>

*Note: Model fit statistic is −2LL. *Degrees of freedom in full model for SRH = 20671 **Degrees of freedom in full model for ACT = 20549 *Degrees of freedom in full model for COMP = 20268 I = intercept, L = linear change, Q = quadratic change, A = additive genetic variance, C = shared environmental variance, E = nonshared environmental variance. *Difference in model fit compared to model 1 is significant at p < .05. **Difference in model fit compared to model 1 is significant at p < .01. ACT = Impact of health on activities; COMP = Health compared to age peers; LGCM = Latent growth curve model; SRH = self-rated health.

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**Table 3.** LGCM Parameter Estimates (SE) From Full Growth Curve Model

<table>
<thead>
<tr>
<th>Parameter</th>
<th>SRH</th>
<th>ACT</th>
<th>COMP</th>
</tr>
</thead>
<tbody>
<tr>
<td>Intercept</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Men</td>
<td>50.33 (0.0002)</td>
<td>50.64 (0.0019)</td>
<td>50.21 (0.0003)</td>
</tr>
<tr>
<td>Women</td>
<td>49.45 (0.0005)</td>
<td>49.76 (0.0023)</td>
<td>49.72 (0.0006)</td>
</tr>
<tr>
<td>Slope</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Men</td>
<td>−0.20 (0.0018)</td>
<td>−0.34 (0.0005)</td>
<td>0.01 (0.0006)</td>
</tr>
<tr>
<td>Women</td>
<td>−0.19 (0.0008)</td>
<td>−0.32 (0.0013)</td>
<td>0.02 (0.0036)</td>
</tr>
<tr>
<td>Quadratic</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Men</td>
<td>−0.004 (0.0001)</td>
<td>−0.008 (0.0001)</td>
<td>−0.002 (0.0001)</td>
</tr>
<tr>
<td>Women</td>
<td>−0.004 (0.0001)</td>
<td>−0.006 (0.0001)</td>
<td>−0.001 (0.0002)</td>
</tr>
</tbody>
</table>

*Note: ACT = Impact of health on activities; COMP = Health compared to age peers; LGCM = Latent growth curve model; SRH = self-rated health.
measures of SH in adulthood from the perspective of genetic and environmental components of variance. Combining data across three longitudinal twin studies that are part of the IGEMS consortium provided sufficient power to test predictions about age changes and sex differences for three SH variables representing different conceptions of SH.

Longitudinal Changes in Mean Subjective Health

Overall, the latent growth curve models provide strong support for within-person age changes in SH, differential frames of reference for different SH items, and modest but significant sex differences in means. The current longitudinal analysis indicated accelerating declines with age in a general SH item (SRH) and an activity-focused item (ACT). Similar to other studies, we found that an age-comparative item (COMP) demonstrated significant improvement with age (Dening et al., 1998; Franz et al., 2017; Sargent-Cox et al., 2008, 2010; Seitsamo & Klockars, 1997). As others have reported, different SH items tap different frames of reference for “good health,” which can then change the weighting of the factors that constitute self-perception of health (Sargent-Cox et al., 2008, 2010).

By combining data from three large studies, the current sample provided ample power to detect sex differences in longitudinal changes in mean SH, a problem in previous studies (McCullough & Laurenceau, 2004). We found sex differences in means only, not in rates of decline; men reported significantly higher levels of SH at age 75 for all three measures. Findings for sex differences in mean SH and rates of change with age have been somewhat mixed (McCullough & Laurenceau, 2004). Sargent-Cox and colleagues (2010) reported sex differences in the age trajectories of an age-comparative SH item, with older men demonstrating a higher likelihood to report poor SH. Evident in Figure 1 is a suggestion that change trajectories converged across sex in late adulthood. Although sex differences in the quadratic terms were not significant for any SH variable, they did approach significance for the age-comparative item (LRT = 3, df = 1, p = .08). Thus in the current study we also find the possibility of a reduction with age in the male advantage in the age-comparative SH item.

Longitudinal Changes in Subjective Health Variance

Previous cross-sectional and longitudinal studies have reported increasing individual differences in measures of SH with increasing age, resulting at least partly from increases in individual differences in physical health (eg, (Franz et al., 2017; Svedberg, Gatz, Lichtenstein, Sandin, & Pedersen, 2005). In the current study, we also report increasing total variance with increasing age, although sex and question type differences were evident. The largest increases in variance were evident for the activity-focused item and variance increases for both the general SH item and the activity-focused item were larger for women than for men. Twin analyses allowed us to demonstrate that the increasing variance resulted primarily from increases in genetic variance in middle adulthood, but from increasing environmental components of variance in late adulthood. Thus, the twin approach supports the conclusion that combination of variables that contribute to SH changes with age, and differ across sex and question type. It is important to note that the changes identified here may reflect true changes with age and/or result from changes in the composition of the elderly population included in the studies as a result of selective survival.

Beyond simply identifying environmental factors as important components of conceptualizations of SH in late adulthood, twin analyses highlighted that shared environmental variance, in particular, increased in late adulthood. Typically, shared environmental variance is defined as the result of a shared rearing environment: it contributes to the similarity of twins who are reared together, but not twins who are reared apart (Plomin, DeFries, Knopik, & Neiderheiser, 2013). Twins in these samples shared their rearing socioeconomic status and may have learned healthy lifestyles in their childhood years that continue to influence their behavior in late adulthood (eg, (Seeman & Crimmins, 2001). Most twin research indicates, however, that the impact of rearing environment tends to decrease with increasing age for many traits, including physical health and other relevant components of SH (eg, (Finkel
et al., 2014; Reynolds et al., 2005). Additionally, evidence suggests that SH is influenced primarily by recent events or current SES levels, as opposed to distal factors (Manderbacka & Lundberg, 1996; Verropoulou, 2012). Shared environmental variance more generally includes any factor in the environment that makes members of both MZ and DZ twin pairs more similar to each other, including correlated environmental effects shared by anyone living in the same culture (Pedersen, Plomin, Nesselroade, & McClearn, 1992). With regard to SH, then, shared environmental variance could include socioeconomic status as well as representations of cultural concepts of health, expectations about help-seeking, and meanings people give to health problems (Jylhä, 2009). Moreover, as aging triggers a re-evaluation of the meaning of “good health,” these shared cultural conceptions of health (either persistent from childhood or developed in adulthood) may become more relevant, resulting in the increasing shared environmental variance observed in the current study.

The increasing influence of cultural conceptions of health with age seemed to play a somewhat larger role for men than for women: across the three measures of SH men demonstrated more shared environmental variance in late life than women. Sex differences were also evident in the extent of genetic variance across question types, with generally more genetic variance in SH for women than for men. These results suggest that men may rely more on cultural conceptions of health when evaluating their own health, whereas women may rely more on physical health conditions, which reflect greater genetic influences than SH measures (Finkel et al., 2014). One reason for this sex difference in conceptualizations of SH may be the differential experience of physical aging between men and women. In general, men experience physical health problems for a relatively short duration, compared with women, who tend to survive their health problems and live longer (Deeg & Kriegsman, 2003; Deeg et al., 2002; Sainio et al., 2006). An additional explanation may arise from the fact that, as a result of sex differences in survival patterns, men are more likely to have survived their age peers, whereas women are more likely to have age peers who also suffer from chronic health conditions (Deeg & Kriegsman, 2003). If this sex difference in survival patterns does play a role in conceptualizations of SH, then we would expect the sex differences in components of variance to be especially pronounced for age-comparative items, which is exactly what we found in the current analyses.

Even though we find sex differences in the amount of genetic variance for SH, a commonality across sexes is the pattern of change in genetic variance with age. For both sexes and across question type, if genetic variance was nonzero, it peaked in middle age and declined in late adulthood. This result is consistent with changes in the conceptualization of SH over the adult life span. In particular, evidence suggests that with increasing age, older adults rely more on perceptions of psychological well being and less on estimations and comparisons of physical functioning (both with self and age peers) to rate their own health, at least until late old age. (Benyamini et al., 2000; Jylhä et al., 1986; Meng & D’Arcy, 2016; Shooshtari et al., 2007; Spuling et al., 2015; Verropoulou, 2012). To the extent that physical health reflects genetic variance (Finkel et al., 2014), genetic components of variance in measures of SH should decline with increasing age as adults focus their attention more on other facets of their health experience, particularly their cultural conceptions of “good health,” in later adulthood. In very old age, the point at which the graphs in Figure 2 generally demonstrate modest increases in environmental components of variance, physical aspects of aging such as chronic diseases become more important to SH (Jylhä et al., 1986).

Limitations

Limitations include many of the statistical assumptions common to structural equation models. The data are assumed to be missing at random and the sample is assumed to be relatively homogeneous. As one focus of the current analysis was on sex differences, it is important to note that patterns of participation and attrition did not differ significantly for men and women. As with any longitudinal sample, attrition occurred in the IGEMS samples. However, using an age-based growth curve model instead of a time-based model allowed us to maximize power, especially for twin pairs with more participation waves. In addition, the age-based model allowed us to center the models at age 75, an age at which all three studies contributed data and thus minimize the impact of a single source (SATSA) for data from early adulthood.

Even though the samples were representative of their respective populations at intake, nonrandom dropout through the course of the longitudinal studies results in increasingly select samples of adults who are healthy enough to participate. Wave-to-wave dropout in these studies was quite low (about 8%), but dropout accumulates across waves. As a result, our analyses have likely underestimated the extent of change with age in measures of SH: SRH and ACT may actually decrease more dramatically, and modest increases in COMP with age may reflect the perception of relatively healthy older adults. Changes in genetic and environmental components of variance may reflect aging or the impact of selective survival. Previous investigation of the impact of survival on twin similarity for SH in the OCTO-Twin sample indicated differences in genetic and environmental components of variance for survivors versus nonsurvivors for men but not for women (Pedersen et al., 1999). Although beyond the scope of the current analyses, survival analyses could be used to investigate whether one of the three measures of SH is best at predicting loss to follow up and the degree of genetic influence on that predictive relationship.
Conclusions

Estimating age changes and sex differences in genetic and environmental contributions to variance in measures of SH allowed us to identify that in late life environmental variance becomes more important in conceptions of SH in surviving older adults. Therefore, researchers attempting to identify the variables that predict SH outcomes in late adulthood (Arnadottir et al., 2011; Bailis et al., 2003; Darviri et al., 2012; Meng & D’Arcy, 2016; Shoohtari et al., 2007) may benefit from focusing their search on identifying relevant environmental factors. For young-old individuals, genetic variance plays a larger role, suggesting that a focus on genetically influence traits that may contribute to conceptions of SH, including physical health, would be fruitful. Moreover, in young-old age, genetic variance plays a larger role for women than men, indicating sex differences in the types of variables that contribute to a conceptualization of SH at this age. Therefore, the next step is to incorporate measures of objective health, psychological variables, and social and financial resources (eg, Finkel et al., 2016), as well as measured genes, to identify the factors that contribute to the genetic and environmental variance identified here.

Supplementary Material

Supplementary data is available at The Journals of Gerontology, Series B: Psychological Sciences and Social Sciences online.

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Conflict of Interest

None reported.

References


