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DOES EDUCATION MEDIATE THE RELATIONSHIP BETWEEN IQ AND AGE OF FIRST BIRTH? A BEHAVIOURAL GENETIC ANALYSIS

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Summary. This study presents a multivariate behavioural genetic analysis of the relationship between education, intelligence and age of first birth. Analyses investigated the mediational role of education in explaining the relationship between intelligence and age of first birth at both the phenotypic and behavioural genetic level. The data come from the National Longitudinal Survey of Youth (NLSY), a nationally representative survey that included genetically informative full- and half-sibling pairs ($n = 1423$ pairs). Respondents were aged 14 to 22 when contacted in 1979. Heritability estimates were 0·32, 0·50 and 0·06 for IQ, education and age of first birth, respectively. Shared environment estimates were 0·35, 0·23 and 0·20 respectively. Common genetic and shared environmental factors were substantial in explaining the relationship between intelligence and education, and also education and age of first birth. Education partially mediated the relationship between intelligence and age of first birth only in the phenotypic analyses. After considering the genetic and shared environmental factors that influence all three variables, evidence for mediation was less convincing. This pattern of results suggests that the apparent mediational role of education at the phenotypic level is in fact the result of underlying genetic and shared environmental influences that affect education, IQ and age of first birth in common.

Introduction

The age of first birth varies enormously by historical period and culture. In the United States, parents of the 'baby boom' generation after World War II had a higher birth rate, lower age of first birth and larger completed family sizes than did more recent cohorts of Americans. Such generational influences clearly point to environmental influences on fertility behaviours. Increased educational levels are often

thought to be one environmental pathway to later age of first birth, particularly among women (Heaton & Forste, 1998; Rindfuss & Hirschman, 1984; Wilkie, 1981). The current study investigated the relationship between intelligence, education and age of first birth using both phenotypic and behavioural genetic analyses. The phenotypic analyses investigated whether education mediated the relationship between intelligence and age of first birth. The behavioural genetic analyses assessed the extent to which covariation between intelligence, education and age of first birth can be ascribed to common genetic and environmental influences. The mediational role of education within the behavioural genetic model was also examined.

Although cohort trends illustrate general patterns of life history reproductive traits, wide variability exists within a population. Researchers believed for some time that reproductive traits were not under genetic influence; rather, environmental influences were thought to play the primary role in explaining variability in reproductive traits. This belief was mainly derived from R. A. Fisher's (1930) Fundamental Theorem of Natural Selection (FTNS), in which Fisher set forth the idea that additive genetic variance will disappear over time for traits and behaviours with large influence on reproductive success. Results from some studies supported this interpretation of Fisher's theorem, as early heritability estimates for completed fertility tended to be low and non-significant (Imaizumi & Nei, 1970; Mealey & Segal, 1993). Many specific behaviours leading to reproduction, however, have been found to reflect genetic influences.

Significant heritability has been associated with the timing of several fertility-related behaviours. Pubertal timing and age at menarche both have demonstrated moderate heritability (0·40 to 0·44) and negligible effects of shared environment (Rowe, 2000). Pickles *et al.* (1998) reported that age at menarche and breast development both show strong genetic effects and no shared environmental effects. Mealey & Segal (1993) reported evidence of genetic influences on age at marriage and age at first date.

Results from recent large-scale studies suggest that heritability estimates can vary as a function of birth cohort and gender. Dunne *et al.* (1997) reported that heritability for age at first intercourse among Australian twins from a young cohort was 0·72 for males and 0·49 for females. Shared environmental effects were found only for females from the younger cohort. Among an older cohort, however, genetic factors were weaker. Heritability was estimated to be 0·32 among females and genetic effects did not account for any of the variance among males. Among this cohort, shared environment accounted for a larger proportion of the variance in age at first intercourse, with estimates of 0·25 for females and 0·42 for males.

In an analysis of birth cohorts of Danish twins, Kohler, Rodgers & Christensen (1999) discovered that the heritability of fertility was zero per cent in Denmark prior to a demographic transition in the late 1800s that sharply reduced population fertility. Heritability was 0·40 to 0·50 thereafter for female cohorts. Shared environmental effects were higher for the pretransitional cohorts and for those cohorts experiencing crises during their early reproductive years. Shared environmental effects were present for most male cohorts, but not for the most recent female cohorts. For male cohorts after 1905, fertility was also moderately heritable. The changing fortunes of genetic influences on heritability were interpreted by Kohler *et al.* in terms of social choices

that people make about having children. They argued that heritability was highest when people, especially women, had greater choice over reproductive behaviour. Shared environmental effects, in contrast, were strongest when choices for demographic and other behaviours may have become more restrictive because of wars and other crises.

Overall, past research suggests that several behaviours related to fertility show clear genetic influences. Furthermore, studies that look at age differences in genetic and environmental influences show a pattern of greater heritability among younger cohorts. While diminished within more recent cohorts, shared environmental factors also contribute to explaining variance in the timing of fertility-related traits and behaviours.

In previous work by Rodgers and colleagues, the heritability of life history traits has been examined using the National Longitudinal Survey of Youth (NLSY) (Rodgers & Doughty, 2000; Rodgers, Rowe & Buster, 1999). The NLSY is a nationally representative survey of adolescents who were 14–22 years old in 1979. Respondents were contacted annually up to 1992, and have been interviewed biennially subsequently. In one study (Rodgers *et al.*, 1999), the age of first intercourse, as reported retrospectively by the NLSY subjects, was found to be heritable, with small to no shared environmental influences. Analysis of selected samples from upper and lower tails of age of onset distributions suggested that genetic influences are important in accounting for both early and late non-virginity. Genetic effects were weak for Blacks, but the smaller sample size of this analysis recommends interpretive caution. In a later study, Rodgers & Doughty (2000) used the same data set to estimate genetic and environmental influences on fertility expectations and completed family size. Both had a heritable component and no shared environmental one.

In this study, just one life history trait is focused on: age of first birth. The timing of first birth shows clear intergenerational continuity within families (Hardy *et al.*, 1998). Children of teenage mothers are more likely to be adolescent parents themselves (Manlove, 1997; Thornberry, Smith & Howard, 1997), whereas children of women who delay parenthood are more likely to also delay childbearing (Hardy *et al.*, 1998). In Kohler *et al.* (1999), the age of first attempt to get pregnant was the most heritable aspect of fertility. Age of first birth is clearly related to age of first attempt to get pregnant, making the former a good marker variable for tracking the extent of genetic influence. Age of first birth is also a good predictor of completed fertility and thus of population growth or decline (Freedman, Thornton & Wallisch, 1981).

This study goes beyond previous studies in that it examines the relationships among intelligence, years of education and age of first birth. IQ is a highly stable individual trait (e.g. there is a 0·63 test–retest IQ correlation on the same test, given twice, once at 11 years of age and then later at 77 years of age; Deary *et al.*, 2000). Phenotypically, IQ and years of education correlate fairly strongly. Mean levels of both IQ and years of education also show historical change. Flynn (1987) presented data showing that IQ scores have been rising linearly in many Western countries during this century. The existence of some IQ gains, especially in non-verbal tests, is generally accepted. Whether this is a cohort or period phenomenon, whether it holds

in all subpopulations, and its causes, are all unspecified (Rodgers, 1999). Cohort and period changes in years of education, however, are indisputable.

Fertility rates also exhibit cohort and period changes. One theory of the decline in female fertility in many places in the world is the increased access of women to education. In developing countries, Heaton & Forste (1998) found education to be significantly related to marriage rates, contraceptive use and fertility rates. In a study of American women, Wilkie (1981) reported that the proportion of women having first births at older ages increased directly with schooling. In a study of four Asian countries experiencing overall demographic declines in fertility, Rindfuss & Hirschman (1984) found education to be a significant predictor of age of first birth, with less educated women having their first child at earlier ages. Some of the mechanisms by which education has been thought to reduce fertility include reduction in child mortality so that women are satisfied with fewer births and increased autonomy for women (Jeffery & Basu, 1996).

In this study, the hypothesis that years of education mediates the effect of IQ on age of first birth at the phenotypic level is examined. A direct mediational view is that IQ influences years of schooling, which in turn creates opportunity costs that result in a delay of childbearing or fathering. If this hypothesis is true, then the correlation between IQ (measured in most cases before childbirth) and age of first birth should be weaker than the one between IQ and education. Retherford & Sewell (1989) found this pattern in a US sample using completed family size as a fertility outcome. They concluded that the effects of IQ on fertility are almost entirely mediated by education.

The next analyses examine genetic and shared environmental influences. A biometric genetic model (Neale & Cardon, 1992) is used to look beyond the phenotypic associations among these three traits to the genetic, shared environmental and non-shared environmental latent variables that may determine them. The biometric model assumes that the observed correlations among IQ, education and age of first birth are brought about by other influences. One possibility is that the genes that influence IQ also influence education and age of first birth. This model implies that the variation in all three outcomes is under genetic control. Psychologically, greater IQ might lead to greater anticipation of the future and greater desire to inhibit fertility, at least when conditions are perceived as being unfavourable to large families. In contrast, shared environmental influences might play the greater role in the covariation among these traits. A common shared influence would be one that affected IQ, years of school attendance and age of first birth simultaneously. Such an influence is one that varies within cohorts spanning only about 8 years (if a cohort effect), or one that varies among neighbourhoods or individual families. The structure of a particular model fit can help guide speculations about the causative influences represented by a latent variable.

Methods

The NLSY sample

The present study utilizes data from the National Longitudinal Survey of Youth (NLSY). In 1979, the NLSY began as a household probability sample of all

14–21-year-old youths in selected households, with $n = 11{,}406$ civilian respondents. Because of this design, many kinship links exist in the NLSY files, including twins, full-siblings, half-siblings, cousins and adoptive siblings. The original sample was followed yearly from 1979 to 1992 and at biennial intervals thereafter. The present study analyses individuals interviewed during 1996, the most recent survey year available. The retention rate for the NLSY surveys has remained close to 90% throughout the years; the overall retention rate for 1996 is 86·7%.

Full- and half-siblings in the NLSY were identified using a computer algorithm that identified 3890 kinship pairs for classification. A conservative linking procedure that most accurately placed siblings into full, mixed full and half, and half-sibling groups classified 60% of the original pairs. Rodgers (1996) and Rodgers *et al.* (1999) provide more details on the linking algorithm. A brief summary is given here.

In 1988 (when they were 23–30 years old), each respondent created a retrospective time line from age 0 to age 18 of whether they lived with their biological mother and/or father in each year. The critical target year was the year in which the sample was drawn, 1979, the only year in which it is known for certain that a given pair of respondents was living together in the same household. By taking the information respondents had retrospectively provided for their age in 1979, an ordered quadruple was constructed as follows: (whether sibling #1 lived with genetic mother in 1979, whether sibling #1 lived with genetic father in 1979, whether sibling #2 lived with genetic mother in 1979, whether sibling #2 lived with genetic father in 1979). Thus a (1,1,1,1) response pattern would indicate two siblings, both of whom were living in the same household, and both of whom were living with their biological mother and father. This response pattern, it was reasoned, must unambiguously be full-siblings. Similarly, a response pattern of (1,0,1,1) indicates two siblings living together in 1979, both of whom were living with their genetic mother, but only one of whom was living with their genetic father. Any response pattern with three '1's and one '0' was classified as half-siblings. Some response patterns left unresolved whether a sibling pair was comprised of full- or half-siblings. For example, a pattern of (0,1,0,1) or (1,0,1,0) shows the siblings shared at least one biological parent, and possibly two. Other ambiguous patterns were classified on the basis of the physical distance from the father's home to the household. When pairs clearly shared only one biological parent, they were classified as half-siblings ($R = 0{\cdot}25$). When pairs shared at least one biological parent but possibly two, they were assigned a coefficient of genetic relatedness (R) of 0·375, a value midway between that of full- and half-siblings (0·5 and 0·25, respectively). Although the actual genetic relatedness of the mixed group is thus unknown, past analyses that have included this group (Buster & Rodgers, 2000; Rodgers *et al.*, 1999) generated results matching those found in studies with more direct measures of kinship level. This convergence of results provides support for the current coding system.

In 1996, a total of 2306 full- or half-sibling pairs were interviewed. The Rodgers *et al.* (1999) algorithm identified 47 pairs as cousins ($R = 0{\cdot}125$), 20 pairs as twins of unknown zygosity ($R = 0{\cdot}75$), 30 pairs as half-siblings ($R = 0{\cdot}25$), 229 pairs as mixed full- and half-siblings ($R = 0{\cdot}375$) and 1410 pairs as full-siblings ($R = 0{\cdot}5$). The cousin group was small, and it also was more likely to violate the assumption that siblings were raised by one family and so were exposed to similar environmental influences.

The small number of twins also prohibited model fitting. These two groups were thus excluded from further analyses. The small size of half-sibling group proscribed inclusion of these pairs as a separate group ($n = 30$). These pairs were therefore added to the mixed-sibling group.

Measures

Armed Services Vocational Aptitude Battery (ASVAB). Participants' ASVAB scores were obtained in 1980, when respondents were 15–23 years old. Within the entire NLSY sample, an IQ variable was created by first standardizing scores on four subtests: word knowledge, paragraph completion, arithmetic reasoning and mathematical knowledge. The standardized scores were then summed to create an overall IQ score.

Both age and ethnicity were significantly related to IQ within the NLSY sample. To control for these two variables, IQ scores were regressed onto respondents' age in 1980 and two dummy variables: one coding African American youths vs White and Hispanic youths, the other representing Hispanic youths vs African American and White youths. Age and ethnicity explained a substantial 25% of the variance in IQ scores ($F_{(3,\ 10{,}925)} = 1210{\cdot}80$, $p < 0{\cdot}001$), the majority of which was contributed by the two dummy variables for ethnic group. The residualized values from this regression equation, standardized into an IQ metric with a mean of 100 and standard deviation of 15, were used in this study.

Years of education. Information on respondents' highest grade completed was drawn from the 1996 survey. Age was not significantly related to level of education ($R = 0{\cdot}00$, $p = 0{\cdot}68$). ANOVA tests revealed significant ethnic differences in years of education: $F_{(2,\ 8199)} = 192{\cdot}63$, $p < 0{\cdot}001$. Among Hispanic respondents, the mean level of education was approximately a high school education ($M = 12{\cdot}2$, SD = 2·1), whereas African American respondents ($M = 12{\cdot}7$, SD = 2·1) and White respondents ($M = 13{\cdot}5$, SD = 2·5) reported higher levels of education. Post-hoc Tukey tests revealed all three comparisons of any two group means to be significantly different. Thus, within the entire sample, education was regressed on the two dummy variables described above representing ethnicity, and the residualized values are used in subsequent analyses.

Age of first birth. Age of first birth was assessed during the 1996 survey. Figure 1 shows the distribution of age of first birth for the sample. There was a slight mean sex difference in the age of first birth ($M_M = 24$; $M_F = 23$). Although this difference was significant, gender only explained 4% of the variance in age of first birth among the entire sample. Thus the main effect of gender was not removed from age of first birth.

Childless individuals, who represent 21·6% of the sample, are not included in the analyses. The decision to exclude them was made for several reasons. First, as shown by Fig. 1, the distribution of age of first birth was nearly normal in shape when the childless individuals were excluded. Thus, by just using the individuals with a birth, there are nearly ideal distributional characteristics for the birth variable for maximum likelihood model fitting. Secondly, those without a first birth are censored because

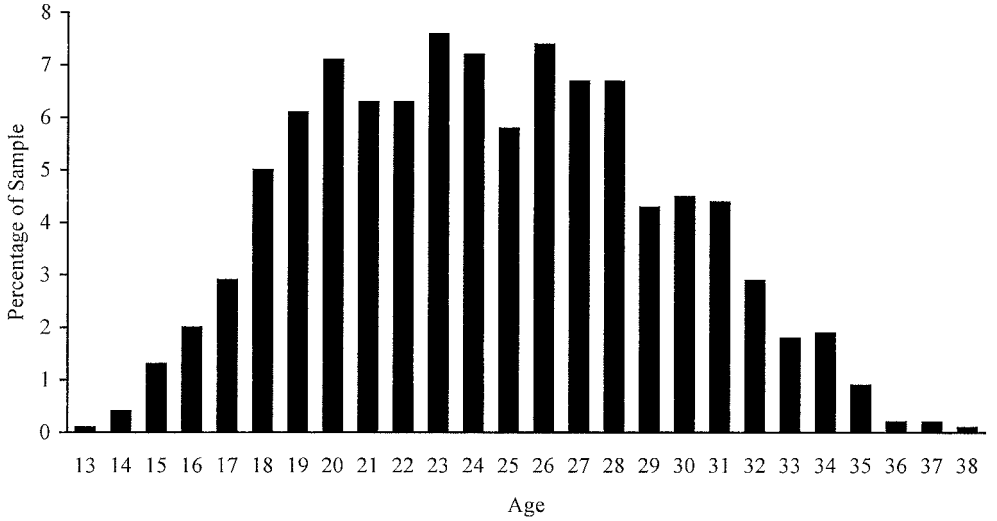

Fig. 1. Distribution of age of first birth.

some individuals may still have a first birth. This effect is not likely to be a strong one, however. Within the entire NLSY sample, only 3% of those aged 39 had their first birth at age 34 or higher. Among the paired sample, 52% of the men and 54% of the women are aged 34 or higher. Thus it seems likely that most of the individuals in this study who will eventually have children have already had their first birth. Finally, some of the currently childless respondents will never give birth. Although complex statistical methods exist for handling censored data, these censored cases present in the sample are probably not a strong bias against or towards finding genetic effects, and the most analytically parsimonious approach is probably to exclude them.

ANOVA tests again indicated significant ethnic differences in age of first birth ($F_{(2,\ 6155)} = 275{\cdot}62$, $p < 0{\cdot}001$). African American respondents reported the youngest average age of first birth ($M = 21{\cdot}8$, SD = 4·8). Hispanics reported a higher average age of first birth ($M = 22{\cdot}8$, SD = 4·6), whereas White respondents reported the oldest age ($M = 25{\cdot}0$, SD = 4·8). Post-hoc Tukey tests revealed all three mean-level differences are significant. By regressing age of first birth on the two dummy variables representing ethnicity, these significant ethnic differences were controlled for within the entire sample. Subsequent analyses use the residualized values from the regression equation.

The final sample included 2846 individuals, 49·5% male and 50·5% female. In 1996, their ages ranged from 31 to 39 ($M = 35{\cdot}0$, SD = 2·2). They had completed, on average, a year more than a high school education ($M = 13{\cdot}0$, SD = 2·4). The education distribution is such that 11·0% of the respondents reported having less than a high school education, 46·9% had a high school education, 22·9% had some college or advanced training, and 19·2% had completed college or had some advanced training. Their mean IQ was 99·9, with a standard deviation of 15·1. The age of first birth ranged from 13 to 38 ($M = 24{\cdot}2$, SD = 4·8).

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Table 1. Model fitting results from phenotypic analyses

Model	χ^2	df	RMSEA	AIC	*p*	Change in χ^2	*p*
Model 1	0·00	0	0·000	0·00	1·00	—	—
Model 2	22·25	1	0·086	22·52	0·000	22·25	0·000

Results

Prior to model fitting, gender differences in sibling similarity were investigated among all three variables (IQ, education and age of first birth). Greater similarity among male or female pairs could be evidence that the underlying genetic and environmental influences differ between the sexes. For example, different genes could influence age of first birth in the two sexes. Environmental influences may also differ by gender, as would be the case if female reproduction was under greater social constraint than male reproduction. Gender differences in the strength of genetic or environmental influences would lead to a difference in the level of sibling resemblance. To look for gender differences in sibling similarity, regression analyses were used predicting sibling_1's values on each variable from sibling_2's value, gender, and a gender × sibling_2 value interaction term. Results from an analysis conducted solely with same-sex pairs did not show evidence of a gender interaction, suggesting that brother and sister pairs were equally similar on these variables. Whether same-sex sibling pairs were more or less similar than mixed-sex sibling pairs was then investigated. Again, regression results did not indicate the presence of an interaction between sibling type and similarity. Thus, both same-sex and mixed-sex pairs were combined in analyses.

The next step in the analysis was to investigate whether education mediated the relationship between IQ and age of first birth at the phenotypic level. A path model in which IQ directly influences age of first birth (path C) and indirectly influences age of first birth through education (paths A and B) was fitted to data from the individuals in the sibling pairs using Mx software. The ordering of the research variables had both conceptual and methodological grounds. Previous researchers have argued that increased education delays age of first birth. Empirical support for this argument suggested that education should precede age of first birth. In the current study, ordering was additionally mandated by timing considerations. The IQ test was administered in 1980, when the average age of the sample was about 18 years and about half were under age 18. Thus, the majority had no children and had not completed their education. Although there are clearly cases that violate this causal ordering, the majority fit an ordering of: IQ test, years of completed education, birth of first child. Thus, the ordering of variables was restricted to that which best matched their temporal ordering.

Table 1 provides the fit indices from path model fitting. In Model 1, all path coefficients were estimated. Because the number of estimated parameters equals the number of observations, the model must provide a perfect fit ($\chi^2_{(0)} = 0{\cdot}00$, $p = 1{\cdot}00$). To test mediation, the path from IQ to age of first birth (C) was set to zero in Model 2. A good fit for Model 2 would provide evidence that education fully mediated the

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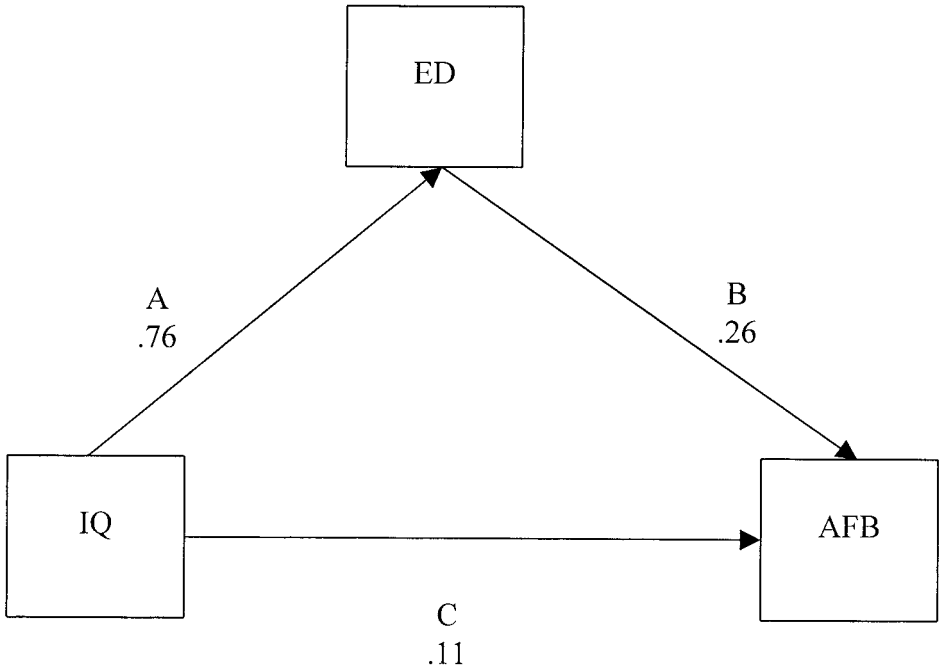

Fig. 2. Illustration of phenotypic mediation model with standardized path estimates.

relationship between IQ and age of first birth. However, setting the path from IQ to age of first birth to zero significantly worsened the model fit (change in $\chi^2_{(1)}=22{\cdot}25$, $p<0{\cdot}001$).

Figure 2 illustrates the phenotypic mediation model and provides the standardized path coefficients from Model 1. The predicted correlation between IQ and age of first birth can be calculated from the standardized path coefficients (AB+C) to be 0·31. While 35% of the relationship between IQ and age of first birth is due to direct effects, the remaining 65% is mediated through education. Overall, the results suggest that although education partially mediates the majority of the relationship between IQ and age of first birth, intelligence also exerts an important direct influence.

As a first step in beginning the behavioural genetic analysis of the relationship among IQ, education and age of first birth, sibling resemblance for these variables was examined. Table 2 presents the correlation and covariance matrices by sibling category. Sibling correlations for almost all of the variables are higher in the full-sibling category than in the mixed category, providing evidence for some genetic influence on all three variables at the univariate level. Looking at cross-trait, cross-sibling correlations (e.g. $\text{Education}_{\text{sib1}} \times \text{Age of first birth}_{\text{sib2}}$) it can be seen that pairs in the full-sibling group have slightly higher cross-trait resemblance for most comparisons than do those in the mixed-sibling group. This provides some evidence of genetic influences at the multivariate level. However, the correlations are not substantially larger among the full-siblings, which suggests that shared environmental influences are also important.

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Table 2. Covariances and correlations between sibling IQ, education (ED) and age of first birth (AFB)

	IQ_1	ED_1	AFB_1	IQ_2	ED_2	AFB_2
Mixed full- and half-siblings (r_g=0·375)						
IQ_{sib1}	166·33	0·53	0·00	0·39	0·27	0·26
ED_{sib1}	12·55	3·42	0·15	0·27	0·35	0·18
AFB_{sib1}	11·34	1·25	19·62	0·05	0·05	0·19
IQ_{sib2}	66·56	6·45	2·74	168·53	0·60	0·23
ED_{sib2}	8·31	1·54	0·55	18·49	5·64	0·19
AFB_{sib2}	16·02	1·55	4·07	13·98	2·14	22·59
Full-siblings (r_g=0·5)						
IQ_{sib1}	215·31	0·62	0·28	0·52	0·37	0·21
ED_{sib1}	21·58	5·69	0·35	0·47	0·51	0·28
AFB_{sib1}	18·37	3·74	20·62	0·22	0·27	0·25
IQ_{sib2}	105·10	15·59	13·88	191·48	0·59	0·28
ED_{sib2}	11·54	2·57	2·60	17·31	4·51	0·39
AFB_{sib2}	13·21	2·85	4·79	16·60	3·53	18·31

Note: Covariance matrix, diagonal and below; correlations above diagonal; n=127 pairs in mixed category, n=686 pairs in full-sibling category (sample size decreased due to listwise deletion of missing data).

Figure 3 shows results from fitting a full biometric model (Neale & Cardon, 1992). This biometric model includes all possible genetic and environmental influences, both those that are common to siblings and those that are unique. Common factors exert an influence on all three variables: IQ, education and age of first birth. The common genetic factor (C_G) represents genetic influences in common to all three variables; the common shared environmental factor (C_{SE}) exerts an environmental influence shared by all variables and equally by siblings; finally, the common non-shared environmental factor (C_{NSE}) also affects all variables, but it is uncorrelated across siblings. Three kinds of specific factors exist in the model, where each specific factor affects only one manifest variable. The terminology of calling these specific latent variables 'factors' is kept, despite their influencing just one variable. The specific genetic factor (S_G) and specific shared environment factor (S_{SE}) are correlated between siblings. The specific non-shared environmental factor (S_{NSE}) is not correlated between siblings; it also represents measurement error in the model. The correlation between siblings of both kinds of genetic factors depends on the coefficient of genetic relatedness, which in full-siblings is fixed to 0·50 and in half-siblings is fixed at 0·375; the latter also makes the assumption that this group contains about an equal number of full- (R=0·50) and half-siblings (R=0·25). Shared environmental factors, both common and specific, are assumed to correlate 1·0 between siblings. Thus, they represent the total cumulative effect of environmental influences common to siblings. Environmental exposures that are not completely identical across siblings may make

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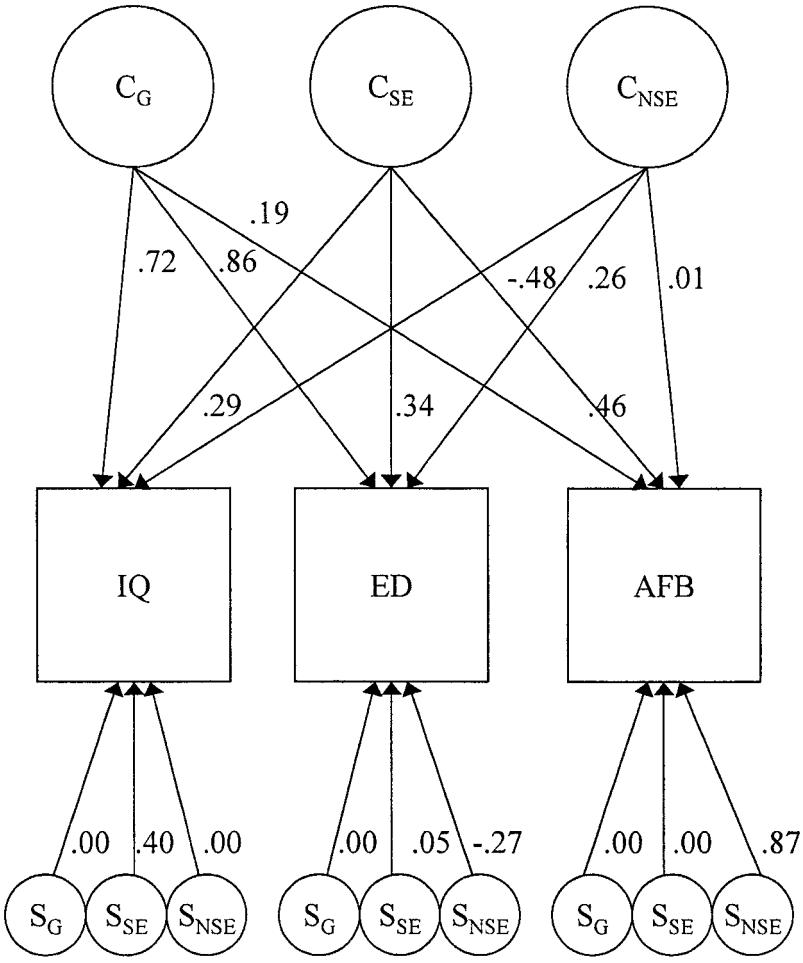

Fig. 3. Illustration of full biometric model with standardized path estimates.

a contribution to this component of sibling resemblance, even though they also contribute to non-shared environmental effects.

The chi-square value was significant, which may indicate a poor fit ($\chi^2_{(24)}=55{\cdot}31$, $p<0{\cdot}001$). However, chi-square values are sensitive to sample size (Bollen, 1989). A better measure of fit, the RMSEA, showed an acceptable fit (RMSEA = 0·071, which fell within the range of 0·05 and 0·08, indicating a reasonable fit; Browne & Cudeck, 1993). The model fit may have been adversely affected by a difference in the variance of IQ between full-siblings and the mixed-sibling group. The variance of IQ was substantially larger among full-siblings, a difference which most likely resulted by chance through sampling variation. A number of submodels were then investigated.

The fit results for the full model and several submodels are shown in Table 3. As shown in the second row of Table 3, dropping the common non-shared environment factor did not significantly change the chi-square values. The third and fourth rows

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Table 3. Model fitting results from behaviour genetic analyses

Model	χ^2	df	RMSEA	AIC	*p*	Change in χ^2	df	*p*
Full model	55·31	24	0·071	7·31	0·000	—	—	—
No common non-shared environment*	55·79	27	0·065	1·79	0·001	0·48	3	0·922
No common genetic environment	69·20	27	0·073	15·20	0·000	13·89	3	0·003
No common shared environment	72·38	27	0·074	18·38	0·000	17·07	3	0·001
Mediation model	55·58	25	0·069	5·58	0·000	0·21**	2	0·900

*The model with no common non-shared environment was chosen as the preferred model.
**Change in χ^2 values for mediation model calculated in comparison with the model with no common non-shared environment factor.

of Table 3 show that dropping either the common genetic factor or the common shared environmental factor did significantly worsen model fit. The preferred model was therefore chosen as one in which the common non-shared environmental factor was dropped.

After dropping the common non-shared environmental factors, some of the specific path estimates remained quite low. Although it is possible to drop these specific paths, it was feared that selectively dropping only certain specific paths would capitalize on chance. Dropping all specific genetic and specific shared environmental paths, however, significantly worsened the model ($\Delta\chi^2_{(6)} = 20{\cdot}46$, $p < 0{\cdot}01$). Thus these paths were retained. The solid lines displayed in Fig. 4 illustrate this reduced biometric model.

To test the mediational hypothesis, a path was added from IQ to education and a path from education to age of first birth (see dotted lines in Fig. 4) to the reduced biometric model. Addition of these paths did not significantly improve the model fit because the reduction of the chi-square was small (change in $\chi^2_{(2)} = 0{\cdot}21$, $p = 0{\cdot}900$). Thus, it is concluded that the mediation effects were carried by the common shared environmental (C_{SE}) and common genetic factors (C_G) already in the model, and use the reduced biometric model to apportion phenotypic variance.

The standardized path coefficients provided in Fig. 4 can be used to estimate the contribution of the common factors to the intercorrelations among the manifest variables and also to estimate total heritability and shared environment effects. The heritability for each variable is the genetic variance divided by the total variance. Because the path estimates are standardized, the total variance for each variable is one. The heritability for each is thus simply the sum of the genetic variance. Similarly, the shared environmental component of variation is the sum of both common and specific shared environment variances. The heritabilities were 0·32, 0·50 and 0·06 for IQ, education and age of first birth, respectively. The shared environment effects were

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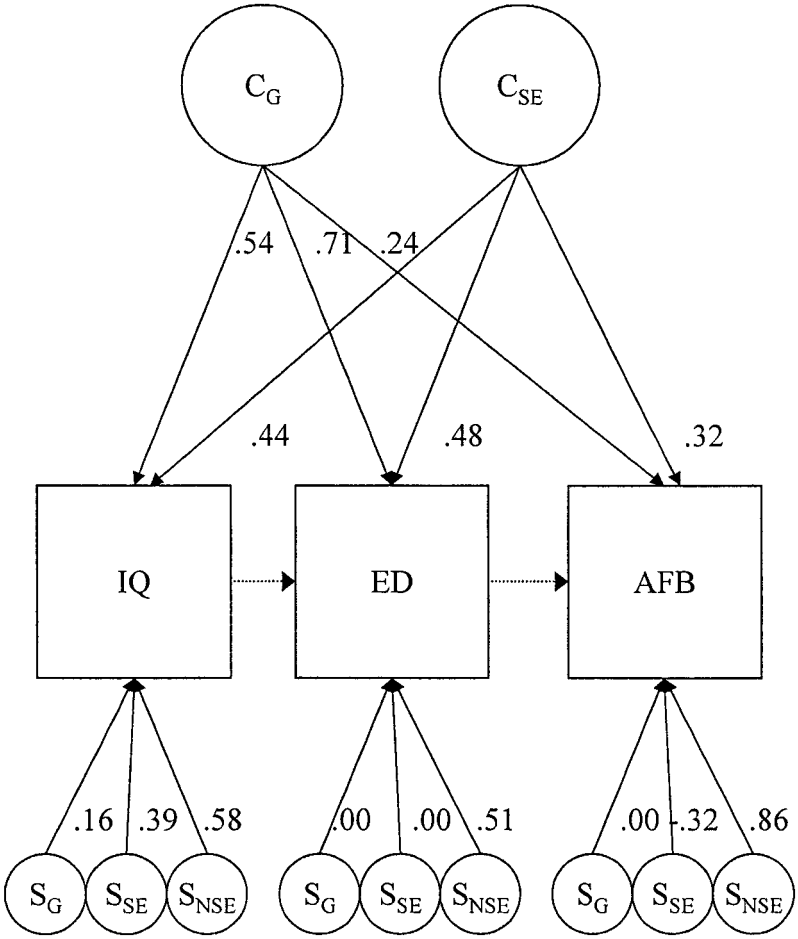

Fig. 4. Illustration of final reduced biometric model with standardized path estimates.

0·35, 0·23 and 0·20 for the same three variables, respectively. The genetic correlation (r_g) of IQ and age of first birth can be estimated from another formula: $r_g = GCOV_{xy}/\sqrt{(V_{Gx}V_{Gy})}$, where $GCOV_{xy}$ is the genetic covariance of x and y and V_{Gx} and V_{Gy} are the genetic variances of x and y, respectively. For example, the genetic correlation of IQ and age of first birth is: $0{\cdot}54 \times 0{\cdot}24/(0{\cdot}32 \times 0{\cdot}06) = 0{\cdot}05$. Table 4 presents the genetic and shared environmental correlations among the three variables using the final biometric model. Genetic correlations are below the diagonal; shared environmental correlations are above the diagonal. The genetic correlation of 1·0 between education and age of first birth is an implication of the model specification because the specific genetic influence on both was estimated to be zero. Thus, all genetic influences between education and age of first birth are common to both.

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Table 4. Genetic and shared environmental correlations among IQ, education (ED) and age of first birth (AFB) within the final biometric model

	IQ	ED	AFB
IQ		0·74	0·53
Education	0·96		0·72
Age of first birth	0·05	1·00	

Note: Correlations below the diagonal represent genetic correlations between variables. Correlations above the diagonal represent shared environmental correlations between variables.

Discussion

In this study, the relationship among IQ, education and age of first birth was examined using both phenotypic and biometric models. In the phenotypic model, it was demonstrated that education partially mediated the association between IQ and age of first birth. According to the model, 65% of the expected 0·31 correlation between IQ and age of first birth was mediated via IQ's association with greater years of education. This finding accords with recent history, when an increase in women's schooling was followed by a decrease in fertility (Wilkie, 1981; Rindfuss & Hirschman, 1984).

The behavioural genetic analyses, however, suggested that after modelling common genetic and environmental influences among all three variables, education did not play a mediational role. In this model, common latent factors were allowed to have an influence on all three variables. With these latent factors in the model, a direct phenotypic mediation of the IQ phenotype on age of first birth via education was no longer necessary; indeed, when these paths were placed into the total biometric model, they failed to create a statistically or substantively meaningful improvement in model fit. The association between IQ, education and age of first birth was carried through the common genetic and environmental influences. The genetic correlation between IQ and age of first birth was 0·05. As expected, this correlation was less than the high (0·96) genetic correlation between IQ and education. A large genetic association between IQ and education has been found by a twin study (Tambs *et al.*, 1989) and accords with a general finding of genetic influence on social class outcomes (Rowe, Vesterdal & Rodgers, 1999). Shared environmental influences were substantially correlated across all three variables.

Total variation in each of the outcomes was also apportioned into genetic and shared environmental influences. Age of first birth was least heritable ($h^2 = 0{\cdot}06$), followed by IQ (0·32) and education (0·50). The shared environmental estimates were 0·20 for age of first birth, 0·23 for education and 0·35 for IQ.

The lack of support for the mediational role of education in this model was somewhat surprising, especially given the support for mediation at the phenotypic

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level. Other researchers, however, have questioned the assumption that education leads to a direct decline in childbearing. In a study of aggregate data from 71 countries, McClamroch (1996) concluded that education seemed to indirectly influence total fertility rates primarily through the percentage of married couples using contraception. Heaton & Forste (1998) proposed that education affects fertility in countries undergoing demographic transition, but once countries pass through their transition, education is no longer as predictive of fertility behaviour. In an analysis of large cohorts of Danish twins, Kohler *et al.* (1999) used different analytic methods to the current study, but nonetheless reached a similar conclusion regarding genetic influences on fertility: they were not mediated through individuals' educational levels.

If the findings of the biometric model are accepted, the apparent phenotypic mediation is spurious on other sources of individual differences. That is, education may not be necessary to delay childbirth – but particular individual differences that lead individuals to seek higher education and to have a higher IQ also inhibit fertility.

Two such traits that are certainly IQ-associated and that could delay childbearing are behavioural inhibition and future orientation. There is an association between the ability to delay gratification and IQ-type abilities, e.g. as in Mischel's classic test of children's ability to wait for a desirable reward rather than receive a less attractive reward immediately (Shoda, Mischel & Peake, 1990). People of higher intelligence may also have a greater future orientation, which along with greater behavioural inhibition may lead them to pursue higher education and to delay the pleasures of having children. Alternatively, higher intelligence may confer greater inhibition because more negative consequences of their behaviour are foreseen, which could be anything from the pain of childbirth to lost career opportunities.

More proximally, higher IQ people are probably better contraceptors. They not only plan their lives more carefully, but when they decide to delay childbearing, they are able to carry out their intentions with the use of birth control pills, condoms or other contraceptive methods. It is thus this addition of a contraceptive technology, more than education advances *per se*, that may allow people with greater IQ to achieve such low levels of fertility and delay childbearing. Convincing proof of the lack of mediation can only be obtained, however, if a culture can be found where IQ is positively associated with age of first birth, contraceptive use is widespread, but where neither men nor women increase their educational levels, or even better, where they stay relatively uneducated. Such a clear test case is unlikely to be found, however, because contraceptive use and better educational opportunities, for the most part, co-occur.

Common shared environmental effects also accounted for a substantial portion of the relationship between intelligence, education and fertility. Because the shared environment is a latent variable in the model, it also may be consistent with many different hypotheses about specific sources of influence. It may be that home environments in which IQ development is fostered are also those that transmit norms, or share norms, with their middle and upper middle class neighbours, against early childbearing. Such motivational effects could be relatively independent of the specific genes that influence IQ level.

Limitations

One limitation of this study lies in the lack of precision in diagnosing sibling pairs. The imprecision biases the design against finding genetic influence, as misclassification of pairs would tend to decrease the expected differences between the sibling groups. In addition, the small difference in coefficients of genetic relatedness (0·375 vs 0·5) yields low statistical power to detect genetic effects. Even with this limitation, however, the study's ability to use a large-scale national US sample provides a unique contribution to the literature on heritability of fertility.

Another limitation of the study lies in the inclusion of only one cohort. While the NLSY is an impressive longitudinal study, past research on fertility behaviours suggests that estimates of environmental and genetic effects can differ substantially over varying cohorts. Thus it is necessary to replicate these results among other age groups.

The study concluded that both shared environment and genetics influence the relationship between intelligence, education and age of first birth. However, the behavioural genetic analysis suggested that education did not mediate the relationship between intelligence and age of first birth, as is typically assumed. Re-examination of the role of education on fertility behaviour is warranted in light of this finding.

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