

March 30, 2015

What Explains the Heritability of Completed Fertility?

Evidence from a Large U.S. Twin Study

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Abstract

In modern societies, individual differences in completed fertility are linked with genotypic differences between individuals. Explaining the heritability of completed fertility has been somewhat inconclusive, with alternative explanations centering on family formation timing, pursuit of education, beliefs about family importance, or other psychological traits. In this paper, I use the twin subsample from the Midlife Development in the United States study to examine these issues. In total, 933 adult twin pairs reported on their completed fertility, age at first birth, age at first marriage, level of education, family formation beliefs, Big Five personality traits, and cognitive ability. I use a quantitative genetic Cholesky decomposition to partition the variance in completed fertility into genetic and environmental variance that is shared with the predictor variables and unique residual variance. I find that genetic influences on completed fertility are strongly related to family formation timing and less strongly, but significantly, with psychological traits.

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In modern, low fertility societies, variation in completed fertility is associated with genotypic differences between individuals (Byars, Ewbank, Govindaraju, & Sterns, 2010; Harden, 2014; Kirk et al., 2001; Milot et al., 2011; Pettay, Kruuk, Jokela, & Lummaa, 2005; Rodgers et al., 2001; Rodgers, Kohler, & Christensen, 2003; Rodgers, Rowe, & Miller, 2000; Zietsch, Kuja-Halkola, Walum, & Verweij, 2014). Some insight into this process can be gained from studies which estimate genetic effects for different segments of the population. When individuals have greater freedom to pursue fertility patterns free from social or economic constraints, a greater proportion of variation in fertility is associated with genetic influences (Bras, Van Bavel, & Mandemakers, 2013; Briley, Harden, & Tucker-Drob, 2015; Kohler, Rodgers, & Christensen, 1999). This indicates that features of the socio-cultural context interact with the expression of genetic influences on fertility. Udry (1996) predicted this effect for low fertility societies. He argued that as social norms and control over fertility weakened (e.g., DeLamater, 1981; Lesthaeghe, 2010; Lesthaeghe & van de Kaa, 1986; van de Kaa, 1987), genetically influenced individual differences would become increasingly linked with the phenotypic expression of fertility. Yet, several alternative explanations are present in the literature to account for the heritability of fertility. In this paper, I contrast explanations centering on family formation timing, pursuit of education, beliefs about the importance of family, and other psychological traits within a large, adult, genetically informative sample. I find that proximal factors, such as fertility timing, can explain the majority of genetic influences on fertility, and psychological characteristics predict genetic influences on fertility via fertility timing.

Explanations for Genetic Influences on Completed Fertility

Generally, genetic influences on completed fertility, a highly complex and socially structured outcome, may be observed if other genetically influenced phenotypes have an effect on levels of fertility. For example, preferences for family size are partially genetically influenced, and these genetic influences are shared with levels of completed fertility (Miller, Bard, Pasta, & Rodgers, 2010). In this example, individuals with genetic predispositions to desire large family sizes tend to have larger families, resulting in genotypic variation becoming linked to variation in completed fertility. Individuals also differ in respect to the timing of their first birth and their first marriage. These phenotypes are partially genetically influenced, and delayed family formation timing is associated with lower completed fertility (Kohler, Rodgers, & Christensen, 2002; Rodgers, Bard, & Miller, 2007; Trumbetta, Markowitz, & Gottesman, 2007). Similarly, individual differences in the pursuit of educational attainment, rather than family formation, are genetically influenced and associated with lower completed fertility (Kohler & Rodgers, 2003; Nisén, Martikainen, Kaprio, & Silventoninen, 2013). Psychological characteristics, such as personality (Jokela, 2012; Skirbekk & Blekesaune, 2013) and cognitive ability (Hopcroft, 2006; Udry, 1978; Van Court & Bean, 1985; von Stumm, Batty, & Deary, 2011), have also been linked to completed fertility. Variation in these phenotypes is substantially influenced by genotypic differences (Bouchard & McGue, 2003).

To complicate matters, these potential explanatory phenotypes are all intercorrelated. Educational success is strongly predicted by cognitive ability (Deary, Strand, Smith, & Fernandes, 2007) and less strongly but substantially by personality (Poropat, 2009). Cognitive ability and personality dimensions are correlated and developmentally intertwined (Cattell, 1987; Goff & Ackerman, 1992). Personality is predictive of fertility intentions (Hutteman, Bleidorn,

Penke, & Denissen, 2012), marriage timing (Jokela, Alvergne, Pollet, & Lummaa, 2011), and childbearing timing (Jokela, Hintsala, Hintsanen, & Keltikangas-Järvinen, 2010). Delayed fertility timing is also predicted by cognitive ability (Neiss, Rowe, & Rodgers, 2002) and educational attainment (Rindfuss, Morgan, & Offutt, 1996). Moreover, many of these associations have been shown to be due to common genetic influences, further obscuring the precise mechanism linking genetic variation to completed fertility (Krapohl et al., 2014; Neiss et al., 2002; Nisén et al., 2013; Wainwright, Wright, Luciano, Geffen, & Martin, 2008).

All of these genetically influenced phenotypes may offer potential explanations for the heritability of completed fertility. As strict social norms for fertility have slowly loosened to allow for a variety of pathways for family formation, individuals are allowed greater freedom to pursue family sizes in line with their genetically influenced preferences, goals, and values. Unfortunately, previous studies have primarily focused on single explanations and not taken a fully multivariate approach. This limitation hinders the ability to conclusively determine whether these associations are unique or shared with other factors, a requirement for properly identifying the mechanisms of genetic influences on fertility.

Method

Participants

The current project uses the Midlife Development in the United States study, a two-wave nationally representative study of adulthood (Ryff et al., 2006). This sample ($N = 7108$) includes a large twin subsample of monozygotic pairs ($n = 354$) and dizygotic pairs ($n = 579$). The sample reflects the diversity of the U.S. population. At the initial wave (1994/1995), participants ranged in age from 25 to 74 years old ($M = 46.38$ years, $SD = 13.00$), and the second wave took place

approximately 10 years later. As described below, I made use of both waves of data to obtain complete fertility histories even for the youngest participants. For the relatively stable demographic and psychological characteristics, I made use of only one measurement wave to limit the potential effect of attrition or age-related change (e.g., Lucas & Donnellan, 2011). In the full sample, a similar number of males ($n = 3395$) and females ($n = 3632$) participated. The racial composition of the sample was predominantly White ($n = 5600$), but participants identified as Black ($n = 321$), Native American ($n = 37$), Asian or Pacific Islander ($n = 57$), some other race ($n = 119$), and multiracial ($n = 42$).

Measures

I drew 13 variables from this dataset. A measure of completed fertility was the primary outcome variable. To explain variance in completed fertility, I used measures of age at first birth, age at first marriage, educational attainment, beliefs about the importance of family, extraversion, agreeableness, conscientiousness, neuroticism, openness to experience, and cognitive ability.

Completed Fertility. A measure of completed fertility was constructed based on the participants' total number of biological children. Participants reported this information at both waves of assessment, and both sources of information were incorporated to create a single variable. The reported number of biological children may be censored by the timing of the survey for younger participants. However, this is a fairly small concern with this sample due to the youngest participants being 34 years old at the second assessment wave. In the United States in 2010, over 85% of period fertility occurred to individuals less than 34 years old (Human Fertility Database). Additionally, 99% of period fertility in the United States occurred to individuals under 41 years of age, and over 85% of the current sample was over 41 years old. For

the vast majority of the sample, completed fertility is known, but additional fertility may occur for a small fraction. This is an important, but minor, limitation. The average participant had 2.09 children ($SD = 1.60$).

Age at First Birth. A measure of age at first birth was constructed based on the participant's age at the time their eldest child was born. Again, participants reported this information at both waves, and this information was integrated. For childless participants, their current age at the time of the survey was entered as their age at first birth. Childless participants over 50 years of age are unlikely to have children for biological reasons. Following the precedent of previous studies (e.g., Kohler et al., 1999), age at first birth was entered as 50 years of age for childless participants over 50 in order to reduce outliers. The average participant had their first child at 28.83 years of age ($SD = 9.35$).

Age at First Marriage. A measure of age at first marriage was constructed based on the participant's age at the time of their first marriage based on information reported at both waves of assessment. This variable was constructed similarly to age at first birth in that unmarried individuals were assigned their current age capped at 50 years of age. Although there is not the same sort of biological limit on age at first marriage as there is for age at first birth, the same coding was applied to maximize comparability. Further, no participants reported a first marriage after age 50. The average participant was first married at 25.87 years of age ($SD = 8.15$).

Educational Attainment. Participants reported their educational attainment at the first assessment wave. Substantial variability was observed for educational attainment. Participants obtained some grade school ($n = 38$), eighth grade/junior high school ($n = 127$), some high school ($n = 516$), GED ($n = 109$), high school degree ($n = 1,951$), 1 to 2 years of college ($n = 1,302$), 3 or more years of college ($n = 333$), 2 year degree ($n = 538$), bachelor's degree ($n =$

1,240), some graduate school ($n = 197$), master's degree ($n = 487$), or a professional degree ($n = 257$).

Beliefs about the Importance of Family. At the first assessment wave, participants reported on their family formation views on a 7-point Likert scale ranging from strongly disagree to strongly agree. Four items were used to make the scale. The items asked whether “[men/women] can have full and happy lives without [marrying/having any children].” The mean response to the four items was taken. This scale had good internal consistency ($\alpha = .88$). The average response to this scale was 5.40 ($SD = 1.53$).

Big Five Personality Traits. At the first assessment wave, participants indicated the accuracy of several self-descriptive adjectives on a 4-point Likert scale ranging from not at all to a lot. Adjectives were selected to index extraversion (“outgoing, friendly, lively, active, talkative”), agreeableness (“helpful, warm, caring, softhearted, sympathetic”), conscientiousness (“organized, responsible, hardworking, careless”), neuroticism (“moody, worrying, nervous, calm”), and openness to experience (“creative, imaginative, intelligent, curious, broad-minded, sophisticated, adventurous”). The mean response was taken, reverse coding were necessary. Internal consistency was good for extraversion ($\alpha = .78$), agreeableness ($\alpha = .80$), neuroticism ($\alpha = .74$), and openness to experience ($\alpha = .77$), but was substantially lower for conscientiousness ($\alpha = .58$). The average response for extraversion, agreeableness, conscientiousness, neuroticism, and openness to experiences was 3.20 ($SD = 0.56$), 3.49 ($SD = 0.49$), 3.42 ($SD = 0.44$), 2.24 ($SD = 0.66$), and 3.02 ($SD = 0.53$), respectively.

Cognitive Ability. At the second assessment wave, participants completed the Brief Test of Adult Cognition by Telephone, an instrument designed to assess cognitive ability (Tun &

Lachman, 2006). This variable was only assessed at the second measurement wave ($n = 3973$, 56% of original sample). A composite was taken based on z-scores of tests of immediate word list recall, delayed word list recall, digits backwards, category fluency, number series, and backward counting. These subtests are designed to assess various domains of cognitive ability and are all intercorrelated (mean $r = .34$). By creating a composite, this variable assesses general cognitive ability.

Analytic Approach

Quantitative genetic methodology makes use of correlations between family members with known genetic similarity. In the classical twin design (Neale & Cardon, 1992), reared together monozygotic twin pairs are compared to reared together dizygotic twin pairs to estimate three variance components. First, additive genetic effects (A) index variation in a phenotype that is associated with genotypic sequence variation between individuals. Second, shared environmental effects (C) index variation associated with between-family environmental differences (i.e., environmental effects that make siblings living in the same home similar). Third, nonshared environmental effects (E) index variation associated with within-family environmental differences (i.e., environmental effects that make siblings living in the same home different, plus measurement error). This decomposition is accomplished by comparing the similarity of monozygotic twins, who share nearly identical genetic material, with dizygotic twins, who share on average 50% of segregating genetic material. If monozygotic twins are more similar on a phenotype than dizygotic twins, this implies genetic influences on the phenotype. To the extent that monozygotic twins are more similar to one another than implied by genetic influences, this is attributable to shared environmental influences. To the extent that monozygotic twins are *dissimilar*, this is attributable to the nonshared environment. In

multivariate extensions of the classical twin design, cross-twin cross-phenotype correlations are the primary outcome of interest. If one twin's score on a phenotype is a better predictor of the other twin's score on a separate phenotype for monozygotic twins compared to dizygotic twins, this implies genetic influences on the covariation of the two phenotypes.

Figure 1A displays the primary analytic approach for the current study. A Cholesky decomposition (Neale & Cardon, 1992) was used to partition the variance in completed fertility into genetic and environmental variance that is shared with the predictor variables and unique residual variance. In this context, the cross-pathways are the primary parameters of interest. If the a_{12} parameter is significant, this indicates that genetic influences on the predictor explain some of the genetic influences on completed fertility. If the c_{12} parameter is significant, it indicates that there are between-family influences on completed fertility that are shared with the predictor variable (e.g., childhood socioeconomic status, religious upbringing, race/ethnicity). If the e_{12} parameter is significant, it indicates that there are within-family influences that are common to the predictor and completed fertility. Put differently, this parameter indicates whether the sibling that is higher (or lower) on the predictor is also higher (or lower) on completed fertility, *after taking genetic and shared environmental confounds into account*. Thus, this parameter represents a quasi-causal effect of the predictor on completed fertility (D'Onofrio, Lahey, Turkheimer, & Lichtenstein, 2013). Parameters labeled with a subscript of $_{11}$ indicate genetic and environmental influences on the predictor variable. Parameters labeled with a subscript of $_{22}$ indicate *residual* genetic and environmental influences after taking into account genetic and environmental influences shared with the predictor.

It may be the case that multiple predictor phenotypes explain genetic variance in fertility. These predictors may explain unique genetic variance in fertility or overlapping genetic variance.

To test for this possibility, the bivariate Cholesky decomposition can be extended to include multiple variables (Figure 1B). In this context, interpretation of cross-paths is similar to multiple regression analysis in the sense that covariation in predictors is controlled. However, the order that variables are entered into the model has a bearing on the interpretation of the cross-paths as variables entered into the model earlier (i.e., toward the left-hand side of the model in Figure 1B) explain variance in later variables (i.e., toward the right-hand side of the model in Figure 1B). Thus, if two variables explain common genetic variance with fertility, the variable entered into the model first would appear to have a unique relation with fertility. This limitation can be avoided by logically entering variables based on time ordering (e.g., age at first birth occurs before completed fertility). For the psychological variables, there is no clear time ordering and interpreting multiple models with different ordering can clarify the unique and overlapping variance components (Loehlin, 1996).

To ensure that the results were not influenced by cohort trends in fertility or sex-differences, all analyses were conducted with phenotypes residualized for sex, age, age², and a sex \times age interaction, as is standard in quantitative genetic analyses (McGue & Bouchard, 1984). For psychological phenotypes that did not display shared environmental effects in univariate analyses, shared environmental effects were dropped from subsequent analyses to aid interpretation and convergence. In this age range, minimal shared environmental effects are very common for psychological phenotypes (e.g., Bouchard & McGue, 2003). Shared environmental influences for the fertility outcomes were not dropped as there is much less previous evidence for minimal effects, and the estimates were non-trivial. All models were fit using full-information maximum-likelihood estimation with *Mplus* statistical software (Muthén & Muthén, 1998-2010).

Results

Completed Fertility

Table 1 presents results from bivariate Cholesky decompositions predicting variance in completed fertility. Approximately 27% of the variance in completed fertility was associated with genotypic differences, 7% with shared environmental effects, and the remaining 66% with nonshared environmental effects. Significant genetic cross-paths were found for age at first birth, age at first marriage, beliefs about the importance of family, agreeableness, and conscientiousness. Fertility timing displayed the strongest genetic association. Genetic influences on age at first marriage fully explained the genetic influences on completed fertility, and genetic influences on age at first birth reduced the residual genetic variance in completed fertility to only marginally significant and accounting for only 9% of the variance in completed fertility. Individuals with genetic predispositions for early fertility timing tend to have larger families. Significant shared environmental cross-paths were found for age at first marriage, indicating shared family-level influences. Significant nonshared environmental cross-paths were found for age at first birth, age at first marriage, and cognitive ability. Holding genetic and family-level influences constant, these results indicate that individuals that delay family formation tend to have smaller family sizes, and individuals with higher cognitive ability tend to have larger families. A marginal nonshared environmental cross-path was also found for agreeableness. The more agreeable (monozygotic) twin (raised in the same shared environment) tends to have slightly fewer children. These effects are fairly small, and residual nonshared environmental effects explain the majority of variance in completed fertility.

To evaluate whether these effects were unique or shared with other predictors, a Cholesky decomposition was fit in which all phenotypes that significantly accounted for genetic variance in completed fertility were included. Age at first marriage was not included in this step

as it fully explained genetic variance in completed fertility, and later analyses explore this covariation more fully. Thus, agreeableness was modeled first, followed by conscientiousness, beliefs about family formation, and age at first birth as predictors of completed fertility. In this model, all genetic cross-paths remained significantly different from zero, except age at first birth ($p = .093$). When age at first birth was modeled first, the genetic cross-path is highly significant ($p < .001$). This indicates that there are common genetic influences on age at first birth, other psychological phenotypes, and completed fertility. In these models, residual genetic effects explained only 7% of the variance in completed fertility ($p = .300$).

Age at First Birth

Because family formation timing was able to largely explain the genetic influences on completed fertility, the remaining analyses focus on explaining genetic variation in age at first birth and marriage. Table 2 presents results from bivariate Cholesky decompositions predicting variance in age at first birth. Approximately 15% of the variance in age at first birth was associated with genotypic differences, 13% with shared environmental effects, and 72% with nonshared environmental effects. Genetic influences on age at first marriage fully explained genetic influences on age at first birth. Additional significant genetic cross-paths were found for beliefs about the importance of family, extraversion, agreeableness, and cognitive ability. Individuals with genetic predispositions for later age at first marriage and high cognitive ability tended to have a later age at first birth, and individuals with genetic predispositions for high levels of beliefs about the importance of family, extraversion, and agreeableness tended to have an earlier age at first birth. A significant shared environmental cross-path was found for educational attainment, indicating that family-level supports for education also delay age at first birth. Significant nonshared environmental cross-paths were found for age at first marriage,

educational attainment, agreeableness, and cognitive ability. Holding genetic and family-level influences constant, individuals with a later age at first marriage, higher levels of educational attainment, and higher levels of agreeableness tend to have a later age at first birth, and individuals with higher levels of cognitive ability tend to have an earlier age at first birth.

To evaluate whether these effects were unique or common to the predictors, cognitive ability, extraversion, agreeableness, and beliefs about the importance of family were modeled as predictors of completed fertility. Age at first marriage was not included as it fully explained the genetic variance in age at first birth, and the next section focuses on this variable. In the full Cholesky decomposition, each psychological phenotype retained a significant genetic cross-path. This indicates that the psychological phenotypes explain separate, small portions of the genetic influence on age at first birth. In this model, residual genetic effects explained only 2% of the variance in age at first birth ($p = .799$).

Age at First Marriage

Table 3 presents results from bivariate Cholesky decompositions predicting variance in age at first marriage.¹ Approximately 29% of the variance in age at first marriage was associated with genotypic differences, 9% with shared environmental effects, and 62% with nonshared environmental effects. Significant genetic cross-paths were found for beliefs about the importance of family, extraversion, and cognitive ability. Individuals with genetic predispositions for higher beliefs in the importance of family and extraversion tend to have an earlier age at first marriage, and individuals with genetic predispositions for high cognitive ability tend to have a later age at first marriage. No significant shared or nonshared

¹ Age at first birth was not included as a predictor of age at first marriage as this model is fully recoverable from the results presented in Table 2 with age at first marriage as a predictor of age at first birth (Loehlin, 1996).

environmental cross-paths were found. In a model that included each phenotype with a significant genetic cross-path, each genetic cross-path remained significant. This indicates that the phenotypes explain unique genetic variance in age at first marriage. However, residual genetic effects explained 24% of the variance in age at first marriage ($p = .004$).

Figure 2 graphically summarizes the genetic cross-paths from each of the demographic characteristics and psychological phenotypes on the fertility outcomes.

Discussion

Individual differences in the level and timing of fertility are associated with genotypic variation between individuals. Modern reproductive behavior in low fertility societies is subject to substantial socio-cultural influences that may interact with an individual's genetic predispositions. For example, some individuals may readily accept novel social norms and values for family formation (e.g., Lesthaeghe, 2010), whereas others may respond more slowly. As social control over fertility practices is diminished in such socio-cultural environments, individuals will be able to express their genetically influenced preferences, desires, goals, or other psychological phenotypes that influence the level or timing of fertility to a greater extent (Udry, 1996). Implicit evidence for this process has been found in that the heritability of fertility tends to be largest for those with the greatest freedom or social capital to pursue individualized fertility goals (Bras et al., 2013; Briley et al., 2014; Kohler et al., 1999). Due to such dynamic interaction between genetic predispositions and the socio-cultural context of fertility, it is possible that levels of fertility will remain linked to genotypic differences between individuals.

In the current paper, I demonstrate that genetic influences on fertility are associated with genetic influences on other demographic and psychological characteristics. Genetic influences on

completed fertility and age at first birth are fully explained by genetic influences on age at first marriage. This implies that marriage timing is a crucial individual difference marker for understanding genetic effects on fertility. Other psychological phenotypes, such as beliefs about the importance of family, extraversion, and cognitive ability, consistently explained genetic variation in fertility timing. Yet, nearly a quarter of the variance in age at first marriage was associated with genetic effects not shared with the demographic characteristics or psychological phenotypes. This implies that there are important other phenotypes that may explain this variation and provide a more mechanistic account of how genetic influences on fertility manifest.

Importantly, each of the fertility outcomes was primarily associated with nonshared environmental variation, which includes unique life experiences, idiosyncratic or time-limited effects, and measurement error. This means that efforts to understand the causes and consequences of fertility timing will need to identify these unique effects that influence fertility trajectories. For example, those that marry earlier tend to have larger families. This effect is mediated both by genetic and nonshared environmental factors. The nonshared environmental effect represents a quasi-causal association between early marriage timing and larger family size. The genetic association may be due to psychological phenotypes (e.g., beliefs about the importance of family, extraversion, cognitive ability), but the nonshared environmental effects were not well-documented in this study. In fact, none of the phenotypes included in this study explained a significant portion of the nonshared environmental effects on age at first marriage. These effects may occur earlier in development, such as early dating relationships.

A number of explanations are present in the literature to explain the heritability of fertility. The current results support some explanations more than others. Fertility timing, both in terms of age at first birth and marriage, can largely explain genetic influences on the level of

fertility. This makes intuitive sense as these variables are very proximate to fertility, and fertility timing is likely influenced by similar motivational attributes as the level of fertility. Cognitive ability and beliefs about the importance of family emerged as the primary psychological phenotypes that were associated with fertility timing. Personality traits displayed some significant associations, but these were small in magnitude. Educational attainment did not significantly predict completed fertility or age at first marriage, but the genetic cross-path effect size was relatively large compared to the psychological phenotypes.

The current study has several important strengths and limitations. A large, genetically informative, adult sample with in-depth psychological assessments was used to explore the genetic and environmental influences on the level and timing of fertility. However, some of the youngest members of the sample may not have fully completed their fertility at the time of the survey. Given the age of the youngest participants and their proportion of the total sample, this is likely a minor concern. Additionally, the assumptions of the twin model, such as the equal environments assumption, are other potential concerns when estimating quantitative genetic models. A wealth of evidence supports the validity of these assumptions (e.g., Conley, Rauscher, Dawes, Magnusson, & Siegal, 2013), and recent molecular genetic work using measured genetic information in unrelated individuals has found similar estimates of heritability for fertility phenotypes (Tropf, Mills, Stulp, Barban, & Snieder, 2014).

To ensure that the current results were not driven by gender differences or cohort trends, age and gender were controlled, as is common in quantitative genetic analyses (Bouchard & McGue, 1983). However, the genetic and environmental associations likely differ across birth cohort or gender (e.g., Kohler et al., 1999). Although the current sample is large compared to many twin studies, it is not sufficiently powered to detect the effects reported here when the

sample is broken down by gender or specific birth cohorts. It may be the case that many of the effects are strengthening over time (Jokela, 2012; Skirbekk & Blekesaune, 2013), and analyses of recently born individuals, who only experience loosely structured fertility norms, would show stronger effects of psychological phenotypes. Further, sex-limitation models may aid in explaining the persistence of genetic influences on fertility outcomes by demonstrating antagonistic pleiotropy (Neale & Cardon, 1992).

Interpretation of the presented models assumed that demographic and psychological variables took chronological and causal precedent over fertility variables. However, bidirectional effects between fertility and psychological development have been documented (Hutteman, Bleidorn, Penke, & Denissen, 2013; Jokela, Kivimäki, Elovainio, & Keltikangas-Järvinen, 2009; Kohler, Behrman, & Skytthe, 2005; Nelson, Kushlev, & Lyubomirsky, 2014). Therefore, the genetic and environmental cross-paths may be reasonably interpreted as genetic influences on fertility that have an effect on the demographic and psychological outcomes. For educational attainment, personality, and cognitive ability, the development of these outcomes is largely established before individuals enter the major childbearing years (Barro & Lee, 2013; Briley & Tucker-Drob, 2014; Tucker-Drob & Briley, 2014). This renders the current interpretation as the most plausible. For beliefs about the importance of family, it is much more likely that an individual's fertility history colors their interpretation about the value of family (Trussell, Vaughan, & Stanford, 1999). For example, individuals with a large family may be more likely to interpret this as something essential to their identity, whereas unmarried individuals may be more likely to interpret marriage and family as something less consequential. However, prospectively measured fertility motivation also predicts fertility outcomes (Miller et al., 2010), which

provides some support to the current interpretation. Additional longitudinal research would be required to fully parse apart these alternative explanations.

In conclusion, the current project demonstrates the importance of integrating genetically informative research into socio-demographic frameworks. In a large, adult, genetically informative sample, variation in completed fertility was linked with genotypic variation across individuals. This effect was fully explained by genetic influences on marriage timing. The timing of marriage represents an early indicator for an individual's ultimate fertility trajectory. Genetically influenced psychological phenotypes, such as personality and cognitive ability, explain some portion of the genetic influences on fertility, but much unexplained variance remains concerning the nonshared environmental effects (e.g., unique life experiences) that influence the level and timing of fertility.

References

- Barro, R. J., & Lee, J. W. (2013). A new data set of educational attainment in the world, 1950-2010. *Journal of Development Economics*, *104*, 184-198.
- Bouchard, T.J., Jr., & McGue, M. (2003). Genetic and environmental influences on human psychological differences. *Developmental Neurobiology*, *54*, 4-45.
- Bras, H., Van Bavel, J., & Mandemakers, K. (2013). Unraveling the intergenerational transmission of fertility: Genetic and shared-environment effects during the demographic transition in the Netherlands, 1810-1910. *History of the Family*, *18*, 116-134.
- Briley, D. A., & Tucker-Drob, E. M. (2014). Genetic and environmental continuity in personality development: A meta-analysis. *Psychological Bulletin*, *140*, 1303-1331.
- Briley, D. A., Harden, K. P., & Tucker-Drob, E. M. (2015). Genotype \times cohort interaction on completed fertility and age at first birth. *Behavior Genetics*, *45*, 71-83. doi: 10.1007/s10519-014-9693-3
- Byars, S.G., Ewbank, D., Govindaraju, D.R., & Stearns, S.C. (2010). Natural selection in a contemporary human population. *Proceedings of the National Academy of Science*, *107*, 1787-1792.
- Cattell, R. B. (1987). *Intelligence: Its structure, growth, and action*. Amsterdam: North-Holland.
- Conley, D., Rauscher, E., Dawes, C., Magnusson, P. K., & Siegal, M. L. (2013). Heritability and the equal environments assumption: Evidence from multiple samples of misclassified twins. *Behavior Genetics*, *43*, 415-426.
- Deary, I. J., Strand, S., Smith, P., & Fernandes, C. (2007). Intelligence and educational achievement. *Intelligence*, *35*, 13-21.
- DeLamater, J. (1981). The social control of sexuality. *Annual Review of Sociology*, *7*, 263-290.

- D'Onofrio, B. M., Lahey, B. B., Turkheimer, E., & Lichtenstein, P. (2013). Critical need for family-based, quasi-experimental designs in integrating genetic and social science research. *American Journal of Public Health, 103*, S46-55.
- Goff, M., & Ackerman, P. L. (1992). Personality-intelligence relations: Assessment of typical intellectual engagement. *Journal of Educational Psychology, 84*, 537-552.
- Harden, K.P. (2014). Genetic influences on adolescent sexual behavior: Why genes matter for environmentally oriented researchers. *Psychological Bulletin, 140*, 434-465.
- Hopcroft, R. L. (2006). Sex, status, and reproductive success in the contemporary United States. *Evolution and Human Behavior, 27*, 104-120.
- Human Fertility Database. Max Planck Institute for Demographic Research (Germany) and Vienna Institute of Demography (Austria). Available at www.humanfertility.org (data downloaded on December 3, 2013).
- Hutteman, R., Bleidorn, W., Penke, L., & Denissen, J. J. A. (2013). It takes two: A longitudinal dyadic study on predictors of fertility outcomes. *Journal of Personality, 81*, 487-498.
- Jokela, M. (2012). Birth-cohort effects in the association between personality and fertility. *Psychological Science, 23*, 835-841.
- Jokela, M., Alvergne, A., Pollet, T. V., & Lummaa, V. (2011). Reproductive behavior and personality traits of the five factor model. *European Journal of Personality, 25*, 487-500.
- Jokela, M., Hintsala, T., Hintsanen, M., & Keltikangas-Järvinen, L. (2010). Adult temperament and childbearing over the life course. *European Journal of Personality, 24*, 151-166.
- Jokela, M., Kivimäki, M., Elovainio, M., & Keltikangas-Järvinen, L. (2009). Personality and having children: A two-way relationship. *Journal of Personality and Social Psychology, 96*, 208-230.

Kirk, K.M., Blomberg, S., Duffy, D.L., Heath, A.C., Owens, I.P.F., & Martin, N.G. (2001).

Natural selection and quantitative genetics of life-history traits in western women: A twin study. *Evolution*, *55*, 423-435.

Kohler, H.-P., Behrman, J.R., & Skytthe, A. (2005). Partner + children = happiness? The effects of partnerships and fertility on well-being. *Population and Development Review*, *31*, 407-445.

Kohler, H.-P., & Rodgers, J.L. (2003). Education, fertility, and heritability: Explaining a paradox. *Offspring: Human Fertility Behavior in Biodemographic Perspective*, eds Wachter, K.W., & Bulatao, R.A. (National Academies Press, Washington D.C.), pp 46-90.

Kohler, H.-P., Rodgers, J.L., & Christensen, K. (1999). Is fertility behavior in our genes? Findings from a Danish twin study. *Population and Development Review*, *25*, 253-288.

Kohler, H.-P., Rodgers, J.L., & Christensen, K. (2002). Between nature and nurture: The shifting determinants of female fertility in Danish twin cohorts. *Biodemography and Social Biology*, *49*, 218-248.

Krapohl, E., Rimfeld, K., Shakeshaft, N. G., Trzaskowski, M., McMillan, A., Pingault, J.-B., ... & Plomin, R. (2014). The high heritability of educational achievement reflects many genetically influenced traits, not just intelligence. *Proceedings of the National Academy of Sciences of the United States of America*, *111*, 15273-15278.

Lesthaeghe, R. (2010). The unfolding story of the second demographic transition. *Population and Development Review*, *36*, 211-251.

- Lesthaeghe, R., & van de Kaa, D.J. (1986). Twee demografische transitie's? *Bevolking: Groei en Krimp. Mens en Maatschappij*, eds Lesthaeghe, R.J., & van de Kaa, D.J. (VanLoghum-Slaterus, Deventer), pp 9-24.
- Loehlin, J. C. (1996). The Cholesky approach: A cautionary note. *Behavior Genetics*, 26, 65-69.
- Lucas, R. E., & Donnellan, M. B. (2011). Personality development across the life span: Longitudinal analyses with a national sample from Germany. *Journal of Personality and Social Psychology*, 101, 847-861.
- McGue, M., & Bouchard, T.J., Jr. (1984). Adjustment of twin data for the effects of age and sex. *Behavior Genetics*, 14, 325-343.
- Miller, W.B., Bard, D.E., Pasta, D.J., & Rodgers, J.L. (2010). Biodemographic modeling of the links between fertility motivation and fertility outcomes in the NLSY79. *Demography*, 47, 393-414.
- Milot, E., Mayer, F.M., Nussey, D.H., Boisvert, M., Pelletier, F., & Réale, D. (2011). Evidence for evolution in response to natural selection in a contemporary human population. *Proceedings of the National Academy of Science*, 108, 17040-17045.
- Muthén, L.K., & Muthén, B.O. (1998-2010). *Mplus user's guide* (6th ed.). Los Angeles, CA: Muthén and Muthén.
- Neale, M.C., & Cardon, L.R. (1992). *Methodology for genetic studies of twins and families*. Dordrecht: Kluwer Academic Press.
- Neiss, M., Rowe, D. C., & Rodgers, J. L. (2002). Does education mediate the relationship between IQ and age of first birth? A behavioural genetic analysis. *Journal of Biosocial Science*, 34, 259-275.

- Nelson, S.K., Kushlev, K., & Lyubomirsky, S. (2014). The pains and pleasures of parenting: When, why, and how is parenthood associated with more or less well-being? *Psychological Bulletin, 140*, 846-895.
- Nisén, J., Martikainen, P., Kaprio, J., & Silventoninen, K. (2013). Educational differences in completed fertility: A behavioral genetic study of Finnish male and female twins. *Demography, 50*, 1399-1420.
- Pettay, J.E., Kruuk, L.E.B., Jokela, J., & Lummaa, V. (2005). Heritability and genetic constraints of life-history trait evolution in preindustrial humans. *Proceedings of the National Academy of Science, 102*, 2838-2843.
- Poropat, A. E. (2009). A meta-analysis of the five-factor model of personality and academic performance. *Psychological Bulletin, 135*, 322-338.
- Rindfuss, R. R., Morgan, S. P., & Offutt, K. (1996). Education and the changing age pattern of American fertility: 1963-1989. *Demography, 33*, 277-290.
- Rodgers, J.L., Hughes, K., Kohler, H.-P., Christensen, K., Doughty, D., Rowe, D.C., & Miller, W.B. (2001). Genetic influence helps explain variation in human fertility: Evidence from recent behavioral and molecular genetic studies. *Current Directions in Psychological Science, 10*, 184-188.
- Rodgers, J.L., Bard, D.E., & Miller, W.B. (2007). Multivariate cholesky models of human female fertility patterns in the NLSY. *Behavior Genetics, 37*, 345-361.
- Rodgers, J.L., Kohler, H.-P., & Christensen, K. (2003). Genetic variance in human fertility: Biology, psychology, or both? *The Biodemography of Human Reproduction and Fertility*, eds Rodgers, J.L., & Kohler, H.-P. (Kluwer, Boston), pp. 229-250.

- Rodgers, J.L., Rowe, D.C., & Miller, W.B. (Eds.) (2000). *Genetic influences on human fertility and sexuality: Theoretical and empirical contributions from the biological and behavioral sciences*. (Kluwer, Boston).
- Ryff, C., Almeida, D.M., Ayanian, J.S., Carr, D.S., Cleary, P.D., Coe, C., & Williams, D. (2006). *Midlife development in the United States (MIDUS II), 2004-2006*. Madison, WI: University of Wisconsin, Survey Center.
- Skirbekk, V., & Blekesaune, M. (2013, August 5). Personality traits increasingly important for male fertility: Evidence from Norway. *European Journal of Personality*. Advanced online publication. doi: 10.1002/per.1936
- Stearns, S.C., Byars, S.G., Govindaraju, D.R., & Ewbank, D. (2010). Measuring selection in contemporary human populations. *Nature Reviews Genetics*, *11*, 611-622.
- Tropf, F., Mills, M., Stulp, G., Barban, N., & Snieder, H. (2014). *Human fertility, molecular genetics, and natural selection in modern societies*. Paper presented at the 2014 Annual Meeting of the Behavior Genetics Association. Charlottesville: VA.
- Trumbetta, S.L., Markowitz, E.M., & Gottesman, I.I. (2007). Marriage and genetic variation across the lifespan: Not a steady relationship? *Behavior Genetics*, *37*, 362-375.
- Trussell, J., Vaughan, B., & Stanford, J. (1999). Are all contraceptive failures unintended pregnancies? Evidence from the 1995 National Survey of Family Growth. *Family Planning Perspectives*, *31*, 246-247.
- Tucker-Drob, E. M., & Briley, D. A. (2014). Continuity of genetic and environmental influences on cognition across the life span: A meta-analysis of longitudinal twin and adoption studies. *Psychological Bulletin*, *140*, 949-979.

- Tun, P. A., & Lachman, M. E. (2006). Telephone assessment of cognitive function in adulthood: The Brief Test of Adult Cognition by Telephone (BTACT). *Age and Ageing, 35*, 629-633.
- Udry, J. R. (1978). Differential fertility by intelligence: The role of birth planning. *Biodemography and Social Biology, 25*, 10-14.
- Udry, J.R. (1996). Biosocial models of low-fertility societies. *Population and Development Review, 22*, 325-336.
- Van Court, M., & Bean, F. D. (1985). Intelligence and fertility in the United States: 1912-1982. *Intelligence, 9*, 23-32.
- van de Kaa DJ (1987). Europe's second demographic transition. *Population Bulletin, 42*, 1-57.
- von Stumm, S., Batty, G. D., & Deary, I. J. (2011). Marital status and reproduction: Associations with childhood intelligence and adult social class in the Aberdeen children of the 1950s study. *Intelligence, 39*, 161-167.
- Wainwright, M. A., Wright, M. J., Luciano, M., Geffen, G. M., & Martin, N. G. (2008). Genetic covariation among facets of openness to experience and general cognitive ability. *Twin Research and Human Genetics, 11*, 275-286.
- Zietsch, B.P., Kuja-Halkola, R., Walum, H., & Verweij, K.J.H. (2014). Perfect genetic correlation between number of offspring and grandoffspring in an industrialized human population. *Proceedings of the National Academy of Science, 111*, 1032-1036.

Figure 1. A. Example Cholesky decomposition. Parameters with subscript “₁₁” represent variance in the predictor. Parameters with subscript “₁₂” represent variance in completed fertility shared with the predictor variable. Parameters with subscript “₂₂” represent unique residual variance in completed fertility. Parameters are reported for additive genetic effects (*a*), shared environmental effects (*c*), and nonshared environmental effects (*e*). Latent variables represent additive genetic effects on the predictor (A_p) and fertility (A_f), shared environmental effects on the predictor (C_p) and fertility (C_f), and nonshared environmental effects on the predictor (E_p) and fertility (E_f). Only one member of a twin pair is represented. **B.** Extended Cholesky decomposition. Interpretation is similar to above, except an additional predictor variable has been included. Multiple additional predictors could also be added. Only one member of a twin pair is represented.

Figure 2. Summary of genetic cross-paths and standard errors from demographic characteristics and psychological phenotypes on fertility outcomes.

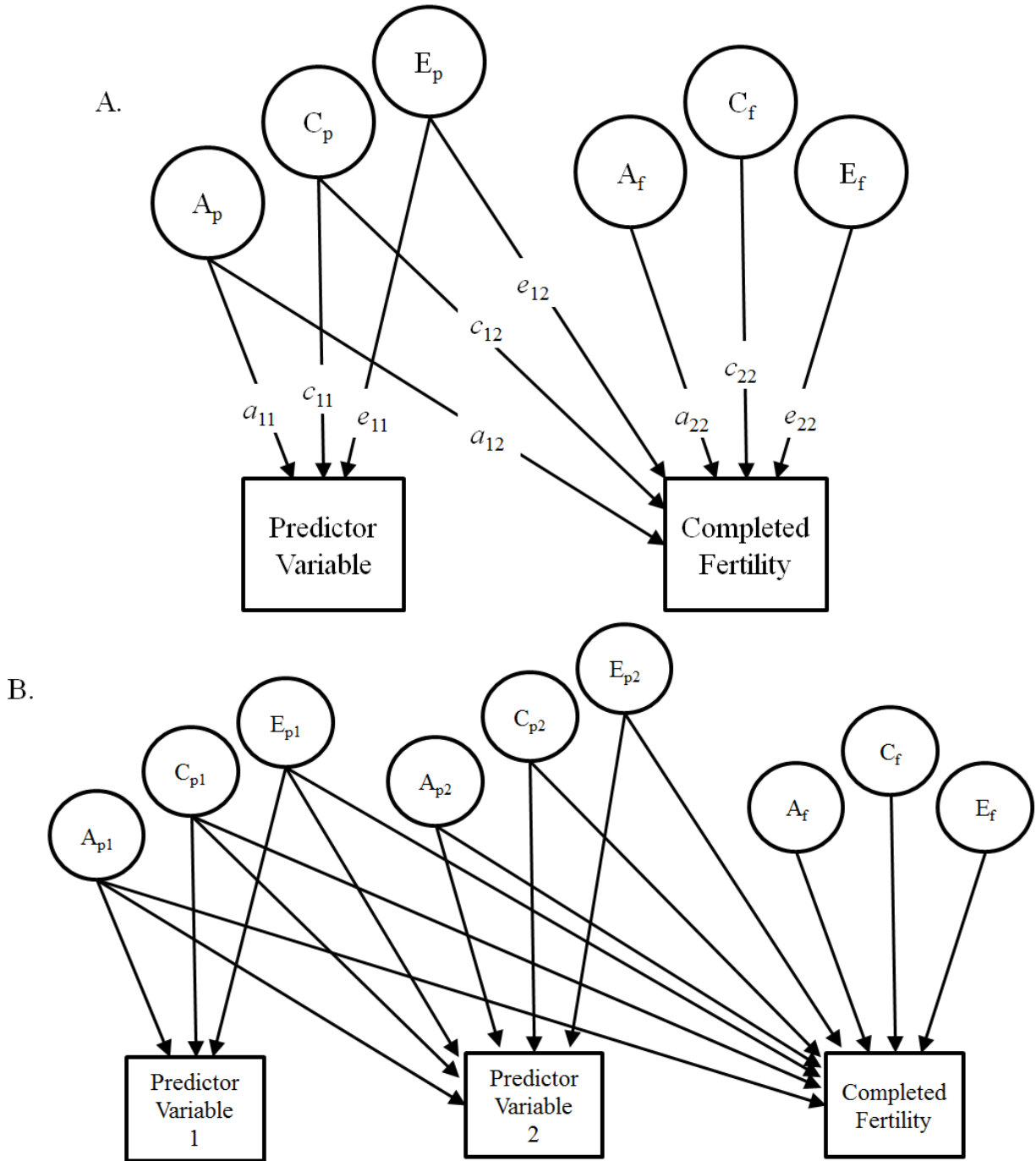


Figure 1.

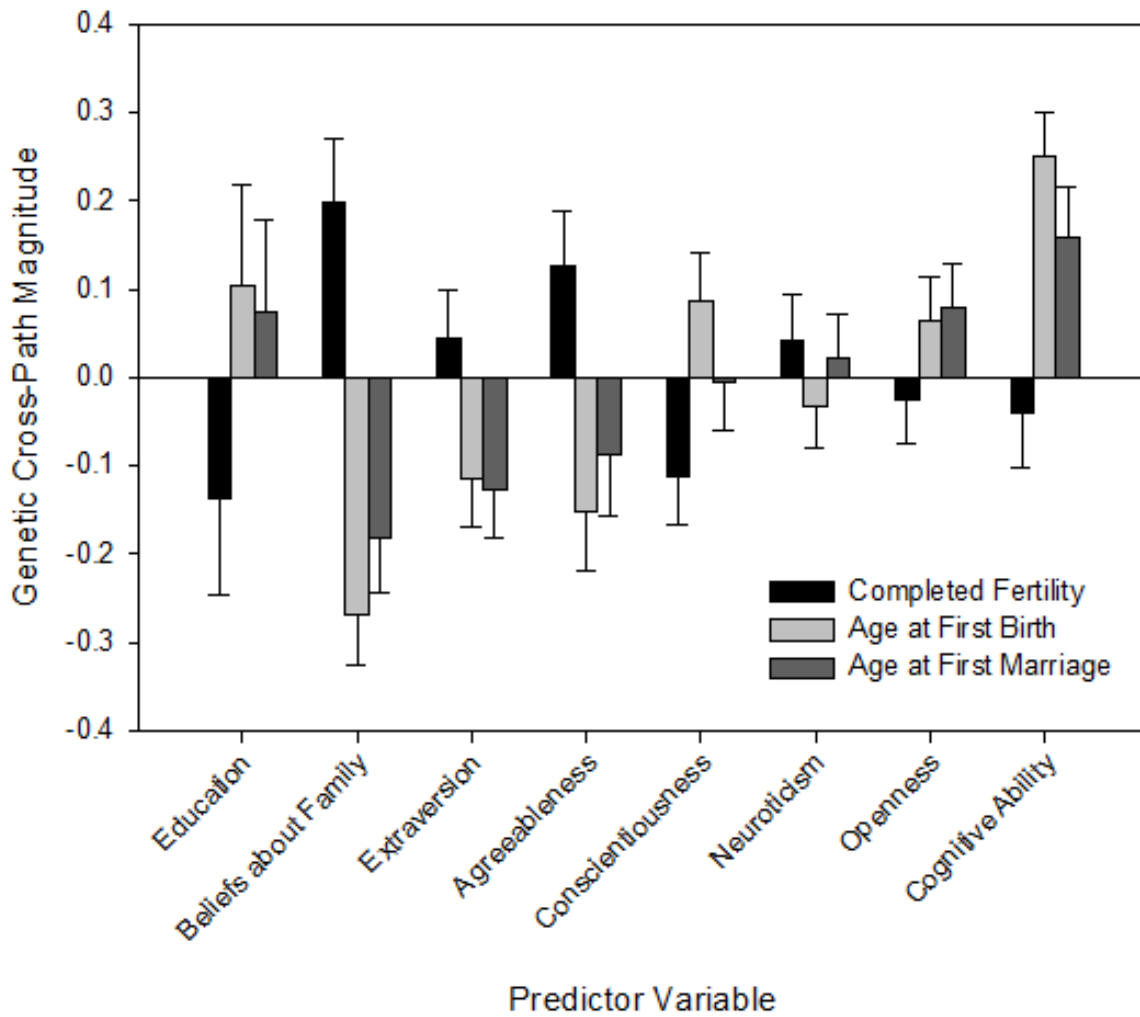


Figure 2.

Table 1. Quantitative genetic decomposition of variance in completed fertility

Predictor	a_{11}	c_{11}	e_{11}	a_{12}	c_{12}	e_{12}	a_{22}	c_{22}	e_{22}
Univariate Completed Fertility	-	-	-	-	-	-	.524 (.132) ***	.269 (.193)	.808 (.036) ***
Age at First Birth	.417 (.180) *	.336 (.158) *	.844 (.036) ***	-.437 (.156) **	-.058 (.211)	-.499 (.037) ***	.295 (.178) †	.251 (.172)	.637 (.028) ***
Age at First Marriage	.530 (.099) ***	.315 (.115) **	.787 (.036) ***	-.507 (.085) ***	.296 (.107) **	-.228 (.036) ***	.000 (.000)	.000 (.000)	.777 (.029) ***
Education	.660 (.071) ***	.489 (.079) ***	.570 (.029) ***	-.136 (.111)	.007 (.123)	-.055 (.039)	.516 (.131) ***	.255 (.205)	.805 (.036) ***
Beliefs about Family Importance	.542 (.044) ***	-	.841 (.029) ***	.199 (.070) **	-	.001 (.036)	.500 (.135) ***	.245 (.211)	.807 (.035) ***
Extraversion	.614 (.033) ***	-	.789 (.026) ***	.043 (.057)	-	-.025 (.036)	.524 (.132) ***	.266 (.196)	.807 (.036) ***
Agreeableness	.511 (.046) ***	-	.860 (.027) ***	.125 (.064) *	-	-.064 (.037) †	.512 (.138) ***	.265 (.197)	.805 (.036) ***
Conscientiousness	.643 (.032) ***	-	.766 (.027) ***	-.113 (.053) *	-	.023 (.038)	.508 (.137) ***	.272 (.190)	.809 (.036) ***
Neuroticism	.677 (.027) ***	-	.736 (.025) ***	.041 (.052)	-	-.055 (.042)	.526 (.131) ***	.265 (.196)	.805 (.034) ***
Openness to Experience	.643 (.031) ***	-	.766 (.026) ***	-.025 (.050)	-	-.017 (.035)	.522 (.133) ***	.270 (.193)	.808 (.036) ***
Cognitive Ability	.774		.634	-.040		.100	.516	.278	.803

(.028)	-	(.034)	(.062)	-	(.050)	(.136)	(.187)	(.035)
***		***			*	***		***

Notes: Parameter estimates presented with standard errors in parentheses. Parameters with subscript “₁₁” represent variance in the predictor. Parameters with subscript “₁₂” represent variance in completed fertility shared with the predictor variable. Parameters with subscript “₂₂” represent unique residual variance in completed fertility. Parameters are reported for additive genetic effects (*a*), shared environmental effects (*c*), and nonshared environmental effects (*e*).

† $p < .10$; * $p < .05$; ** $p < .01$; *** $p < .001$

Table 2. Quantitative genetic decomposition of variance in age at first birth

Predictor	a_{11}	c_{11}	e_{11}	a_{12}	c_{12}	e_{12}	a_{22}	c_{22}	e_{22}
Univariate Age at First Birth	-	-	-	-	-	-	.383 (.199) †	.361 (.149) *	.850 (.036) ***
Age at First Marriage	.548 (.147) ***	.295 (.195)	.783 (.042) ***	.446 (.134) **	.223 (.186)	.493 (.050) ***	.000 (.000)	.210 (.051) ***	.681 (.023) ***
Education	.661 (.071) ***	.488 (.079) ***	.571 (.029) ***	.105 (.113)	.279 (.114) *	.090 (.044) *	.381 (.194) *	.212 (.276)	.844 (.036) ***
Beliefs about Family Importance	.543 (.044) ***	-	.840 (.029) ***	-.268 (.057) ***	-	.000 (.033)	.300 (.249)	.347 (.152) *	.847 (.036) ***
Extraversion	.615 (.033) ***	-	.788 (.026) ***	-.114 (.055) *	-	.058 (.038)	.368 (.148) *	.362 (.148) *	.847 (.036) ***
Agreeableness	.509 (.046) ***	-	.861 (.027) ***	-.151 (.068) *	-	.080 (.037) *	.350 (.223)	.363 (.148) *	.847 (.036) ***
Conscientiousness	.644 (.032) ***	-	.765 (.027) ***	.087 (.055)	-	.037 (.040)	.389 (.194) *	.348 (.154) *	.848 (.036) ***
Neuroticism	.676 (.027) ***	-	.737 (.025) ***	-.034 (.047)	-	.000 (.036)	.386 (.197) *	.357 (.152) *	.850 (.036) ***
Openness to Experience	.643 (.031) ***	-	.766 (.026) ***	.064 (.050)	-	.032 (.037)	.383 (.197) †	.356 (.152) *	.849 (.036) ***
Cognitive Ability	.772 (.028) ***	-	.635 (.033) ***	.250 (.050) ***	-	-.149 (.050) **	.301 (.256)	.356 (.151) *	.835 (.036) ***

Notes: Parameter estimates presented with standard errors in parentheses. Parameters with subscript “11” represent variance in the

predictor. Parameters with subscript “₁₂” represent variance in completed fertility shared with the predictor variable. Parameters with subscript “₂₂” represent unique residual variance in completed fertility. Parameters are reported for additive genetic effects (*a*), shared environmental effects (*c*), and nonshared environmental effects (*e*).

† $p < .10$; * $p < .05$; ** $p < .01$; *** $p < .001$

Table 3. Quantitative genetic decomposition of variance in age at first marriage

Predictor	a_{11}	c_{11}	e_{11}	a_{12}	c_{12}	e_{12}	a_{22}	c_{22}	e_{22}
Univariate Age at First Marriage	-	-	-	-	-	-	.538 (.154) ***	.307 (.192)	.785 (.042) ***
Education	.659 (.072) ***	.489 (.079) ***	.571 (.029) ***	.073 (.106)	.151 (.112)	.026 (.039)	.529 (.159) **	.272 (.233)	.786 (.043) ***
Beliefs about Family Importance	.537 (.045) ***	-	.844 (.029) ***	-.182 (.062) **	-	-.036 (.034)	.526 (.153) **	.275 (.211)	.783 (.042) ***
Extraversion	.614 (.033) ***	-	.790 (.025) ***	-.126 (.056) *	-	.056 (.038)	.522 (.159) **	.309 (.190)	.783 (.042) ***
Agreeableness	.507 (.046) ***	-	.862 (.027) ***	-.088 (.070)	-	.047 (.037)	.524 (.159) **	.313 (.188) †	.786 (.042) ***
Conscientiousness	.642 (.032) ***	-	.766 (.027) ***	-.006 (.055)	-	.032 (.037)	.538 (.154) ***	.306 (.192)	.785 (.042) ***
Neuroticism	.676 (.027) ***	-	.737 (.025) ***	.023 (.049)	-	-.045 (.035)	.539 (.153) ***	.305 (.193)	.784 (.042) ***
Openness to Experience	.644 (.031) ***	-	.765 (.026) ***	.078 (.051)	-	.008 (.035)	.533 (.155) **	.305 (.193)	.785 (.042) ***
Cognitive Ability	.776 (.027) ***	-	.631 (.033) ***	.158 (.058) **	-	-.084 (.061)	.516 (.165) **	.307 (.191)	.779 (.041) ***

Notes: Parameter estimates presented with standard errors in parentheses. Parameters with subscript “ $_{11}$ ” represent variance in the predictor. Parameters with subscript “ $_{12}$ ” represent variance in completed fertility shared with the predictor variable. Parameters with subscript “ $_{22}$ ” represent unique residual variance in completed fertility. Parameters are reported for additive genetic effects (a), shared environmental effects (c), and nonshared environmental effects (e).

† $p < .10$; * $p < .05$; ** $p < .01$; *** $p < .001$