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Genetic and Environmental Factors Affecting Self-Rated Health from Age 16–25: A Longitudinal Study of Finnish Twins

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Abstract We analyzed genetic and environmental determinants of self-rated health and its change from adolescence to early adulthood. Questionnaires were mailed to Finnish twins born 1975–1979 at ages 16, 17, 18½ and, on average, 25 years of age ($N = 2465$ complete twin pairs). The data were analyzed using quantitative genetic methods for twin data by the Mx statistical package. Heritability of self-rated health was greatest at age 16 (63%, 95% confidence intervals (CI) 56–67%, men and women together) and declined steadily to age 25 (33%, 95% CI 25–41%). The residual variation was due to unshared environments. Health ratings at different ages were modestly correlated ($r = 0.33$ – 0.61). These correlations were mainly due to genetic factors, but unshared environment also contributed to them. An important challenge for further research is to identify environmental influences contributing to self-rated health independently of, or in interaction with, genetic factors.

Keywords Self-rated health · Adolescence · Heritability

Self-rated health is a widely used indicator in health research. This indicator is typically based on a single question asking the respondents to rate their current health status on scale from good to bad. Although self-rated health is a “subjective” measure, it has been found to be a good predictor of mortality (Idler and Benyamini 1997), functional disability (Idler and Kasl 1995), and the use of health services (Miilunpalo et al. 1997), suggesting that self-rated health and medically confirmed health indicators are closely interrelated. This is supported by a previous methodological study (Manderbacka 1998) which found that self-rated health is primarily based on physical ill-health and functional disability rather than psychological characteristics or mental health. Previous studies have also indicated that self-rated health is unidimensional rather than multidimensional, i.e., the same factors contribute both to good and bad self-rated health (Manderbacka et al. 1998). Self-rated health is therefore a useful, simple and inexpensive indicator of a person’s general health status.

Although the demographic, socioeconomic and psychosocial determinants of self-rated health, as well as its predictability of further health outcomes, have been extensively studied, the genetic architecture of self-rated health remains poorly understood. In previous twin studies from Denmark (Christensen et al. 1999), Finland (Leinonen et al. 2005), Sweden (Harris et al. 1992; Svedberg et al. 2001), Norway (Røysamb et al. 2003) and the USA (Romeis et al. 2000) environmental factors not shared by family members

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explain a major part, i.e. from 60% to over 90%, of the variation of self-rated health. These studies have, however, produced contradictory results on whether the remaining phenotypic variation is due to common environmental factors, i.e. environment shared by family members, or to genetic factors. These inconclusive results are probably partly due to small sample sizes in some of these studies but may also be explained by differences in age between the study samples, as it is possible that heritability of self-rated health changes with age. Previous studies have shown, for example, a clear change in heritability of intelligence as genetic factors become more important from childhood to adulthood (Plomin and Spinath 2004). The heritability of self-rated health in adolescence or the changes of it during ageing have not been studied.

In this study, we investigate the relative contribution of genetic and environmental influences on self-rated health, using a longitudinal design, based on identical measurement of self-rated health on four occasions from adolescence, i.e. 16 years, to early adulthood, i.e. 25 years, in a sample of 2465 complete Finnish twin pairs.

Data and methods

The data were derived from the *FinnTwin16* study cohort described in detail elsewhere (Kaprio et al. 2002). A baseline survey questionnaire was sent during the years 1991–1995 to all Finnish twins born 1975–1979 within 2 months after their 16th birthday. Zygosity was defined using questions on physical similarity during school age. This method has shown high reliability in Finnish twin data (Sarna et al. 1978). The number of twin individuals with known zygosity was 4940 including 2465 complete twin pairs. The response rate was 88% in this baseline survey. Three follow-up questionnaires were sent to all persons who participated in the baseline survey. The first follow-up questionnaire was sent in the month after the 17th birthday, the second follow-up questionnaire, on average, 6 months after the 18th birthday and the third follow-up questionnaire at semi-annual intervals during the years 2000–2002 when the participants were, on average, 25-years-old (range 22–27 years). The response rates in these follow-up surveys were 90%, 95% and 88%, respectively. In each survey, one reminder was sent to those who did not respond to the questionnaire, and subsequently, non-respondents were called by phone. The average delay of response was 40 days in the baseline survey and 29, 28 and 31 days in the follow-up surveys, respectively. The exact mean ages at

the time when the respondent returned the questionnaire were 16.2 (SD 0.14), 17.1 (SD 0.08), 18.6 (SD 0.17) and 24.5 (SD 0.94) years, respectively.

In each of the four waves of assessment, self-rated health was assessed by an identical question that offered five preset response alternatives. The question reads: “What do you think about your current health status? Is it (1) Very good; (2) Fairly good; (3) Average; (4) Fairly poor; or (5) Poor”. Because very few respondents reported their health as poor, this category was combined with fairly poor health.

The data were analyzed using quantitative genetic methods for twin data based on structural equation modeling (Neale and Cardon 1992). The models were fitted by the Mx statistical package using raw data input (Neale 2003). Classical twin analysis allows decomposition of the phenotypic variation into additive genetic variation (A), dominance genetic variation (D), which includes the interaction of alleles in the same locus (dominance) as well as the interaction between the alleles over all relevant loci (epistasis), environmental variation common to co-twins (C) and environmental variation unique to each twin individual including measurement error (E). Because we only have information on monozygotic (MZ) and dizygotic (DZ) twin pairs reared together, dominance genetic and common environmental effects cannot be simultaneously modeled. Also using twin data only, we cannot determine the possible effects of assortative mating and gene–environment interaction and have to assume that both are negligible. If phenotypic assortment by health exists, this will inflate DZ correlations and may consequently cause overestimation of the common environmental variance component and underestimation of heritability. The presence of gene–environment interaction is confounded with the additive genetic component. In other words, the additive genetic component estimated in the current twin sample may include both a main effect of genetic factors and genetic differences in susceptibility to environmental conditions.

As self-rated health was measured on an ordinal scale, a threshold model was used to estimate the contributions of genetic and environmental factors. We first modeled self-rated health separately at each age, where we tested whether the thresholds of self-rated health were similar for males and females and for MZ and DZ twins using nested likelihood ratio tests. The best fitting models were used in subsequent multivariate modeling, in which we used a Cholesky decomposition of the variance (Fig. 1). Such modeling assumes that specific genetic and environmental factors affect each phenotype but these factors can also affect other

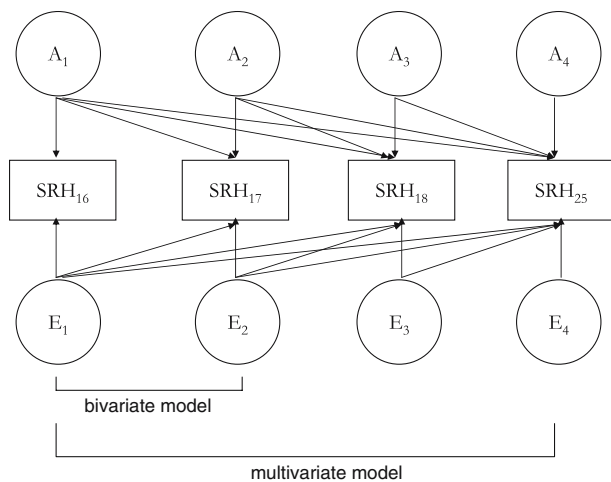


Fig. 1 Schematic presentation of full Cholesky model for self-rated health at ages 16, 17, 18 and 25 years (A_1 – A_4 additive genetic variance components, E_1 – E_4 unshared environmental variance components, SRH_{16} – SRH_{25} self-rated health at 16–25 years of age)

phenotypes or, as in this study, the same phenotype at different ages. Thus, the Cholesky decomposition allows the phenotypic variation and covariance in self-rated health measures between different ages to be ascribed to common genetic and environmental factors affecting these measures. We firstly decomposed pairwise correlations of self-rated health between different ages to genetic and environmental correlations using separate bivariate models. Further, we tested hypotheses of the genetic architecture of the change in self-rated health from age 16 to 25 comparing restricted tetravariate Cholesky models to a full Cholesky model. We tested whether there is only one genetic component (A_1) affecting self-rated health measures at 16–25 years of age by eliminating all pathways from other genetic components (A_2 – A_4). Secondly we tested

whether specific environmental factors affecting self-rated health are unique to each age by allowing each unshared environmental component (E_1 – E_4) to affect self-rated health at one age only.

Self-rated health was treated as an ordinal level variable in the statistical modeling using a threshold model and assuming that a normally distributed standardized liability function underlies the observed measures of self-rated health. For this reason, we also computed polychoric correlations instead of intraclass correlations. During the course of modeling, we found that fitting a tetravariate Cholesky model using ordinal level data is problematic because this model is very sensitive to starting values of the parameters in the model. We solved this problem by treating self-rated health as an ordinal level variable in univariate and bivariate models but as a continuous variable in tetravariate models. The tetravariate Cholesky models were used only to test the hypothesis on the underlying genetic architecture but not to estimate parameters.

Results

Table 1 presents the proportion of participants at each age in the categories of self-rated health. In both men and women, 80–90% of the participants reported their health as very or fairly good. The proportion of participants who reported their health as very good was higher in men and MZ twins than in women and DZ twins. This proportion also declined from age 16 to 25 years in both sexes. The proportion of respondents reporting less than good health increased in women, but remained stable in men across the age groups.

Polychoric correlations of self-rated health within twin pairs at each age are presented in Table 2. MZ correlations were higher than DZ correlations and no

Table 1 Proportion of participants by self-rated health at ages 16, 17, 18 and 25 by sex and zygosity

	MZ twins				DZ twins			
	16 years (%)	17 years (%)	18 years (%)	25 years (%)	16 years (%)	17 years (%)	18 years (%)	25 years (%)
<i>Males</i>								
Very good	46	45	47	39	43	41	40	36
Fairly good	42	40	40	49	44	46	45	51
Average	11	14	11	11	12	12	13	11
Fairly or very bad	1	1	2	1	1	1	2	2
N	664	609	595	526	1707	1591	1585	1398
<i>Females</i>								
Very good	41	34	34	29	34	30	27	28
Fairly good	47	53	51	54	51	54	53	54
Average	11	12	13	15	14	15	18	16
Fairly or very bad	1	1	2	2	1	2	2	2
N	926	911	898	843	1643	1599	1595	1503

Table 2 Number of complete twin pairs and polychoric correlations within MZ, same-sex DZ and opposite-sex DZ twin pairs for self-rated health at ages 16, 17, 18, and 25

Age	MZ		DZ		MZ		DZ		DZ	
	Males		Same-sex males		Females		Same-sex females		Opposite-sex pairs	
	<i>N</i>	<i>r</i>	<i>N</i>	<i>r</i>	<i>N</i>	<i>r</i>	<i>N</i>	<i>r</i>	<i>N</i>	<i>r</i>
16	331	0.59	426	0.35	462	0.65	395	0.35	851	0.13
17	299	0.54	390	0.34	452	0.56	381	0.20	785	0.21
18	288	0.45	389	0.20	445	0.54	376	0.14	779	0.19
25	243	0.31	304	0.11	402	0.38	342	0.13	661	0.14

systematic differences were seen between men and women in the magnitude of the correlations. At age 16 years, the opposite-sex DZ correlation was lower than the same-sex DZ correlations in men and women, but at later ages no corresponding difference was seen.

We started the statistical modeling by exploring the best fitting univariate model at each age (Table 3). Thresholds in men and women differed statistically significantly (saturated model 2, $P < 0.001$) at all ages. At age 16, the threshold differed for MZ and DZ twins

Table 3 Model fit statistics for univariate models for self-rated health at ages 16, 17, 18 and 25

	Age 16	Age 17	Age 18	Age 25
<i>Saturated model 1</i>				
χ^2	9775	9465	9744	8864
d.f.	4905	4675	4639	4235
<i>Saturated model 2 (same thresholds for men and women)</i>				
$\Delta\chi^2$ compared to saturated model 1	66	86	112	51
Δ d.f. compared to saturated model 1	27	27	27	27
<i>P</i> -value of $\Delta\chi^2_{d.f.}$	$P < 0.001$	$P < 0.001$	$P < 0.001$	$P < 0.001$
<i>Saturated model 3 (same thresholds for MZ and DZ twins)</i>				
$\Delta\chi^2$ compared to saturated model 1	38	31	30	13
Δ d.f. compared to saturated model 1	24	24	24	24
<i>P</i> -value of $\Delta\chi^2_{d.f.}$	$P = 0.038$	$P = 0.170$	$P = 0.193$	$P = 0.962$
<i>ACE model¹</i>				
$\Delta\chi^2$ compared to saturated model	21	23	18	13
Δ d.f. compared to saturated model	18	18	18	18
<i>P</i> -value of $\Delta\chi^2_{d.f.}$	$P = 0.305$	$P = 0.179$	$P = 0.433$	$P = 0.812$
<i>AE model 1¹</i>				
$\Delta\chi^2$ compared to saturated model	1.10	1.50	0	0
Δ d.f. compared to saturated model	2	2	2	2
<i>P</i> -value of $\Delta\chi^2_{d.f.}$	$P = 0.578$	$P = 0.472$	$P = 1.000$	$P = 1.000$
<i>AE model 2¹</i>				
$\Delta\chi^2$ compared to AE model 1	1.49	0.06	1.47	0.72
Δ d.f. compared to AE model 1	2	2	2	2
<i>P</i> -value of $\Delta\chi^2_{d.f.}$	$P = 0.475$	$P = 0.969$	$P = 0.480$	$P = 0.699$
<i>AE model 3¹</i>				
$\Delta\chi^2$ compared to AE model 1	15	1.74	0.61	0.18
Δ d.f. compared to AE model 1	1	1	1	1
<i>p</i> -value of $\Delta\chi^2_{d.f.}$	$P < 0.001$	$P = 0.188$	$P = 0.436$	$P = 0.670$

¹ Different thresholds for men and women and for MZ and DZ twins

ACE model = additive genetic/ common environment/ unshared environment model with different variance components for men and women and sex-specific genetic effect

AE model 1 = additive genetic/ unshared environment model with different variance components for men and women and a sex-specific genetic effect

AE model 2 = additive genetic/ unshared environment model with the same variance components for men and women and sex-specific genetic effect

AE model 3 = additive genetic/ unshared environment model with different variance components for men and women and no sex-specific genetic effect

(saturated model 3, $P = 0.038$) but it was not statistically significant at later ages. An additive genetic/common environment/unshared environment (ACE) model, with different thresholds for men and women as well as for MZ and DZ twins, fitted well to the data, and we found no statistically significant decline in the fit of the model, compared to the saturated model, at any age. This suggests that the assumptions of twin modeling were not violated. A model that included additive genetic and unshared environmental variance components was most parsimonious; no difference in the magnitude of the variance components between men and women was observed (AE model 2). Sex-specific genetic effects were found at age 16 ($P < 0.001$) but not at later ages (AE model 3). Thus, in further univariate and multivariate modeling, we used the AE model with different threshold values for men and women, as well as different thresholds for MZ and DZ twins, but the same additive genetic and unshared environmental variance components for men and women, and a sex-specific genetic effect at age 16, but not at later ages.

Figure 2 presents the estimates of variance components of additive genetic and specific environmental factors in the final AE univariate models at each age (men and women together). The genetic architecture of self-rated health showed a clear age pattern. The proportion of the variation of self-rated health explained by additive genetic factors was highest at age 16 (63%, 95% CI 56–67%). After age 16, the proportion of additive genetic variance declined steadily and was lowest at age 25 (33%, 95% CI 25–41%).

Table 4 presents the polychoric correlations of self-rated health between different ages and additive genetic and unshared environmental correlations behind these cross trait correlations. The magnitude of the cross trait correlations declined along with increased time between the surveys. The correlation of

self-rated health at age 17 was 0.58 with self-rated health at age 16 and 0.61 with self-rated health at age 18. The correlation between self-rated health at ages 16 and 25 was much lower, i.e. 0.33. Both additive genetic and unshared environmental correlations were statistically significant. Additive genetic correlation explained a larger proportion of the trait correlations of self-rated health compared to unshared environmental correlation and increased more in importance as a function of the time between the surveys: additive genetic correlation explained 67% of the correlation of self-rated health between ages 17 and 18 but as much as 83% of the trait correlation between ages 16 and 25.

Finally we fitted a tetrivariate Cholesky model to test hypotheses on the genetic architecture of the change of self-rated health from age 16 to 25 (models not shown). We firstly tested the hypothesis that there is only one genetic factor affecting self-rated health at each age. This model fitted the data poorly compared to a full Cholesky model ($\Delta\chi^2_6 = 195$, $P < 0.001$) suggesting that there are different genetic factors affecting self-rated health at different ages. Secondly, we tested whether there are no unshared environmental factors common to self-rated health at different ages but only specific unshared environmental factors at each age. The model fitted the data poorly ($\Delta\chi^2_6 = 127$, $P < 0.001$) and the hypothesis was consequently rejected. This result was in accordance with those based on bivariate models showing statistically significant unshared environmental correlations.

Discussion

Our results suggest that genetic factors are likely to have major effects on self-rated health. The magnitude of these effects declined with age. The genetic effects were strongest in adolescence when more than half of the variation of self-rated health was accounted for by genetic variation, and it then declined to a third of the total variation in early adulthood. This declining role of genetic factors was somewhat unexpected since if genetic factors affect self-rated health through, for example, health behaviors, one might expect that this effect would strengthen rather than weaken during ageing. Such a mechanism has previously been found on intelligence from adolescence to adulthood (Plomin & Spinath 2004). The remaining variation of self-rated health was due to unshared environmental factors whereas common environmental factors did not have any effects. The proportion of the variation of self-rated health explained by additive genetic and unshared environmental factors was similar in men and

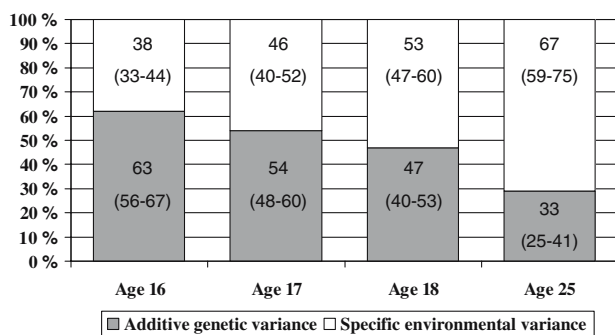


Fig. 2 Proportion of the variation of self-rated health at ages 16, 17, 18 and 25 years accounted for variation by additive genetic and unshared environmental factors

Table 4 Polychoric correlations of self-rated health between ages 16, 17, 18 and 25 and correlations between additive genetic and unshared environmental factors explaining these trait correlations in bivariate Cholesky models

	Age 1	Age 2	Trait correlation		Additive genetic correlation			Unshared environmental correlation		
			<i>r</i>	95% CI	<i>r</i>	95% CI	% explained of trait correlation	<i>r</i>	95% CI	% explained of trait correlation
16	17	0.58	0.56–0.60	0.43	0.38–0.47	73%	0.16	0.12–0.20	27%	
16	18	0.52	0.50–0.54	0.40	0.35–0.42	75%	0.13	0.12–0.17	25%	
16	25	0.33	0.30–0.36	0.28	0.22–0.33	83%	0.06	0.01–0.11	17%	
17	18	0.61	0.59–0.63	0.41	0.36–0.45	67%	0.20	0.16–0.24	33%	
17	25	0.39	0.36–0.42	0.32	0.27–0.40	80%	0.08	0.03–0.13	20%	
18	25	0.45	0.43–0.47	0.31	0.27–0.36	68%	0.14	0.09–0.20	32%	

women. The only sex related difference in the genetic architecture of self-rated health was a sex-specific genetic effect at age 16. It is possible that this effect is related to sex differences in pubertal timing, because puberty is completed for most girls by age 16 but is still ongoing for most age-matched boys.

Our results on the genetic architecture of self-rated health are largely in accordance with previous studies on the heritability of self-rated health in young and early middle age adults. In a previous Norwegian study on 18–31 year-old men and women (Røysamb et al. 2003), a very similar genetic component was found both in men (38%, 95% CI 28–45%) and in women (27%, 95% CI 22–30%) as that found in the present study at age 25 (33%, 95% CI 25–41%). A US study on Vietnam War veterans with mean age of 38 years (Romeis et al. 2000) also reported a very similar heritability estimate for self-rated health (40%, 95% CI 36–43%). The four other previous twin studies on self-rated health included mainly late middle age or elderly participants (Christensen et al. 1999; Harris et al. 1992; Leinonen et al. 2005; Svedberg et al. 2001). Thus, the results of available twin studies suggest that the heritability of self-rated health ranges from 30 to 40% in early adulthood and middle age. We are unaware of any previous twin study on self-rated health in adolescence. In the present study, the heritability of self-rated health in adolescence was greater than that in early adulthood.

The overall self-rated health status among our study subjects was very good: more than 80% rated their health as very or fairly good, which is to be expected in a young study cohort. However, there were some systematic differences in the health status. Above all, the proportion of those who reported very good health declined among men and women from age 16 to 25. The background of this decline is not fully clear since chronic diseases are unlikely to strongly contribute to health within the studied ages. Also the proportion of women reporting very good health was lower than that among men at all ages. This result is in accordance with

previous studies which have reported a higher prevalence of less than good self-rated health as well as other health problems in women than in men in adolescence (Sweeting 1995; Sweeting and West 2003) and in early adulthood (Rahkonen et al. 1995). Further, we found that the proportion of subjects reporting very good health was higher in MZ than DZ twins. Since MZ twins are more prone to various prenatal and neonatal disorders seen as lower birth weight and higher neonatal mortality in MZ twins, especially monozygotic MZ twins, compared to DZ twins (Loos et al. 1998), it is unlikely that this difference has a biological background. In contrast, it is more likely that the better self-rated health of MZ twins compared to DZ twins is associated with psychosocial factors, such as support from the co-twin or other significant others.

A key focus of this study was to examine the persistence of self-rated health from adolescence to early adulthood. The correlations of self-rated health were not particularly high, i.e. about 0.6, even between subsequent years and they declined markedly with age. A previous Finnish study showed that the test-retest agreement of perceived health was around 70% (Martikainen et al. 1999). Although this can be regarded as fairly good reliability, together with short-term fluctuation in health status it can well explain why the intra-individual correlations were not higher than those found.

Genetic factors explained roughly 70% of the correlations of self-rated health between subsequent years, and this proportion increased to more than 80% with increased time between the measurements of self-rated health. The genetic factors affecting self-rated health were, however, not fully similar from adolescence to early adulthood, but rather new occasion-specific genetic influences appeared at each age. Unshared environmental factors explained the rest of the intra-individual correlations. Even when examining self-rated health between ages 16 and 25, the unshared environmental correlation, albeit quite low, was

nevertheless statistically significant. For successive years, the unshared environmental correlations were moderate. This suggests that the unshared environmental component affecting self-rated health is not fully due to measurement error or short term fluctuation in health, but there are environmental effects on self-rated health which may last from adolescence to early adulthood. Identifying such effects is an important task for further research.

A particularly challenging question is which specific factors underlie behind the variation of self-rated health. A previous methodological study suggested that physical ill-health contributes more to self-rated health than mental ill-health (Manderbacka 1998) and this is also supported by results showing that self-rated health is predictive of mortality (Idler and Benyamini 1997). However, these studies are based on middle aged participants, and in adolescents and young adults the factors affecting self-rated health may be different. If the factors affecting self-rated health differ at different ages, this may also explain the changing heritability of self-rated health over age. For example, many mental disorders show even higher heritability than physical health problems and may contribute to the high heritability estimates of self-rated health in adolescence (Boomsma et al. 2002). In this study, we found that at age 25 the Spearman correlation between self-rated health and the number of psychosomatic symptoms was 0.41 (95% CI 0.37–0.44) in men and 0.37 (95% CI 0.34–0.40) in women. Thus psychosomatic problems are likely to partly explain the variation of self-rated health, but there may also be other factors contributing to it.

Our study is prone to sources of bias common to twin and survey based health studies. The quantitative genetic model used makes the assumptions of random mating and lack of gene–environment interaction. Assortative mating is probably not a problem in this study because it should lead to increasing DZ correlation and consequently to overestimation of the common environmental component, which was not found in this study. However, there may be gene–environment interaction and this estimated as a part of additive genetic component. Thus, a part of the additive genetic effect found in this study may reflect rather genetic based differences in susceptibility to environmental exposures than an independent genetic effect. There are also limitations related to the survey method used. We found that loss to follow-up was not independent of the baseline health status, but the prevalence of less than good self-rated health at age 16 was higher among non-respondents to at least one of the follow-up surveys (15% in men and 18% in women) compared to the respondents to all surveys (12% and

14%, respectively). Thus, the slight decline in health status during the follow-up period may be underestimation if persons with health problems are less likely to respond. Further, it is possible that persons interpret the question on their health differently. This may lead to an increasing number of discordant twin pairs and thus overestimation of unshared environmental variance.

Our study sheds new light on the associations between early life and health status. Previous studies have suggested that low parental socio-economic position and poor living conditions in childhood are risk factors for several health problems in adulthood, even when adult socio-economic position is adjusted for (Elstad 2005; Mäkinen et al. 2006; Power et al. 2005). Our results partly challenge the interpretations of these findings since environmental factors common to co-twins, such as family background, had negligible effects on health in adolescence and early adulthood. Our results do not, however, exclude the possibility that childhood family environment or other environmental factors common to co-twins may affect later health, since the effect of these factors on health can occur in an interplay with genetic factors.

In conclusion, genetic factors play an important role in self-rated health, especially in adolescence. Additionally, environmental factors also contribute to self-rated health. A key challenge for further studies is to clarify the environmental factors that are likely to contribute to self-rated health independently or in interplay with genetic factors.

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