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Age Differences in Genetic and Environmental Influences for Health From the Swedish Adoption/Twin Study of Aging

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This cross-sectional study explored the etiology of variability in self-reported health. The sample comprises adult twins participating in the Swedish Adoption/Twin Study of Aging and includes identical (MZ) and fraternal (DZ) twin pairs who have been reared together or reared apart. Two different components of overall health status are analyzed: an index of chronic health problems and self-rated health. Height and weight were included to assess the representativeness of the twin data. Individual differences increased across age for both measures of health, and there were significant age differences in the genetic and environmental etiologies of this variation. Genetic variance showed a twofold increase for chronic illness up until age 70. Environmental influences during adulthood appear important later in life. For self-rated health, genetic effects were important in the older age groups; however, the increase in total variation is predominately due to unique environmental influences.

INDIVIDUAL differences in behavioral and biological measures are substantial and persist throughout life, according to several studies on aging (Palmore, 1970, 1974; Thomae, 1976). Contrary to traditional views, which hold that declines in physical functioning and health are universal in the later part of the life span, individual differences are pronounced (Bell, Rose, & Damon, 1966; Palmore, 1970, 1974; Thomae, 1976). Some people live long and healthy lives, while others experience failing health over a large portion of their adult years. Thus, individual aging profiles do not always conform to average, species-typical functions (Shock, 1985).

Different influences have been hypothesized to account for variation across age. For instance, life-span paradigms emphasize that accumulated effects of unique environmental experiences (Baltes, Reese, & Lipsett, 1980) become relatively more important. Theories of biological aging focus on the importance of genetic effects (for a review, see Hayflick, 1985) and predict that inter-individual variation in functions of the DNA explains the increase in age-related differences. Examples include a decline in the integrity of the genetic machinery or age-related changes that may be coded for in the DNA. It is probably misleading to seek a universal answer. Rather, age differences in the genetic and environmental variance architecture of individuality will likely vary from phenotype to phenotype.

This study focused on age differences in the etiology of individual variation for health. Although the literature addressing this issue is sparse, there is some evidence illustrating differences (across generations) in the relative contribution of genes and environment for diastolic blood pressure (Sims, Hewitt, Kelly, Carroll, & Turner, 1986). Specifically, variance due to genetic influences decreased from early adulthood to middle age, and the role of unique environmental experiences increased.

Another study on age differences in health did not explore the genetic and environmental sources of variation, but did find significant age differences in the salient factors that predict self-rated health. Specifically, symptomatology and physical fitness best explained the self-rating of health in the group aged 31–35; psychic well-being and symptoms were most important in the group aged 51–55, and chronic diseases played the largest role in the oldest group aged 71–75 (Jylhä, Leskinen, Alanen, Leskinen, & Heikkinen, 1986). If these explanatory variables are differentially influenced by genes and environments, then we would expect age differences in the genetic and environmental factors that contribute to variation in health.

Research to date suggests that overall health status comprises distinct components including objective and subjective health (Brook et al., 1979; Liang, 1986). The two measures in this study tap into these different aspects of health. The questions explored here are: Does variability increase across age? and, Are there age differences in the contribution of genes and environments to this variation?

Method

Sample. — The sample consists of 758 intact pairs of twins participating in a cross-sectional component of the Swedish Adoption/Twin Study of Aging (SATSA). Detailed descriptions of the sample, including the method of zygosity determination, have been published elsewhere (McClearn et al., 1991; Pedersen et al., 1991). Briefly, a multidimensional questionnaire was sent out in 1984 to all surviving pairs of twins reared apart (TRA) and a matched sample of

twins reared together (TRT). Both members of 351 TRA pairs and 407 TRT pairs responded to the questionnaire. The sample of responders included 99 pairs of monozygotic twins reared apart (MZA), 238 pairs of dizygotic twins reared apart (DZA), 166 pairs of monozygotic twins reared together (MZT), 221 pairs of dizygotic twins reared together (DZT), and 34 pairs of twins of unknown zygosity. The twin analyses presented here are based on the 724 pairs with known zygosity. Among the TRAs, 48% were separated by their first birthday, 64% by the age of 2, and 82% by age 5. Ages range from 26–86 years (M = 58.6; SD = 13.6); 72% of the twins were older than 50, and 60% were female.

Measures. — The questionnaires assessed several dimensions of physical health and functioning. Two health measures based on self-report are included in this study. The first scale (SUMILL) represents the objective component of health. It surveys general health status and measures the number of organ systems reported to be affected by a chronic health problem. SUMILL was designed to index "constricted homeostasis" (Rowe, 1985) brought about by a general age-related decline in the function of the body's organ systems. Thus, it reflects a general reduction in physiological resilience that is not associated with a specific diagnosis. The scale is based on answers to 51 health items inquiring whether the participant has, or has ever had, particular health problems or diagnoses. Most of the health questions are from the OARS (Duke University, 1978) health battery. The items were reduced to 13 categories representing organ systems or a positive diagnosis of cancer. Each item was assigned to only one of the following categories: cardiovascular, respiratory, neurologic, metabolic, gastrointestinal, musculoskeletal, urologic, female reproductive, visual, auditory, skin, allergies, and cancer. An individual's score is the summed total of categories that were reported to be affected by at least one chronic health problem.

The designation and assignment of items to the illness categories were based on recommendations from physicians at the Karolinska Institute in Stockholm, as well as reports in the literature that describe this type of scale (Bayer, Whissell-Buechy, & Honzik, 1981; Liang, 1986). Several investigations into the validity of self-reported illnesses have been conducted, and the general conclusion is that these self-ratings are fairly objective measures of health (Fillenbaum, 1978; Liang, 1986; Rosencranz & Pihlblad, 1970). Furthermore, previous research based on a Swedish sample revealed 90–100% congruence between responses from interviews and questionnaires for health-related items (Cederlöf, Friberg, & Lundman, 1977).

The subjective component of health is measured by a self-rated health scale (SRHEALTH). Gerontological research frequently employs self-rated health because it encompasses functional and physical status within the context of aging (Liang, 1986; Maddox & Douglass, 1974; Stenback, Kumpulainen, & Vauhkonen; 1978; Tessler & Mechanic, 1978). The scale consists of the following four questions: (1) How would you rate your general health status? (2) How would you rate your health status compared to 5 years ago? (3) How would you rate your health status compared to others in your age group? (4) Do you think your health prevents you from doing things you would like to do? This scale is similar to self-rated health in the OARS but has the additional item listed as number 3 above. Because the metrics for the items were not isomorphic, the items were standardized separately (M = 0, SD = 1) and then summed. A high score indicates a more positive health rating. Cronbach's alpha is .76.

In addition to the health measures, self-reported height in centimeters and weight in kilograms were also analyzed. Height and weight are important because genetic factors contribute substantially to variation in both phenotypes, and individual variation is large. Thus, they are useful for assessing the representativeness of the twin data in this sample.

Analytical procedures. — Age differences in individual variation and its etiology were explored using structural equation modeling by means of LISREL VI (Jöreskog & Sörbom, 1986). Members of like-sexed twin pairs are perfectly correlated for gender, and this can inflate measures of twin similarity if gender is correlated with the phenotype. To correct for possible main effects of gender, all the scales scores were residualized using a regression technique (McGue & Bouchard, 1984).

The hypothesis that individual differences are invariant across age was examined by testing the fit of a model that constrained the total variances to be equal across four age groups: less than 50 years old, 50–59 years old, 60–69 years old, and greater than 70. An acceptable fit implies no age differences in individual variability. Conversely, an unacceptable fit signifies age differences in individuality, but it does not identify where these differences occur.

Genetic and environmental analyses. — The adoption/twin design provides a powerful methodology to separate and test how genetic and environmental effects contribute to similarity and differences among twins (Plomin, DeFries, & McClearn, 1990). Resemblance of twin pairs reared together arises from shared genes and shared environments. Genetic influences are typically subdivided into additive (G_a) and nonadditive (G_{na}) components. The distinction refers to the way in which alleles co-act to determine expression of the phenotype: The additive component reflects the summed effects of alleles within and across loci, and the nonadditive component includes the interaction of alleles within (dominance) and across (epistasis) different loci.

Environmental effects may also contribute to twin similarity. Shared rearing environment (E_s) pertains to experiences shared by twins who grew up together. The importance of E_s is indicated if twins reared together are more similar than twins reared apart. Correlated environments (E_{corr}), refer to other shared environmental influences, such as post-rearing experiences, that may contribute to twin similarity regardless of the rearing status. Contact with one's co-twin during adulthood is an example of a correlated environmental effect that may be particularly relevant for older twins. Effects due to E_{corr} represent residual similarity not accounted for by genetic or shared rearing environmental influences. Finally, nonshared environmental (E_{ns}) influences are experiences that are unique to individuals and therefore contribute only to differences within twin pairs. Errors of measurement, which contribute to twin differences, are included in E_{ns}.

Phenotypic resemblance among twins is represented by the intraclass correlation, t, which is a ratio expressing the sum of shared sources of variation to total variation. The types of genetic and environmental effects contributing to variation, and estimates of their relative importance, may be assessed by comparing the intraclass correlations across the groups defined by zygosity and rearing status (Jinks & Fulker, 1970). However, in this study the correlations are used to guide model-fitting analyses which have the advantages that they have greater power, multigroup data are employed to produce a simultaneous solution, and comparative modeling can be conducted.

Several reports have described the basic model for the SATSA data (e.g., Pedersen, Gatz, Plomin, Nesselroade, & McClearn, 1989). In review, the covariances of the twins in the four groups (MZA, MZT, DZA, and DZT) can be expressed in terms of eight equations that provide the basis for generating expected mean squares between and within pairs. The equations reflect the expectations of a biometrical model (Jinks & Fulker, 1970) specifying how G_a, G_{na}, E_{ns}, E_s, and E_{corr} contribute to inter- and intrapair similarities and differences in the four groups defined by zygosity and rearing status. The modeling procedure generates parameter estimates by fitting the observed mean squares to the mean squares expected under the biometrical model and yields a test of the overall fit of the model.

Age effects in the importance of genetic and environmental factors were investigated through comparative modeling. The basic strategy is to determine whether a freed (full) model, which estimates all of the parameters independently for each age group, provides a significantly better fit than a constrained (reduced) model, which specifies that each parameter is equal across ages (e.g., G_a age group 1 = G_a age group 2 = G_a age group 3 = G_a age group 4). The relative fits of these two models can be assessed by testing the difference in chi-square values, which itself is distributed as a chi-square. Age differences are suggested if the freed model provides a significantly better fit over the constrained version. However, this test does not disclose the nature of the age differences. Follow-up models, conducted separately for each age group, are necessary to determine if the age differences arise from: (a) a difference in total variance across the age groups; (b) differences in the proportional contribution of genes and environment across age; or (c) different genetic and environmental effects across age.

Results

Descriptive statistics for the measures of the sample divided into the four age groups are presented in Table 1. Correlations of the measures with age indicated a significant mean trend for SRHEALTH ($r = -.18$, $p = .0001$), SUMILL ($r = .27$, $p = .0001$), height ($r = -.30$, $p = .0001$), and weight ($r = -.06$, $p = .018$). Compared to the younger participants, the older subjects tend to rate their health less positively, report more chronic illnesses, are shorter, and weigh less.

The distribution for SUMILL is positively skewed, indicating that most people score relatively low on this scale. In order to preserve the variance differences across age groups, the scales were not transformed; however, in model-fitting applications, skewness usually inflates the chi-square statistic, thereby increasing the likelihood of a poor fit that is not due to model specification error. Therefore, an alternative fit index, the relative likelihood ratio, was used. This is the ratio of the chi-square to its degrees of freedom; a small value, about 2 or 3 to 1, is indicative of an adequate fit (Jöreskog & Sörbom, 1986).

Age Differences in Individual Variation

As shown in Table 1, variation increased significantly for both SUMILL (χ^2 (3) = 62.34, $p = .000$) and SRHEALTH (χ^2 (3) = 61.00, $p = .000$). For weight, the variances are not homogeneous (χ^2 (3) = 23.21, $p = .000$), but show a nonlinear rise and fall. In contrast to the increased variation for health, individual differences are fairly consistent across age groups for height (χ^2 (3) = 5.30, $p = .151$). Indeed, inspection of the variances listed in Table 1 for this measure does not suggest any linear trend in individual variation for height.

Age Differences in the Etiology of Individual Variation

Intraclass correlations. — The intraclass correlations for the four age groups by zygosity and rearing status are presented in Table 2. For SUMILL, height, and weight, the importance of genetic influences is indicated by MZ correlations that are greater than DZ correlations. Furthermore, genetic influences may be both additive and nonadditive for SUMILL and weight, because the MZ correlations are more than twice the DZ correlations. For SUMILL, the correlations increase considerably in the oldest DZ twins in both rearing groups. This pattern does not conform to a genetic model, but could be attributed to correlated environmental effects, cohort effects that come into play later in life, or both. For SRHEALTH, the pattern of correlations varies

Table 1. Descriptive Statistics for Physical Health and Stature by Age Group

Age Group	*N*	Mean	Variance	Minimum	Maximum
SUMILL					
<50 years	394	1.39	1.64	0	8
50–59	286	1.61	1.88	0	6
60–69	425	2.12	2.79	0	8
≥70	297	2.55	3.51	0	10
SRHEALTH					
<50 years	393	0.83	5.09	−9.1	5.2
50–59	282	0.34	8.01	−9.1	5.2
60–69	435	−0.18	9.75	−9.1	5.2
≥70	299	−0.59	11.20	−9.1	5.2
Height					
<50 years	392	170.78	78.73	150	198
50–59	288	167.95	89.92	150	191
60–69	436	166.91	79.04	141	188
≥70	305	163.18	74.37	143	187
Weight					
<50 years	396	68.37	152.59	42	114
50–59	290	70.57	183.06	47	140
60–69	438	70.03	145.48	40	120
≥70	309	65.36	105.54	40	101

Table 2. Intraclass Correlations for Measures of Physical Health and Stature by Zygosity, Rearing, and Age Group

Group[a]	Age Group <50	50–59	60–69	⩾70
SUMILL				
MZA	.55 (22)**	.43 (18)	.49 (36)**	.33 (22)
MZT	.47 (47)***	.41 (37)**	.48 (42)***	.41 (29)*
DZA	.13 (72)	.01 (48)	.11 (58)	.41 (41)**
DZT	−.11 (55)	.11 (37)	.23 (67)	.51 (51)***
SRHEALTH				
MZA	−.01 (22)	.19 (18)	.40 (35)**	.40 (21)
MZT	.25 (46)	.29 (37)	.20 (48)	.16 (29)
DZA	.28 (72)*	.21 (46)	.26 (62)*	−.01 (41)
DZT	.19 (53)	.25 (36)	.04 (68)	.28 (54)*
Height				
MZA	.89 (22)***	.86 (17)***	.89 (33)***	.64 (22)***
MZT	.88 (45)***	.83 (39)***	.72 (44)***	.84 (29)***
DZA	.51 (73)***	.56 (48)***	.50 (64)***	.23 (43)
DZT	.32 (52)*	.71 (38)***	.41 (70)***	.42 (72)***
Weight				
MZA	.76 (22)***	.82 (19)***	.58 (33)***	.72 (22)***
MZT	.74 (46)***	.79 (38)***	.71 (48)***	.66 (30)***
DZA	.29 (75)**	.44 (49)**	.48 (65)***	.08 (45)
DZT	.36 (53)**	.16 (38)	.48 (70)***	.22 (57)

Note. Number of twin pairs is in parentheses.

[a]MZA = monozygotic twins reared apart; MZT = monozygotic twins reared together; DZA = dizygotic twins reared apart; and DZT = dizygotic twins reared together.

*$p < .05$; **$p < .01$; ***$p < .001$.

considerably across age groups. There is some evidence of genetic effects, but differences between the MZ and DZ values are less than for the other measures. Rather, the correlations show that environmental effects unique to the individual contribute substantially to variability in self-rated health. In general, there is little evidence for differential twin similarity dependent on rearing status (i.e., the TRTs are not consistently more similar than the TRAs). Thus, sharing the same rearing environment may not contribute substantially to variation for these measures.

Modeling analyses. — Copies of the LISREL programs and input data matrices are available from the authors upon request. As described earlier, tests for significant age differences in the genetic and environmental effects were explored through comparative modeling. Selection of the particular parameters modeled for each phenotype was based on the intraclass correlations. The models and results from the chi-square difference test are reported in Table 3.

For both health measures, the freed model fit significantly better than did the constrained version, indicating age differences in the parameter estimates. This confirms conclusions based on the twin correlations alone for SUMILL because the MZ correlations are greater than the DZ correlations in all of the age groups younger than 70 but not in the oldest age group. An additional analysis testing for age differences in the three younger groups was completed for SUMILL. The parameters included nonadditive genetic, nonshared environmental, and correlated environmental effects. Results

Table 3. Tests for Age Differences in Genetic and Environmental Contributions to Variation

Measure	Model	χ^2 Difference[a]	df	p
SUMILL	$G_{na} + E_{ns} + E_{corr}$	58.90	9	.000
SRHEALTH	$G_a + E_{ns} + E_{corr} + E_s$	55.10	12	.000
Height	$G_a + E_{ns}$	7.21	6	.313
Weight	$G_a + G_{na} + E_{ns}$	15.39	9	.085

[a]Chi-square difference test of the model constrained across all age groups compared to the model in which the parameters are estimated independently for each age group.

also showed significant age differences (χ^2 difference = 26.77 (6), $p = .000$), even though the oldest group was excluded from this analysis.

In contrast to the health measures, age differences were not significant for either height or weight, implying that genetic and environmental effects do not contribute differentially to variation across age. These findings are consistent with the pattern of intraclass correlations (Table 2), which show that twin similarity in the different groups is more consistent for height and weight than for the health measures.

Subsequent analyses were completed to determine the nature of differences in the genetic and environmental contributions to variation in health. Each age group was analyzed separately to find the best-fitting model that was also congruent with the intraclass correlations. The absolute and proportional variance components for the genetic and environmental parameters for each age group are reported in Table 4. For both health measures, the variances generated by the model recapture the observed variability across the age groups.

Different effects account for the substantial increase in total variance for SUMILL. Across the three youngest groups, there is a twofold increase in genetic variation, as well as a gradual rise in the effects of nonshared environment. However, sizable effects due to correlated environments, which did not play a role before age 70 and were either estimated as zero or unidentified in the models, emerge after age 70.

Figure 1 presents the variance decomposition across age groups as estimated from the models for SUMILL. The graphic representation shows clearly that both the relative and absolute variance values differ across age, first due to genetic effects and then due to nonshared and correlated environmental effects after age 70.

The outcomes were quite different for SRHEALTH (Table 4). The freed model revealed that twin similarity is primarily due to correlated environments in the two younger groups and is explained by genetic effects in the two older groups. However, in all groups, most of the variation is attributable to the influence of nonshared environment, which accounts for the large increase in phenotypic variance (Figure 2). Despite the increase in the variance associated with nonshared environments, its relative contribution to total individual differences does not vary across age groups.

Because there were no significant age differences for height and weight, the final analyses were based on data pooled over age. For height, 83% of the variation was due to

Table 4. Total Variance Described in Terms of Its Genetic (G) and Environmental (E_{ns} and E_{corr}) Components Across Age Groups for Health Measures for the Freed Models

Age Group	Total Variance	Absolute (±*SE*) G	Absolute (±*SE*) E_{ns}	Absolute (±*SE*) E_{corr}	Relative[a] G	Relative[a] E_{ns}	Relative[a] E_{corr}	χ^2	df	*p*	χ^2/df
				SUMILL[b]							
<50	1.64	0.70 ± .10	0.94 ± .08	—	43%	57%	—	8.95	6	.18	1.49
50–59	1.85	0.74 ± .13	1.11 ± .09	—	40%	60%	—	1.30	6	.97	0.22
60–69	2.75	1.48 ± .11	1.28 ± .09	—	54%	46%	—	9.67	6	.14	1.61
≥70	3.36	—	1.82 ± .08	1.55 ± .13	—	54%	46%	14.65	6	.02	2.44
				SHRHEALTH[c]							
<50	5.25	—	4.06 ± .10	1.19 ± .18	—	77%	23%	3.02	6	.81	0.50
50–59	7.86	—	5.93 ± .15	1.93 ± .26	—	75%	25%	4.10	6	.66	0.68
60–69	9.87	2.86 ± .27	7.02 ± .17	—	29%	71%	—	9.40	6	.15	1.57
≥70	10.87	2.85 ± .38	8.02 ± .23	—	26%	74%	—	6.07	6	.42	1.01

Note. *SE*s are given for the square roots of the absolute variance estimates.
[a]Percentage of total observed variance, which is G, E_{ns}, or E_{corr}.
[b]The genetic parameter estimated is nonadditive (G_{na}). E_{corr} was only estimated in the oldest age group.
[c]The genetic parameter estimated is additive (G_a).

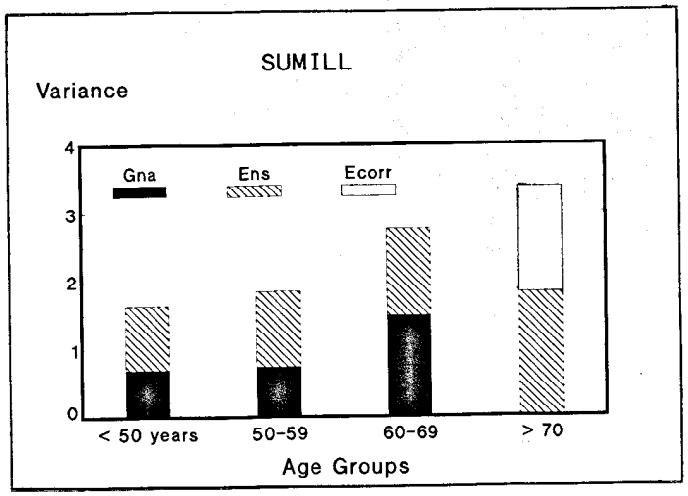

Figure 1. Variance decomposition for SUMILL into genetic (G_{na}), nonshared (E_{ns}), and correlated environmental (E_{corr}) components.

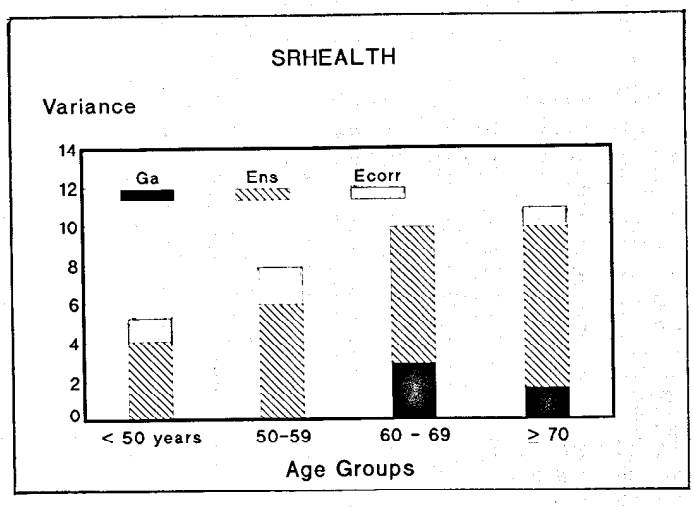

Figure 2. Variance decomposition for SRHEALTH into genetic (G_a), nonshared (E_{ns}), and correlated environmental (E_{corr}) components.

genetic effects and 17% was attributable to nonshared environments ($\chi^2(6) = 7.28, p = .296$). For weight, 75% of the variance was accounted for by genetic effects and the remaining 25% by nonshared environment ($\chi^2(6) = 12.24, p = .057$). Although the fit of this model is borderline, the ratio of chi-square to degrees of freedom is 2.0, indicating that the model is acceptable.

DISCUSSION

Mean Age Differences

The correlations of the measures with age were significant for SUMILL and SRHEALTH. Older participants reported more health-related problems and rated themselves "less healthy" than younger participants. The findings here are at odds with reports that the elderly tend to assess their health more positively than younger individuals (Cockerham, Sharp, & Wilcox, 1983; Linn & Linn, 1980). To date, there is no consensus in the literature about average trends in self-rated health. It may be that age differences in self-rated health are more complicated than those defined by a simple linear trend. For instance, a fine-grained age analysis revealed that the old-old tend to rate their health as better than the young-old (Essex, 1986). Furthermore, another study did not find average age differences in health self-ratings per se, but did find significant age differences in the factors that affected those ratings (Jylhä et al., 1986). These results suggest that self-rated health must be ascertained in the context of an individual's age and own illness experience.

The average drop in ratings of health across age in this study is in agreement with a more recent explanation about why self-rated health scores may be less positive among the

elderly. Although collective health (measured objectively) has improved, subjective feelings of healthiness have declined in some cultures, partially because people may expect better health given medical advances (Barsky, 1988). Thus, cohort effects may occur for this measure.

Age Differences in Individual Variation

The general expectation that interindividual differences increase with age (Baltes et al., 1980; Baltes & Reese, 1984) has been debated by Bornstein and Smircina (1982), whose review of the literature led them to conclude that stable variability is the rule. Their conclusion may be premature because few studies have investigated this question, and it is most probable that age-related differences in individuality depend on the phenotype of interest. Indeed, cross-sectional analyses of the personality scales in the SATSA revealed four different patterns of age differences in variability: a *classic* pattern showed increased differences, a *uniform* pattern showed no differences, a *constricting* pattern showed decreased differences, and an *irregular* pattern showed nonlinear differences (McClearn et al., 1991). Only six of 22 scales displayed the classic pattern. The health data in this study support the classic pattern: Both SUMILL and SRHEALTH show a significant increase in interindividual differences, but height and weight did not.

Age Differences in the Etiology of Individual Variation

Given that individual differences increase with age, the next question was to determine which effects account for this excess variation. Evidence of age differences in the genetic and environmental influences was found for the health measures, but not for height or weight. The modeling outcomes revealed that these age effects are characterized by significant differences in the genetic and environmental contributions to variation and are not due merely to the increase in variation across age groups. Furthermore, the actual pattern of age differences in the variance components were not the same for the two health measures.

SUMILL. — Significant age differences in the etiology of individuality are primarily related to an increase in genetic variance across the three younger age groups. The picture changes for the group older than 70 years, where there is a convergence of MZ and DZ correlations accounted for by a dramatic increase in similarity among the DZ twins. The result is that the parameter for correlated environments, rather than the parameter for genetic effects, is significant for the oldest group.

It is difficult to interpret this biphasic genetic effect for SUMILL from these cross-sectional data. One explanation is that the values for twins in the oldest age group reflect a cohort effect. The twins who were at least 70 years old at the time of the questionnaire were born before industrialization of Sweden, and the important influences contributing to variation in health could be vastly different for those who benefited from the medical progress that accompanied the industrialization.

This is not the first twin study to report a convergence of MZ and DZ scores in old age. In a 21-year longitudinal study of aging twins, Jarvik and Bank (1983) reported that similarity was greater in the identical than the fraternal pairs at initial testing, but this differential twin resemblance by zygosity disappeared over time. For digit span, the fraternal twins were actually more similar than the identical twins at the last measurement occasion. The authors posited that convergence of identical and fraternal twin similarity is partially explained by the longitudinal design itself, because the fraternal pairs who are least similar for traits related to survival drop out. Indeed, blood group data revealed that the fraternal pairs who remained in the sample were more similar at initial testing than those who had dropped out. However, due to substantial sample attrition at the 21-year follow-up, these findings should be interpreted cautiously.

In the case of SUMILL, one might expect that differential sample attrition forces a bias such that only the most similar dizygotic twins are participating in the study in the oldest age group. This could occur if an individual from a dizygotic twin pair experiences an event, such as illness, which would lead to the exclusion of the least similar pairs from the sample. The sample bias would be most pronounced in senescence after many pairs had been dropped.

Whereas we do not have adequate longitudinal data in the SATSA sample to investigate such a bias, there are illness data from two points of measurement for a subsample (339 pairs) of the SATSA twins. A reduced version of the SUMILL scale was constructed from questionnaire data in 1963 and the 1984 SATSA mailout (Harris, 1988). Preliminary findings did not support the sample attrition hypothesis for the illness scale, because the DZ twin pairs who remained in the longitudinal sample were not more similar at the initial assessment than the DZ twin pairs who dropped out. Rather, the longitudinal results mirrored the cross-sectional findings from the present study and revealed a cohort effect. For the cohort younger than 70 years old in 1984, there was little change in MZ and DZ correlations from 1963 to 1984. However, for the cohort of twins who were at least 70 years in 1984, there was a clear convergence of MZ and DZ correlations from 1963 to 1984, and genetic effects disappeared. The differential pattern of change in twin similarity across 21 years for the two cohorts is in agreement with the cross-sectional results presented here and suggests that experiences common to the cohort born prior to 1914 contribute significantly to variation in health for the oldest twins in this sample.

Self-rated health. — In contrast to SUMILL, the increase across age in variability for SRHEALTH is primarily accounted for by nonshared environmental influences. These findings are consistent with life-span paradigms, which propose that the cumulative effects of unique experiences will result in an increase in interindividual differences. It is not surprising that it is the nonshared environmental influences that become increasingly more important for self-rated health, because ratings for this measure are strongly mediated by experiences that are associated with aging (Essex, 1986). These experiences (such as impairment, illness, or death of one's peers) are individually experienced environmental effects. Clearly, further research is needed to identify more specifically the nature of these nonshared environmental effects that are operating.

Sample Representativeness

Heritabilities for height and weight are frequently used to determine comparability of different twin samples. One important comparison is between this sample and the population-based Swedish Twin Registry. There is substantial conformity between the intraclass correlations in this study and those reported for height and weight from data collected in 1963 on more than 18,000 pairs of twins in the Swedish Twin Registry (Harris, 1988). Other comparisons are also relevant: The average heritability in most twin samples is approximately .80 for height and is generally lower (.60) for weight (Kaprio, Sarna, Koskenvuo, & Rantasalo, 1978). The data reported here reflect these general expectations and indicate that this sample is representative of twin samples.

CONCLUSIONS

The purpose of this study was to explore age differences in genetic and environmental sources of variation for health. In summary, cross-sectional data suggest that interindividual differences increase during the later part of the life span for measures representing different aspects of health status, but not for measures of stature. More exciting, however, is the finding that age differences in the etiology of individuality are significant. For SUMILL, which is a measure of reduced homeostasis, genetic variance seems to account for increasingly more of the differences in health until age 70, when a type of correlated or shared environmental effect may appear. This environmental influence may signify a cohort effect for the twins who were born prior to 1914 and industrialization in Sweden. In comparison, SRHEALTH, which is a more subjective measure of health status, is characterized by a considerable increase in variation that is almost entirely attributable to an increase in nonshared environmental influences unique to the individual.

As demonstrated by these data, whether genetic or environmental effects become relatively more important across age depends upon both the phenotype and the age range sampled. The findings presented here, based solely on cross-sectional data, are the first step in examining individual differences in health across the life span. Ultimately, conclusions about the genetic and environmental influences that contribute to individual variation will rest on the triangulation of research investigating age differences, age changes, and cohort influences.

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