Perfect genetic correlation between number of offspring and grandoffspring in an industrialized human population

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Reproductive success is widely used as a measure of fitness. However, offspring quantity may not reflect the genetic contribution to subsequent generations if there is nonrandom variation in offspring quality. Offspring quality is likely to be an important component of human fitness, and tradeoffs between offspring quantity and quality have been reported. As such, studies using offspring quantity as a proxy for fitness may yield erroneous projections of evolutionary change, for example if there is little or no genetic variance in number of grandoffspring or if its genetic variance is to some extent independent of the genetic variance in number of offspring. To address this, we performed a quantitative genetic analysis on the reproductive history of 16,268 Swedish twins born between 1915 and 1929 and their offspring. There was significant sex limitation in the sources of familial variation, but the magnitudes of the genetic and environmental effects were the same in males and females. We found significant genetic variation in number of offspring and grandoffspring (heritability = 24% and 16%, respectively), and genetic variation in the two variables completely overlapped—i.e., there was a perfect genetic correlation between number of offspring and grandoffspring. Shared environment played a smaller but significant role in number of offspring and grandoffspring; again, there was a perfect shared environmental correlation between the two variables. These findings support the use of lifetime reproductive success as a proxy for fitness in populations like the one used here, but we caution against generalizing this conclusion to other kinds of human societies.

fertility | fecundity | children | grandchildren | selection

Measuring selection and projecting evolutionary change, including in contemporary human populations (1), relies on validly measuring fitness (i.e., the genetic contribution to future generations). Fitness is usually measured by a metric of reproductive success, i.e., offspring quantity (1, 2). However, offspring quantity may be a poor proxy for fitness when there is nonrandom variation in the reproductive quality of offspring (ref. 3; e.g., due to differences in offsprings' viability, attractiveness to mates, or intrasexual competitive ability). For example, a female might have few offspring but increase their reproductive quality (and the female's own fitness) by investing parental care and resources in the offspring, by choosing a mate who invests in the offspring (4), and/or by choosing a mate whose superior (5) or more compatible (6) genetic makeup improves the genetic quality of the offspring. A second female might have more offspring but fewer grandoffspring (and so lower fitness) if she and her mate(s) confer lesser material or genetic benefits to her offspring. The same of course applies to males.

Given humans' exceptionally slow life history (~15 y to sexual maturity) and high degree of biparental investment in offspring, the quality of those offspring is likely to be an important component of fitness in humans (7, 8), and extended parental investment improves quality of offspring in terms of their reproductive success (9). Research from preindustrial societies provides evidence for a tradeoff between offspring quantity and reproductive quality (e.g., refs. 2, 8, and 10–12), and there is evidence in postindustrial societies that offspring quantity is associated with lower parental investment in each offspring (13) and with detriments in offspring quality measures such as intelligence (14) and childhood growth (15) (see ref. 16 for a review of quantity–quality tradeoffs in humans). Such tradeoffs could mean that number of offspring might be a misleading indicator of longer-range (i.e., better) measures of fitness, e.g., number of grandoffspring.

Evolutionary change in a trait (i.e., the shift in population mean over generations) due to selection depends on the trait's genetic covariation with fitness (17–19). In this way (i.e., using the Robertson–Price identity), recent high-profile studies have projected evolutionary change in human traits (20, 21). However, because they used number of offspring to measure fitness, the projected magnitude or direction of evolutionary change could be wrong. For example, although previous research has revealed genetic variation (39% of the total variation) in number of offspring (22), there might be little or no genetic variation in number of grandoffspring, which would yield little or no long-term evolutionary change. Alternatively, if there is genetic variation in number of grandoffspring, it might not be captured by the genetic variation in number of offspring (e.g., because of the genetic variation in traits relating to maternal investment, mate choice, or mate retention), which would affect the magnitude or direction of the genetic covariation with the trait. However, it could be that the genetic variation in number of grandoffspring completely overlaps (i.e., $r_g = 1.0$) with the genetic variation in

Significance

Reproductive success (offspring quantity) is widely used as a measure of fitness (genetic contribution to future generations). Accurate predictions of the direction and magnitude of evolutionary change using this measure depend on the untested assumption that the genes influencing number of offspring are the same as those influencing number of grandoffspring. Using a population sample of identical and nonidentical Swedish twins and their descendants, we show that the genetic influences on number of offspring and grandoffspring are identical, supporting the use of reproductive success as a measure of fitness in comparable human populations.

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number of offspring, which would validate using number of offspring as a measure of fitness.

The classical twin design uses the greater genetic similarity of identical twins (100%) compared with nonidentical twins (50%) to partition traits' variance and covariance into genetic and environmental sources. Here we examine Swedish twins born between 1915 and 1929 ($n = 16{,}268$) and their number of offspring and grandoffspring born, which, for the vast majority of the sample, reflect lifetime reproductive fitness in both generations (*Methods*). We estimate the genetic variation in these variables and assess whether there are genetic influences on number of grandoffspring that are independent of the genetic influences on number of offspring.

Results

Preliminary Analyses. Table 1 shows means and variances for the sample. We first tested the assumption that identical and nonidentical twins are comparable except for their level of genetic similarity—inequality of means and variances of identical and nonidentical twins could suggest nonrandom sampling or sibling interaction effects that could bias the estimation of variance components (23). There were no significant mean or variance differences between identical and nonidentical twins for number of offspring or grandoffspring. As such, all means and variances were equated between identical and nonidentical twins in subsequent analyses. Women had more recorded offspring ($\chi^2_1 = 4.81$, $P = 0.03$) and grandoffspring ($\chi^2_1 = 3.69$, $P = 0.05$) than did men, although the differences were small—the differences were presumably due to the fact that 3.6% of the population did not have a recorded father (unknown paternity), whereas unknown maternity was virtually nil. The variance in number of offspring did not differ significantly between the sexes ($\chi^2_1 = 2.29$, $P = 0.13$), but women showed greater variance than men in number of grandoffspring ($\chi^2_1 = 6.22$, $P = 0.01$).

There were significant positive correlations of year of birth in both males and females for number of offspring and grandoffspring, so the year of birth was retained as a covariate in subsequent modeling.

Twin Pair Correlations. Twin pair correlations and cross-twin cross-trait correlations are shown in Table 2. All twin pair correlations were significantly greater than zero. Identical twin pair correlations were greater than the corresponding nonidentical twin pair correlations, suggesting genetic effects, which will be formally tested in the next section, *Genetic Analysis*. Opposite-sex twin pairs were significantly less similar than nonidentical same-sex pairs for both number of offspring ($\chi^2_1 = 5.48$, $P = 0.02$) and number of grandoffspring ($\chi^2_1 = 6.39$, $P = 0.01$), indicating an imperfect overlap in the source of familial (i.e., genetic or shared environmental) variation in males and females (i.e., sex limitation)—for example, different genes influencing the variables in males and females.

Genetic Analysis. To estimate the relative magnitudes of the genetic and environmental components of variance, we use standard quantitative genetic analysis, which determines the genetic (A), shared environmental (C), and residual (E) values that are most likely given the observed data. The most powerful method to estimate the relative magnitude of genetic and environmental influences on two correlated variables is in a bivariate analysis (rather than two univariate analyses), because the bivariate method takes advantage of the extra information in cross-twin cross-trait correlations. Most importantly, a bivariate design also allows us to analyze the overlap in genetic and environmental variation in the two variables.

Table 1. Means and variances of number of offspring born and number of grandoffspring born

	No. of offspring		No. of grandoffspring	
	Mean	Variance	Mean	Variance
Males ($n = 7578$)	1.76	2.16	3.28	11.56
Females ($n = 8690$)	1.90	2.25	3.56	12.18
Total ($n = 16268$)	1.84	2.21	3.43	11.92

The variance component estimates from a bivariate Cholesky decomposition are shown in Table 3. Note that we do not include opposite-sex twins in the variance component estimation because of the aforementioned significant sex limitation—we have too little information to tell whether it is the genetic effects and/or the shared environmental effects that are sex limited, and a model leaving both cross-sex genetic and cross-sex shared environmental correlations free to be estimated would be nonidentified. However, as a guide to the extent of sex limitation of the genetic effects, we ran a bivariate model including opposite-sex twins, assuming no sex limitation in shared environmental effects and leaving the cross-sex genetic correlation free to be estimated; this yielded a cross-sex genetic correlation of 0.34 (where a cross-sex genetic correlation of 1 would indicate the same genetic factors underlie the trait in males and females and 0 would indicate entirely different genetic underpinnings in each sex).

As can be seen in Table 3, male and female parameter estimates from the bivariate analysis (as represented in Fig. 1) were remarkably similar, and so we also present parameter estimates constrained to be equal in males and females—unless otherwise specified, from this point on we will refer to these male–female-equated estimates. One genetic factor ($A1$ in Fig. 1) had a modest but significant influence on both number of offspring and number of grandoffspring, accounting for 24% [coefficient of additive genetic variation (CV_A) = 39.58] and 16% (CV_A = 40.26) of the variance, respectively. The genetic influences on number of grandoffspring that are independent of genetic influences on number of offspring were estimated at zero (i.e., parameter a22 in Fig. 1 and Table 3), with the 95% confidence intervals suggesting the true value is likely to be very close to zero—the upper confidence interval was only 1% variance accounted for. The shared environment contributed less, but significantly, to both number of offspring and grandoffspring, accounting for 4% and 10% of variation, respectively. Again, shared environmental influences unique to number of grandoffspring were estimated at zero (parameter c22), also with narrow confidence intervals. The only influences unique to number of grandoffspring were residual factors, which could include any biological or environmental variables not shared between twins (e.g., random chance, idiosyncratic experiences, unique peer influences, stochastic biological effects), along with measurement error (e.g., inaccuracies in offspring data).

Genetic and environmental correlations are estimated in the model, but the same values can be derived from the parameter estimates in Table 3 using the formulas in *SI Appendix*, Box 1. For example, the genetic correlation between number of offspring and grandoffspring is given by $r_A = \frac{a11 \times a21}{\sqrt{a11^2} \times \sqrt{a21^2 + a22^2}} = \frac{\sqrt{0.24} \times \sqrt{0.16}}{\sqrt{0.24} \times \sqrt{0.16 + 0.00}} = 1.00$. The corresponding shared environmental correlation, r_C, was also equal to 1.0, indicating complete overlap in the genetic and shared environmental variation in number of offspring and grandoffspring. The corresponding residual correlation was substantially less than 1.00 ($r_E = 0.80$), which is why the phenotypic correlation was also imperfect ($r = 0.85$). The genetic, shared environmental, and residual correlations accounted for 22%, 8%, and 70% of the phenotypic correlation, respectively (see formulas in *SI Appendix*, Box 1). For more details on using the classical twin design to decompose variance between two variables,

Table 2. Twin pair correlations for number of offspring and number of grandoffspring

Zygosity	No. of offspring	No. of grandoffspring	Cross-twin cross-trait
Identical females	0.30 (0.25, 0.35)	0.27 (0.21, 0.32)	0.27 (0.23, 0.32)
Identical males	0.29 (0.23, 0.35)	0.22 (0.16, 0.28)	0.25 (0.20, 0.31)
Nonidentical females	0.15 (0.10, 0.19)	0.18 (0.14, 0.23)	0.16 (0.12, 0.20)
Nonidentical males	0.14 (0.09, 0.19)	0.17 (0.13, 0.22)	0.15 (0.10, 0.19)
Opposite sex	0.09 (0.05, 0.13)	0.12 (0.08, 0.15)	0.10 (0.07, 0.14) (MoFg) 0.10 (0.06, 0.14) (FoMg)

Numbers in parentheses are 95% confidence intervals. Cross-twin cross-trait correlations are the correlation between number of offspring of one twin and number of grandoffspring of the other twin. FoMg is the correlation between female offspring and male grandoffspring; MoFg is the correlation between male offspring and female grandoffspring.

see *SI Appendix*, *Using the Classical Twin Design to Decompose the Covariance Between Two Traits*.

Discussion

We found that both number of offspring and number of grandoffspring showed modest heritability (24% and 16%, respectively), but that there was no genetic variation in number of grandoffspring that was not accounted for by the genetic variation in number of offspring. Likewise, shared environment (e.g., parental influences, religious/cultural upbringing, rural/urban background, family socioeconomic conditions, etc.) contributed significantly to the variation in number of offspring and grandoffspring (4% and 10%, respectively), but there was no shared environmental influence that was unique to number of grandoffspring. The sources of familial effects on both variables partly differed between the sexes—this is especially noteworthy in the context that such sex limitation is very rarely detected in complex human traits (24). However, the magnitudes of the genetic and environmental effects on each trait were remarkably similar between males and females.

Our results have implications for the understanding of the relative importance of offspring quantity and quality to evolutionary change in developed human societies where the mean and variance in reproductive success are low and survival of offspring to reproductive age is high. The results suggest that in such populations, because genetic variation in reproductive fitness captures all of the genetic variation in longer-range fitness, factors that affect offspring reproductive quality without affecting offspring number will not effect an evolutionary response. Although there could be a very small amount of genetic variance unique to number of grandoffspring that we have failed to detect due to sampling error, it is certainly of vastly lower magnitude than that accounted for by genetic variation in offspring number.

This perfect genetic correlation of offspring quantity with second-generation fitness suggests that studies of humans in developed nations that have estimated evolutionary responses using lifetime reproductive success as a proxy for fitness (e.g., refs. 20, 21, and 25) are probably justified in doing so, as are future studies of this sort. Until now this has just been assumed, which had cast uncertainty on the conclusions of such studies. It probably matters little how lifetime reproductive success is measured—we counted all births, which fully allows for manifest variation in offspring quality, and more restrictive measures (e.g., only counting offspring that survive to sexual maturity) could only exhibit even stronger correlation with number of grandoffspring. However, the present findings also recommend caution regarding another aspect of the aforementioned studies. Byars et al. (21) and Stearns et al. (20), as well as studies in natural-fertility populations (e.g., refs. 26–28), estimated genetic variance in reproductive success using nontwin family relationships, which cannot properly disentangle genetic from shared environmental effects because they are confounded (i.e., genetic and environmental similarity are probably associated). We find that shared environmental effects, although relatively small, contribute significantly to familial similarity in number of offspring and more so in number of grandoffspring (accounting for more than a third of the familial variance). This recommends some caution when estimating genetic correlations with reproductive success from nontwin family data, but the fact that familial variation is mostly genetic also reinforces the point that estimating genetic correlations from nontwin family data is better than relying on

Table 3. Squared path coefficients from Fig. 1 for models of males and females and with the paths equated between sexes

Variance component	Parameter	Males	Females	Male and female parameters equated
Genetic factors (*A*)	$a11^2$	0.24 (0.13, 0.31)	0.24 (0.14, 0.32)	0.24 (0.17, 0.30)
	$a21^2$	0.16 (0.05, 0.26)	0.17 (0.07, 0.27)	0.16 (0.09, 0.24)
	$a22^2$	0.00 (0.00, 0.01)	0.00 (0.00, 0.02)	0.00 (0.00, 0.01)
Shared environment (*C*)	$c11^2$	0.03 (0.00, 0.11)	0.05 (0.00, 0.12)	0.04 (0.01, 0.09)
	$c21^2$	0.09 (0.02, 0.16)	0.10 (0.03, 0.18)	0.10 (0.04, 0.15)
	$c22^2$	0.00 (0.00, 0.02)	0.00 (0.00, 0.02)	0.00 (0.00, 0.02)
Residual (*E*)	$e11^2$	0.73 (0.68, 0.78)	0.71 (0.67, 0.76)	0.72 (0.68, 0.76)
	$e21^2$	0.49 (0.44, 0.54)	0.49 (0.44, 0.53)	0.49 (0.45, 0.52)
	$e22^2$	0.27 (0.26, 0.29)	0.24 (0.23, 0.26)	0.26 (0.25, 0.27)

Numbers in parentheses are 95% confidence intervals. Squared path coefficients represent the proportion of variance in the observed trait to which an arrow (Fig. 1) is pointing that is accounted for by the latent factor from which the arrow originates. *A*1 represents the genetic variation that influences both number of offspring and grandoffspring (via paths a11 and a21). *A*2 represents the genetic variation that influences number of grandoffspring (via path a22) but do not influence number of offspring. The same applies to the corresponding *C* and *E* factors and paths.

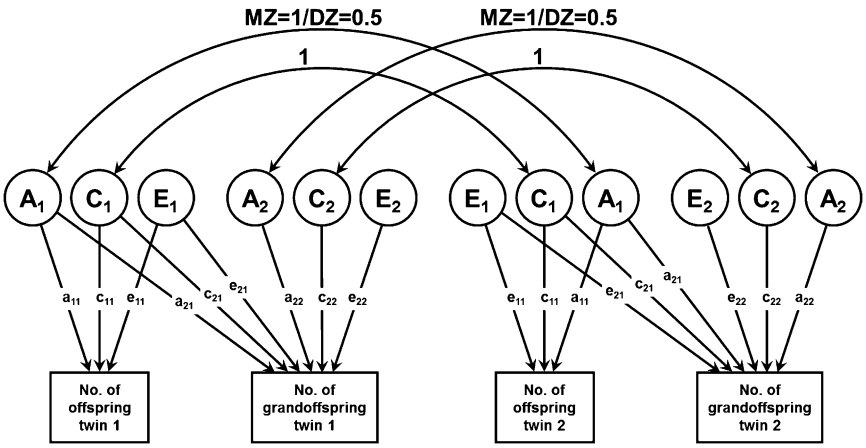

Fig. 1. Path diagram of a Cholesky decomposition. Circles represent latent factors: *A*, genetic; *C*: shared environmental; and *E*, residual. These latent factors influence the observed variables (rectangles) via the paths (arrows). The first latent genetic variable (*A*1) explains the genetic influences on number of offspring and the correlated genetic influences on number of grandoffspring. The second latent genetic variable (*A*2) is uncorrelated with *A*1 and explains the remaining heritability of number of grandoffspring. Corresponding latent structures apply to *C* and *E* latent variables. Parameter estimates are reported in Table 3.

phenotypic correlations as proxies for genetic correlations (19), as is often done (e.g., refs. 11, 25, and 29).

The present study should be replicated in other developed nations, but we see no obvious reasons to expect very different results. However, there are many reasons why the findings may not generalize to developing societies, and especially not to traditional, natural-fertility societies of the kind that characterized much of our evolutionary history. Scarce or unreliable resources, high mortality rates, and high birth rates in such societies probably mean that the quality of offspring varies more (e.g., more offspring die before reproductive age; less-equal distribution of resources, e.g., nutrition during development; more variation in serious illness during development; etc.) and reproductive consequences of these differences are probably greater, due to natural fertility and high birth rates. As such, it remains to be tested in preindustrial societies whether there is genetic variation unique to number of grandoffspring (and hence to what extent reproductive success is a good proxy for fitness).

The present study has several limitations that need to be taken into account. As well as the data truncation noted in *Methods* and the 3.6% of offspring with unknown paternity, there are likely to be additional instances of nonpaternity (i.e., biological father is someone other than who it is presumed to be, e.g., in cases of cuckoldry). This would contribute to error variance, because the offspring of the least-investing fathers (on average, those fathering extrapair offspring) could be linked to the wrong family (i.e., wrongly included or excluded from a twin's lineage). However, nonpaternity is uncommon (around 1–3%) in Western societies including Sweden (30) and so should not have greatly affected our results. Another caveat stems from limitations of the classical twin design, which precludes nonadditive genetic effects from being modeled along with shared environmental effects; nonadditive genetic effects may nonetheless be present and unaccounted for, in which case shared environmental variance would be underestimated (31). However, our estimate of genetic effects should provide a relatively robust estimate of the total genetic variance (additive plus nonadditive) (31).

Keeping in mind these caveats, our findings reveal a perfect genetic correlation between reproductive success and longer-range fitness in an industrialized human population, a finding of key importance when interpreting and designing studies aiming to estimate evolutionary change in human characters.

Methods

Participants. Participants were drawn from the Swedish Twin Registry, a population-based study of twins (32). This study was approved by the Regional Ethics Committee at Karolinska Institutet, Stockholm, Sweden. Informed consent was not required because an independent government agency (Statistics Sweden) merged and anonymized the data, and the code identifying the individuals was destroyed after merging. The twins' zygosity was determined by answering the question, "During childhood, were you and your twin partner as alike as 'two peas in a pod' or not more alike than siblings in general?", a method which has been shown to accurately determine zygosity in 95% of twin pairs (32). The question has been included in questionnaires sent out to all Swedish twins in different waves; two waves, in 1961 and 1963, cover the current twin population. In Sweden a personal identification number was introduced in 1947 for all individuals alive and living in Sweden, and onwards for all born in Sweden. By use of the personal identification number all Swedes are linked to their parents in the Multi Generation Register. The coverage is almost complete for Swedes born in 1933 and later, who were alive and living in Sweden in 1947, and practically complete for all born in 1947 and onwards (see ref. 33 for more details). Thus, we were able to accurately register all births of twins alive in Sweden by 1961–1963, and all of their offspring born in 1933 and onwards. We limited the twin sample to those born between January 1, 1915 and December 31, 1929. This criterion aimed to optimize the tradeoff between achieving the largest sample size (necessary for obtaining precise parameter estimates) and minimizing truncation due to twins reproducing earlier than our records of offspring start (1933) or twins' offspring reproducing later than our records of grandoffspring end (2009). As it is, truncation occurs such that the earliest-born twins' births before age 18 y are not included in our data, and the latest-born twins' grandoffspring are not included if they had not yet been born 80 y after the twins' own birth. Using the birth rates observed at various ages in cohorts for which we have untruncated data, we estimate that ∼0.12% of individuals in our sample are missing one or more offspring because of truncation and ∼1.3% of individuals are missing one or more grandoffspring (see *SI Appendix, Assessing the Impact of Truncation* for details).

Statistical Analysis. Phenotypic (observed) variation in a trait can in principle be partitioned into genetic and environmental (i.e., nongenetic) sources. In practice this is often done by testing the phenotypic similarity of individuals in families or pedigrees with known genetic relatedness. However, genetic and environmental similarity are likely to be correlated, and this confound makes it difficult to distinguish genetic and shared (family) environmental sources of variance using standard family data. Identical and nonidentical twins provide a natural experiment that allows genetic and shared environmental influences to be disentangled, because both identical and nonidentical pairs share the same family environment (e.g., home environment and socioeconomic status) whereas, genetically, identical twin pairs are twice as similar (100%) as nonidentical twin pairs (50% on average). As such, genetic sources of variance, *A*, predict greater trait similarity in identical pairs than in nonidentical pairs; if additive genetic were the only source of variance in a trait we would

expect a twin correlation of 1 for identical pairs and 0.5 for nonidentical pairs. In contrast, shared environmental sources of variance, *C*, predict equal similarity of identical and nonidentical twin pairs; if shared environment were the only source of variance in a trait, we would expect a twin correlation of 1 for both identical and nonidentical pairs. Residual variance, *E* (e.g., due to idiosyncratic experiences, stochastic biological effects, measurement error), is uncorrelated in both identical and nonidentical pairs. In reality, observed identical and nonidentical twin correlations generally reflect a combination of these sources of variance, and structural equation modeling determines the combination that best matches the observed data.

A bivariate twin design enables the phenotypic variation of two variables, and covariation between them, to be partitioned into *A*, *C*, and *E* sources; that is, we can estimate the extent to which the observed correlations between variables is due to overlap in genetic influences (genetic correlation, r_g or r_A), shared environmental influences (shared environmental correlation, r_C), or residual factors (residual correlation, r_E). Fig. 1 shows a path diagram representing the resemblance between identical and nonidentical twins in a bivariate design in which twin pairs (twin 1 and twin 2) are each measured on two variables, number of offspring and number of grandoffspring. The first latent genetic variable (*A*1) explains the genetic influences on number of offspring and the correlated genetic influences on number of grandoffspring. The second latent genetic variable (*A*2) is uncorrelated with *A*1 and explains the remaining heritability of number of grandoffspring. Corresponding latent structures apply to *C* and *E* latent variables.

This path diagram can be translated directly into structural equations representing the expected covariances of identical and nonidentical twins (*SI Appendix*, Table S2). In the freely available matrix algebra program, Mx (34), full information maximum-likelihood modeling is used to determine the parameters of these structural equations that best fit the observed data. We can test whether a given parameter(s) is significantly different from zero by fixing the parameter(s) to zero and testing the change in goodness of fit of a model [distributed as χ^2] against the change in degrees of freedom (reflected by the difference in the number of parameters estimated); likewise, we can test if two parameters significantly differ from each other by constraining them to be equal and similarly testing the change in model fit.

This methodology is standard in human quantitative genetics, and further details can be found in *SI Appendix* and elsewhere (35, 36). As per standard procedure, year of birth is modeled as a fixed effect on the mean so as not to spuriously inflate twin pair correlations (since members of a twin pair have the same year of birth). To check the robustness of the results across different modeling software, we replicated the main analysis in M*plus* (37), yielding equivalent results.

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