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Genotype $\times$ Cohort Interaction on Completed Fertility and Age at First Birth

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Abstract

Microevolutionary projections use empirical estimates of genetic covariation between physical or psychological phenotypes and reproductive success to forecast changes in the population distributions of those phenotypes over time. The validity of these projections depends on relatively consistent heritabilities of fertility-relevant outcomes and consistent genetic covariation between fertility and other physical or psychological phenotypes across generations. However, well-documented, rapidly changing mean trends in the level and timing of fertility may have been accompanied by differences in the genetic mechanisms of fertility. Using a sample of 933 adult twin pairs from the Midlife Development in the United States study, we demonstrate that genetic influences on completed fertility and age at first birth were trivial for the 1920 to 1935 birth cohort, but rose substantially for the 1936 to 1955 birth cohort. For the 1956 to 1970 birth cohort, genetic influences on completed fertility, but not age at first birth, persisted. Because the heritability of fertility is subject to change dynamically with the social context, it is difficult to project selection pressures or the rate at which selection will occur.

Keywords

Fertility; age at first birth; selection; quantitative genetics; second demographic transition

Fertility outcomes are important for the well-being of individuals (Kohler et al., 2005; Nelson et al., 2014), for demographic policy initiatives regarding the global population (Connelly, 2008), and for understanding potential evolutionary selection factors operating in modern populations (Stearns et al., 2010). Microevolution refers to the relatively short-term changes in the distributions of phenotypes in the population across generations due to genetic covariation between physical or psychological phenotypes and reproductive success. A number of recent empirical studies report heritable covariation between reproductive success and other phenotypes, an indication that natural selection may be occurring on those phenotypes (Byars et al., 2010; Kirk et al., 2001; Milot et al., 2011; Pettay et al., 2005; Zietsch et al., 2014). For example, documenting genetic correlations between medical and physiological indices and lifetime reproductive success in a contemporary multigenerational American sample, Byars et al. (2010) projected that natural selection would lead to

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Conflict of Interest

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reductions in total cholesterol, blood pressure, height, and age at first birth accompanied by increases in body weight and age at menopause over the following ten generations. The validity of such projections depends on relatively consistent heritabilities for fertility outcomes and consistent genetic covariation between fertility and the phenotypes under investigation across generations. Yet very few empirical studies have tested this assumption. In contrast, most social-demographic theories of fertility behavior emphasize dramatic cross-generational changes in the determinants of fertility (Cherlin, 2010; Coale & Watkins, 1986; Lesthaeghe, 2010).

We take a biodemographic perspective on fertility that emphasizes the importance of biological and genetic influences, but still seriously considers the dynamics between biology and the social environment (Harden, 2014; Rodgers et al., 2001; Rodgers et al., 2000; Udry, 1996). Integrating social-demographic and biodemographic perspectives on fertility, we hypothesize that, because heritable characteristics influence whether individuals adopt novel fertility practices, historical transitions that introduce innovative fertility practices will lead to a magnification of genetic influences on fertility behavior. This process would imply that the magnitude of microevolutionary selection pressures changes over relatively short time periods. We test these predictions by examining a genotype $\times$ birth cohort interaction on completed fertility and age at first birth using a multi-cohort twin sample.

Changing Demographic Regimes: Ready, Willing, and Able

The level and timing of fertility has changed dramatically over the past century. In the United States, changing fertility practices have resulted in decreased births (Sutton et al., 2011) and increases in age at first birth (Mathews & Hamilton, 2009), age at first marriage (Goodwin et al., 2009), cohabitation (Cherlin, 2010), and the percentage of total fertility due to non-marital fertility (Ventura, 2009). In lay terms, these rapidly changing patterns of fertility and family formation are commonly attributed to the “sexual revolution” of the 1960s. Lesthaeghe (2010; Lesthaeghe & van de Kaa, 1986; van de Kaa, 1987) terms these new patterns the *second demographic transition* (SDT), a name that signifies a conceptual sequel to the decline in fertility that accompanied increases in life expectancy starting in Western, industrialized nations around 1800, known as the *first demographic transition* (Coale & Watkins, 1986; Lee, 2003; Notestein, 1945). Following the SDT, many values associated with fertility behaviors relaxed to allow for nontraditional family formation, childbearing outside of marriage, and greater acceptance of childlessness. Changing values combined with effective contraceptive technology facilitated diverse pathways to family formation.

The SDT marks a decline in the relevance of mechanisms of social control and the rising importance of individual preferences and values (DeLamater, 1981; Lesthaeghe & Surkyn, 2002). Coale (1973) provided the most influential and general¹ model of what leads some individuals to adopt new practices while others retain old fertility patterns. He argued that three conditions are necessary: individuals must be ready, willing, and able to adopt new

¹Similar models of innovative behavior (e.g., Rogers, 1983) are broadly applicable to topics ranging from agriculture (Ryan & Gross, 1943) to technology (Mahajan et al., 1990).

fertility practices. The readiness component reflects the economic cost-benefit analysis of having children or forgoing childbearing. As the cost of support and education rises per child, the costs of having more children outweigh the benefits. Once this balance shifts toward having fewer children, individuals are ready to accept innovative practices that favor lower childbearing. The willingness component refers to the social legitimacy of the new practices. As long as new fertility behaviors are stigmatized or violate cultural taboos, individuals will not be willing to participate in the new fertility regime. As the novel behaviors become more typical, willingness to innovate rises. Finally, the ability component refers to the capacity to consciously control fertility outcomes. If no contraceptive technology existed, it would be impossible for individuals to act on economic or personal preferences. This highlights an important point. For fertility innovation to occur, all three conditions must be jointly met. Economic, psychological, and informational/technical factors supporting innovation must all be present.

Lesthaeghe and Neels (2002) tracked the bottleneck characteristics of the ready, willing, and able model of fertility innovation across Europe. Their analysis of structural and ideational factors suggested that it is the willingness factor, not the ready or able factors, that limits (or facilitates) the spread of demographic innovations. Willingness to adopt new, unusual, and sometimes prohibited practices is important for individual innovation and also for leading broader cultural acceptance. That is, individual differences in willingness are especially predictive of behavior when fertility practices are in a state of flux. Consistent with this idea, personality traits are increasingly important predictors of fertility outcomes for more recent-born twentieth century cohorts (Jokela, 2012; Skirbekk & Blekesaune, 2013).

Genotype × Cohort Interaction

The willingness to adopt new social practices is fundamental to fertility transitions, with the more willing individuals being early adopters and less willing being later adopters. As more individuals adopt novel practices, social norms shift toward the new behaviors, and enforcement of previous social norms diminishes. Udry (1996, p. 328) hypothesized that when individuals' ability to choose between alternative behaviors is maximized, "the more variance in their behavior is controlled by biological forces." Thus, the trends observed during the SDT, such as a reduction of social control over fertility and the rising importance of individual preferences, should lead to an increase in genetic influences on fertility. Individual differences in personality, values, and motivations are genetically influenced (Bouchard & McGue, 2003), as are fertility-relevant phenotypes such as ideal number of children (Miller et al., 2010), fecundity (Rodgers et al., 2003), fertility timing (Kohler et al., 2002; Rodgers et al., 2007), union formation timing (Trumbetta et al., 2007), and the pursuit of competing career goals (e.g., educational attainment; Kohler & Rodgers, 2003; Nisén et al., 2013). Because these genetically influenced, willingness-relevant factors interact with the macro-environmental context, the expression of genetic influences on fertility may change dynamically. When social control wanes and these types of individual characteristics increase in importance, genetic influences would be maximized.

Kohler et al. (1999) provided the first empirical support for Udry's (1996) prediction in regards to fertility. They found a spike in the heritability of completed fertility in Danish

cohorts following the start of the first and second demographic transitions. When fertility practices were relatively stable in the population, family-level environmental influences (which may represent influences such as socioeconomic status and subcultural norms) explained far more variance in fertility than genetic influences. A similar genotype $\times$ cohort interaction has recently been found for Dutch cohorts during the first demographic transition (Bras et al., 2013). Highlighting the dynamic connection between social context and the expression of genetic influences on fertility, this study found that heritability increased to a greater extent for wealthy women living in urban and religiously liberal environments. These women may have had greater freedom to pursue fertility practices in line with their individual preferences compared to women situated in less flexible contexts. Yet these findings have not been widely replicated, and it is unclear whether they generalize beyond the northern European context. Carlson (2003) described the United States as a demographic exception to European trends. Compared to lowest-low fertility found in Europe (e.g., Kohler et al., 2002), the United States has a high fertility rate and is dissimilar in political, religious, economic, and ethnic/racial context. Establishing cohort-related changes in genetic influences on fertility outcomes in the United States would thus be strong evidence for the generalizability of this process.

Based on previous theoretical (Udry, 1996) and empirical (Bras et al., 2013; Kohler et al., 1999) work, we hypothesize that genetic influences on fertility outcomes will increase during demographic transitions. As novel fertility practices become widely adopted and society converges on a new, culturally-approved “fixation point” for behavior, we hypothesize that individual differences in willingness (and associated genetic differences) will decrease in magnitude. Figure 1 presents this hypothesis graphically. In this example, completed fertility in the population fell by one child for birth cohorts from 1920 to 1970. However, some individuals adopt novel fertility behaviors quickly (“early adopters”), whereas others are slow to adopt new practices (“late adopters”) or may never fully adopt new norms (e.g., higher fertility among Mormon individuals; Thornton, 1979). As genotypic differences contribute to whether an individual is an early or late adopter, heritability is maximized *during* the transition period.

In the current project, we track genetic influences on the level and timing of fertility across the SDT. We used the twin sub-sample of the Midlife Development in the United States (MIDUS) sample to estimate genetic influences on total number of children and age at first birth, a phenotype previously linked to evolutionary selection pressures (Byars et al., 2010). Based on the model of fertility innovation, we hypothesized that genetic influences on the level and timing of fertility would be maximally expressed during the SDT compared to before or after. To identify such dynamic trends, we used a variety of parametric and nonparametric analytic strategies based on the classical twin model (Neale & Cardon, 1992) to estimate genetic and environmental influences on completed fertility and age at first birth.

Method

Participants

The data used in the current study was collected as part of the MIDUS study, a two-wave, nationally representative study of health and well-being in adulthood (Ryff et al., 2006). The

first wave of the study took place in 1994 to 1995, and the second wave took place in 2004 to 2006. Most importantly for the current study, the project sampled a wide range of ages at the initial wave (ages 25–74 years) in order to obtain a complete picture of mid-life. The sample includes participants born in cohorts from the 1920s to the 1970s. This makes the sample uniquely suited to study life course trends in response to secular changes in the social context of childbearing. The full sample includes 7108 participants with a relatively even split of males ($n = 3395$) to females ($n = 3632$). Participants varied in terms of the educational background from some grade school ($n = 38$) to professional degrees ($n = 257$). The modal response was a high school degree ($n = 1951$), followed closely by 1–2 years of college ($n = 1302$) and college degree ($n = 1240$). The vast majority of participants identified as White ($n = 5600$), with small numbers identifying as Black ($n = 321$), Native American ($n = 37$), Asian or Pacific Islander ($n = 57$), some other race ($n = 119$), and multiracial ($n = 42$). The twin subsample was composed of monozygotic pairs ($n = 354$), same-sex dizygotic pairs ($n = 335$), and opposite-sex dizygotic pairs ($n = 244$). As described below, we combined information from both waves of data and did not exclude any observations. We track phenotypic secular trends in fertility in the full sample, and we track genetic and environmental contributions to fertility in the twin subsample.

Measures

Level of fertility—At both waves, participants were asked their total number of biological children. For participants that only had data for the initial wave, we used this information. For participants that completed both waves, we used the most recent number of biological children as this represents increases in the number of children born in the interim between waves. Importantly, the level of fertility is potentially censored above for those that have not fully completed their childbearing at the time of the study. This is a relatively small concern for the present analysis because the majority of participants had reached an age at which the majority of fertility is completed in the population at the time of the study. More than 98% of the sample was over 34 years of age. In the United States in 2010, more than 85% of period fertility occurred to individuals under age 34 (Human Fertility Database). More than 85% of participants were over age 41 years, and over 99% of period fertility in the United States in 2010 occurred to individuals under this age.

Timing of fertility—At both waves, participants were asked the birth year of each of their children. The eldest child of each participant was determined, and the age of the participant when the child was born was calculated. For participants who only completed the first wave, we used only that information. For participants who completed both waves, we attempted to construct the most valid estimate for age at first birth. For participants that had no children at the first assessment, but did have children at the second assessment, we used the age reported at the second wave. For participants that reported having children at both waves, we averaged the age at first birth reported across the two waves because recall of ages can be erroneous (Preston et al., 2001, p. 214–223). However, this proved to be a minor problem as the two estimates were highly correlated ($r = .92$). Similar to the level of fertility, age at first birth is censored by the timing of the survey. For participants that have not had any children, it is only known that age at first birth will occur after the survey or never if the participant forgoes childbearing. As described above, the majority of the sample had

completed the major childbearing ages. Following the precedent and recommendations of other researchers (e.g., Kohler et al., 1999), we entered age at first birth as the current age of the participants for childless participants. For childless participants over age 50, it is unlikely that they will have children, and we therefore set their age at first birth to 50 in order to minimize influential outliers.

Analytic Approach

All models were estimated using full-information maximum-likelihood estimation with *Mplus* statistical software (Muthén & Muthén, 1998–2010). To ensure that our quantitative genetic results were not driven by sex differences, as only dizygotic twins can have opposite sexes, we residualized the phenotypes of the effect of sex in the full sample (McGue & Bouchard, 1984). To aid in interpretability, standardized residuals were used for the quantitative genetic analysis. Importantly, this standardization was performed relative to the pooled mean and variance of all cohorts; the results were not standardized in reference to specific birth years. For all behavior genetic analyses, we report raw variance in the outcome, not proportions of variance, such that genetic and environmental contributions at any given birth year do not necessarily sum to 1.0.

Results

Non-parametric Local Structural Equation Modeling (LOSEM)

We hypothesized that genetic influences on fertility would change dynamically across birth cohorts with increases followed by possible decreases over historical time. As it is difficult to make predictions about the specific functional form of this trend, we first applied a non-parametric approach, and then used a variety of parametric approaches as confirmatory steps. We used LOSEM to provide continuous, weighted estimates of model parameters (see Hildebrandt et al., 2009). This approach is similar to kernel regression techniques (Li & Racine, 2007), such as locally weighted regression (i.e., LOESS; Cleveland & Devlin, 1988), but is applied in a structural equation modeling context. Following the recommendations of Hildebrandt et al. (2009, p. 96), we applied a weighting kernel with a bandwidth described by the sample size and standard deviation of the moderator (i.e., $2 * N^{(-1/5)} * SD$). This bandwidth selection is designed to optimally weight the data to minimize and balance bias (i.e., oversmoothing) and variability (i.e., undersmoothing) in the estimates (Li & Racine, 2007). Data closer to a focal birth year received more weight in the analysis. For example, when the focal birth year is 1930, participants born close to 1930 receive much weight in the analysis, but participants born close to 1960 receive little. Moving the focal birth year from 1920 to 1970 generated locally weighted and smoothed estimates. Our moderator (birth year) was not included in the structural component of any LOSEM model, but it was instead incorporated as a weighting variable.

Phenotypic Cohort Trends—To evaluate phenotypic trends, we estimated the mean and variance of the fertility outcomes using 51 models using the full MIDUS sample. The separate models were calculated with the focal value of the weighting variable set to each birth year from 1920 to 1970, and the weighting variable was calculated based on distance from the focal birth year. Figure 2 presents cohort trends in the mean and variance of

number of biological children and age at first birth adjusted for the effect of sex, as well as separately by participant sex. We used LOSEM rather than other non-parametric methods (e.g., LOESS) to maximize the comparability with the following quantitative genetic models.

Participants born more recently had fewer children later in the lifespan. Completed fertility fell from approximately 3 children per individual to 1.5 children per individual over the interval. This trend was similar for males and females. From 1930 to 1960 age at first birth rose by approximately two years, but small counter trends were found for the very early (i.e., 1920s) and very recent (i.e., 1960s) birth cohorts. This trend was similar for males and females, but males tended to have later ages at first birth for all birth cohorts. The identified cohort trends largely match those found across periods in the United States (Mathews & Hamilton, 2009; Sutton et al., 2011). Additionally, variance in both outcomes declined substantially across birth cohorts. Variance in completed fertility fell sharply from 1920–1950 and then plateaued. Variance in age at first birth remained steady until 1950, and then nearly halved in magnitude over the next 20 years.

Quantitative Genetic Cohort Trends—To evaluate trends in genetic and environmental influences, we used a classical univariate quantitative genetic model applied to monozygotic (MZ) and dizygotic (DZ) twins reared together (Neale & Cardon, 1992). This model decomposes variance in an outcome into that due to additive genetic influences (A), shared environmental influences (C; i.e., family-level effects that make siblings living in the same household similar), and nonshared environmental influences (E; i.e., individual-level effects that make siblings living in the same household different, plus measurement error). By definition, twins raised in the same household have identical shared environments. Based on genetic theory, MZ twins are assumed to have identical genetic material, and DZ twins are assumed to share 50% of segregating genetic material on average.

The non-parametric LOSEM approach allowed us to estimate an unrestricted account of dynamic shifts in genetic and environmental influences by recording the locally weighted variance components for each birth year. Importantly, the LOSEM approach still relies on the parametric specification and the associated assumptions of the classical twin model. For example, we assume that an ACE model correctly partitions variance in the phenotypes for all birth cohorts, that MZ and DZ twins receive equal environments, and that there is no assortative mating for the phenotypes. However, the approach is non-parametric in the sense that the parameters of the classical twin model were freely estimated in separate models for each focal birth year.

Figure 3AB summarizes the quantitative genetic LOSEM results. Substantial shared environmental influences, but not genetic influences, were evident until roughly the 1935 birth cohort for both outcomes. Following the 1935 birth cohort, genetic influences rose in importance and shared environmental influences fell in importance. Genetic effects essentially replaced the shared environmental effects. For completed fertility, genetic influences persisted (and shared environmental influences remained negligible) up to the end of data availability. For age at first birth, genetic influences declined and shared environmental influences rose in importance after the 1955 birth cohort. By the end of data

availability, genetic influences on age at first birth were negligible and shared environmental influences were substantial. The influence of the nonshared environment declined across birth cohorts for both outcomes. The nonshared environmental trend resembled the decline in variance observed at the phenotypic level.

An alternative way to visualize the LOSEM results is to plot the predicted relationship between birth cohort and fertility behavior for two theoretical groups of individuals, who score 1 *SD* above and below the mean of the latent genetic factor. These genetic factor scores can be conceptualized as dispositions toward early versus late adoption of fertility practices. As shown in Figure 4, there is no difference between early and late adopters in completed fertility before 1935. Individuals born after 1935 with genetic dispositions for early adoption quickly fall to levels equivalent to the expected endpoint of the population average trend. Individuals with genetic dispositions for late adoption, on the other hand, persist in having larger numbers of children. Differences between early and late adopters in completed fertility are still observed for the 1970 birth cohort. For birth cohorts between 1930 and 1960, early and late adopters differ in age at first birth, but the expected trends converge after 1960.

Continuous Parametric Moderator Models

Standard gene $\times$ environment interaction models (Purcell, 2002) specify that the genetic and environmental effects on a phenotype vary as a linear function of a continuous moderator (which produces quadratic variance estimates across the moderator due to squaring the pathway). Because LOSEM indicated non-linear trends in genetic and environmental effects across birth cohorts (see Figure 3AB), we implemented continuous gene $\times$ environment interaction models using two non-linear functional forms.

First, we used a spline function (Marsh & Cormier, 2002) to fit two connected linear interaction segments across the range of the moderator. To accomplish this approach, we created two variables based on the birth year variable centered at 1920. The first variable indicated birth year from 1920 to 1950, and the second variable indicated additional years beyond 1950. For example, an individual born in 1930 would receive a value of 10 for the first variable and a value of 0 for the second variable, indicating that he/she was born 10 years after 1920. An individual born in 1960 would receive a value of 30 for the first variable and a value of 10 for the second variable. This coding scheme recreates the information of the birth year variable, but allows separate effects to be estimated before and after 1950.

Second, we specified that the genetic and environmental effects on the phenotypes be a quadratic function of the birth year variable centered at 1945. This specification produces variance estimates that are quartic with respect to the moderator and, in contrast to the spline approach, does not rely on the specification of turning points in advance.

In both the spline and quartic approaches, we specified that the genetic and environmental effects on the phenotypes be a linear function of two moderator variables. In the spline approach, the first moderator variable reflects heterogeneity of genetic and environmental influences across birth years 1920 to 1950, and the second moderator variable reflects

heterogeneity of genetic and environmental influences across birth years 1951 to 1970. In the quartic approach, the first moderator variable reflects heterogeneity associated with birth year, and the second moderator variable reflects heterogeneity associated with birth year squared. Each model included the main effect of both moderator variables on the phenotype.

Table 1 reports the parameter estimates for the spline and quartic approach to gene $\times$ environment interaction models. These results are presented graphically in Figure 3CDEF. The results largely confirm the LOSEM results and provide parameter estimates with standard errors. Genetic influences on completed fertility slowly increase across early birth years and then stall. Genetic influences on age at first birth reach a peak for individuals born around 1950 and fall to zero for both earlier and later birth cohorts. In contrast to the LOSEM results, the spline and quartic models for age at first birth imply relatively large genetic influences for very early birth cohorts (i.e., 1920–1930). However, the point expectations in this region are very imprecise (i.e., large standard errors), and are likely due to the parametric constraints of the model, rather than reflecting a “true” spike in heritability for early birth cohorts. Apart from this deviation, results are consistent across analytic approaches.

Multiple Group Models

To complement the approaches that treat birth year continuously, multiple group models were used to determine whether all variance components were necessary to describe variation in the phenotypes and specify alternative models that include dominance genetic influences (D). Based on the LOSEM results, we estimated discrete variance components for different “bins” of birth cohorts. That is, we collapsed individuals born relatively close in chronological time to form discrete groups rather than model birth year continuously. Inspection of the non-parametric results for completed fertility indicated one transition point (roughly 1935), and inspection of the non-parametric results for age at first birth indicated two transition points (roughly 1935 and 1955). To ensure comparability of the results across phenotypes, we created the same groups for both phenotypes: the birth years of 1920–1935, 1936–1955, and 1956–1970. The 1920–1935 cohort was composed of 52 MZ and 95 DZ pairs, the 1936–1955 cohort was composed of 166 MZ and 285 DZ pairs, and the 1956–1970 cohort group was composed of 136 MZ and 194 DZ pairs. For completed fertility, MZ twins correlated at .091 (95% CI = $-.181, .363$), .482 (95% CI = $.364, .600$), and .318 (95% CI = $.169, .467$) for the three cohort groups, and DZ twins correlated at .309 (95% CI = $.123, .495$), .213 (95% CI = $.097, .329$), and .170 (95% CI = $.027, .313$). For age at first birth, MZ twins correlated at .103 (95% CI = $-.167, .373$), .295 (95% CI = $.152, .438$), and .306 (95% CI = $.147, .465$) for the three cohort groups, and DZ twins correlated at .249 (95% CI = $.057, .441$), .152 (95% CI = $.030, .274$), and .372 (95% CI = $.245, .499$).²

²Although monozygotic twin correlations appear lower than dizygotic twin correlations for some cohorts, they are not statistically different from one another. Equating the twin covariance across zygosity does not result in a significant increase in χ^2 for completed fertility for the 1920–1935 birth cohort ($\chi^2 = 1.921$, $df = 1$, $p = .17$), age at first birth for the 1920–1935 birth cohort ($\chi^2 = .782$, $df = 1$, $p = .38$), or age at first birth for the 1956–1970 birth cohort ($\chi^2 = .670$, $df = 1$, $p = .41$). Additionally, constraining the mean, variance, and covariance to be equal across zygosity resulted in no statistically significant loss of model fit in these groups. This is consistent with no genetic influences on fertility for certain birth cohorts, as theoretically predicted.

Table 2 presents the model fit statistics for all multiple group models tested. Model fit was excellent across all models. Table 3 presents the point estimates of genetic and environmental influences on completed fertility and age at first birth for discrete cohorts. We first estimated ACE pathways for each cohort group separately. Mirroring the continuous approaches, shared environmental effects were not found for completed fertility for the second or third group and were not found for age at first birth for the second group. Next, we replaced these nonsignificant shared environmental parameters with dominance genetic parameters. None of these parameters reached statistical significance, and all additive genetic parameters that were significant in the baseline model remained significant. Finally, we fitted a trimmed model in which pathways that were not significant in any model were constrained to zero. This model was preferred based on the model fit statistics reported in Table 1. Thus, there was no evidence of genetic influences on either phenotype for the 1920–1935 birth cohort. For age at first birth, there was no evidence of genetic influences for the 1956–1970 birth cohort. Additive genetic influences, but not dominance genetic influences, were detected for the remaining birth cohorts.

Genetic and Environmental Correlation between Phenotypes

To evaluate genetic and environmental correlation between completed fertility and age at first birth, we used a correlated factors model for the identified discrete cohort groups (Neale & Cardon, 1992). This multivariate quantitative genetic model estimates the correlation between the genetic and environmental influences on two outcomes. For the 1920–1935 and 1956–1970 cohorts, we found no evidence of genetic correlation between completed fertility and age at first birth due to minimal genetic influences on one or both outcomes. However, we found statistically significant, negative genetic correlation for the 1936–1955 cohort ($-.990$, 95% CI = -1.333 , $-.647$). This indicates that the same set of genetic factors that disposed individuals toward higher completed fertility in this cohort also disposed individuals toward earlier age at first birth. We found no statistically significant shared environmental correlation for any cohort group. We found statistically significant, negative nonshared environmental correlation for the 1920–1935 ($-.646$, 95% CI = $-.768$, $-.524$), 1936–1955 ($-.585$, 95% CI = $-.675$, $-.495$), and 1956–1970 ($-.681$, 95% CI = $-.767$, $-.595$) cohort groups. This indicates that, after taking into account genetic and shared environmental influences on fertility, individuals that have a later age at first birth tend to have fewer children, and this effect appears to be general across different social contexts.

Sensitivity Analyses

We performed two sensitivity analyses. First, we evaluated whether participant sex moderated the reported trends, but these estimates tended to be imprecise due to low power. Second, we evaluated the robustness of the reported interaction by fitting the quartic continuous moderator model to both phenotypes only for cohort groups that displayed significant genetic influence. This included participants born between 1936 to 1970 for completed fertility and 1936 to 1955 for age at first birth. Results from these models found significant birth year trends in genetic influence on both phenotypes across this limited span. The pattern of results largely matched that found in the full sample.

Discussion

Genetic influences on fertility outcomes are dynamic. Our results indicate that the heritability of fertility in the United States changed fluidly with the emergence of novel social norms. As old social norms begin to be replaced by new norms, individuals have greater choice in their fertility outcomes: they can continue with the status quo, or they can adopt the newer social tides. Because such behaviors are likely to be affected by genetically influenced dispositions, genetic influences are predicted to increase during such periods of social change (Bras et al., 2013; Kohler et al., 1999). Consistent with these predictions, our results indicate that in the early years of the SDT, genetically influenced dispositions led some individuals to continue with conventional patterns of family formation, while genetically influenced dispositions led other individuals to express non-normative or progressive fertility behaviors.

Projecting evolutionary selection forces into the future will need to consider the possibility of social changes that either constrain or facilitate the expression of genetically influenced mechanisms of fertility. When societal forces constrain expression, it would be expected that the rate of evolution would slow, but during transitions that maximize heritability, the rate of evolution may increase dramatically. For example, we found essentially no genetic correlation between age at first birth and completed fertility before or after the SDT, but during the SDT, the genetic correlation was nearly perfect. Further, Zietsch et al. (2014) cautioned against the use of pedigrees (e.g., parent-child correlations; Byars et al., 2010) to estimate heritability because such approaches confound estimates of genetic and shared environmental effects. Similarly, we find that the relative importance of genetic and shared environmental effects change across cohorts in tandem with societal change. This process would further obscure evolutionary projections unless quantitative genetic methods (e.g., twin approaches) are employed to estimate possible changes in heritability and genetic correlation.

Apart from Kohler et al. (1999), few previous studies have examined the interactive nature of genetic effects and environmental contexts by leveraging changing social forces through chronological time. One notable exception is a study by Heath et al. (1985) that found increases in the heritability of educational attainment following increased social access to education. In other words, genetic influences were maximized when individuals had greater freedom to make choices in line with their interests, rather than family background. With these results in mind, researchers interested in social forces can examine changing heritabilities across cohorts to evaluate the dynamic interaction between individual preferences and social control.

The current study has several important strengths and limitations. We used a diverse, genetically informative, multi-cohort sample to replicate previous findings (Bras et al., 2013; Kohler et al., 1999) in a substantially different environmental context. The results of the current study are similar to those previously reported, strengthening the generalizability of this effect. Further, our results are conceptually supported by findings from recent studies indicating that heritable personality traits (Bouchard & McGue, 2003) are increasingly associated with fertility outcomes for more recent birth cohorts (Jokela, 2012; Skirbekk &

Blekesaune, 2013). A strength of quantitative genetic studies is that it is not necessary to test the relation between a circumscribed trait and fertility. Rather, the current study indexed all genetically influenced individual differences that were relevant to fertility. We did not, however, examine specific avenues for genetic influence on fertility, such as preferences, motivation, pursuing educational attainment, or fecundity.

A further limitation is that we were unable to conclusively test whether the identified genetic and environmental trends differed for males and females. Despite the relatively large twin subsample of the MIDUS dataset, data coverage was limited when broken down both by specific birth years and participant sex. Sex differences in cohort effects in heritability and genetic covariation would be very interesting to examine in future research, as these may serve as mechanisms of antagonistic pleiotropy.

One may wonder whether the observed results were primarily driven by monozygotic correlations slightly lower than dizygotic correlations for certain cohort groups. Because the majority of the increase in heritability occurs *during* the second demographic transition (i.e., 1936 to 1955), significant birth year trends in genetic influence were still detected on both phenotypes when cohort groups with lower monozygotic compared to dizygotic correlations were excluded. Further, we expected zero heritability for some cohort groups based on previous theory (Udry, 1996; Figure 1) and empirical evidence (Kohler et al., 1999), and consistent with this expectation, we observed statistically equivalent monozygotic and dizygotic correlations.

Some caution should be used when interpreting the cohort trends for the most recent cohorts as some members may not have fully completed their fertility. Indeed, research indicates that more recent cohorts increasingly delayed fertility (Billari et al., 2007) and pursued educational attainment (Snyder & Dillow, 2013). Participants with censored fertility histories in this study may therefore have been more highly educated than those with complete fertility histories. As pursuit of education may partially mediate genetic influences on fertility (Kohler & Rodgers, 2003; Nisén et al., 2013), because of censoring, we may have failed to detect some genetic influences on fertility for recent cohorts. Fortunately, this is a relatively small concern considering the age of the participants at the time of the most recent survey (roughly 35 years old) and that the major finding of the study was an increase in genetic influences across cohorts that had certainly completed their fertility (i.e., birth years from 1920 to 1950). Additionally, censoring is unlikely to affect the results for age at first birth because this phenotype is a discrete event that typically occurs by the late-20's even within the current population (Mathews & Hamilton, 2009).

An important and still unaddressed question is the extent to which genetic influences on completed fertility will persist. Our results indicate that genetic influences on age at first birth gradually rose and faded across progressively more recent cohorts. This is consistent with a societal shift to a novel equilibrium point and norm for age at first birth. Our results do not indicate a similar decrease in the heritability of completed fertility following the SDT. One interpretation would be that society has moved more slowly to reach a stable equilibrium in regards to family size preferences, but society will eventually reach one as late adopters slowly conform over time (Lesthaeghe, 2010). A second possibility is that a

stable equilibrium may not be reached because of persistent clustering of individuals around different fertility norms that may, in part, be due to genotypic differences (Bishop, 2009). A third possibility is that post SDT fertility norms are weak, with much individual choice allowed and encouraged. Opportunities to distinguish between these alternatives will emerge as researchers continue to track patterns of fertility further into the 21st century – and continue to conduct integrative research that considers both genetic differences and social contexts.

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References

- Billari FC, Kohler H-P, Andersson G, Lundström H. Approaching the limit: Long-term trends in late and very late fertility. *Pop Dev Rev.* 2007; 33(1):149–170.
- Bishop, B. *The Big Sort: Why the Clustering of Like-Minded America is Tearing us Apart.* New York, NY: Houghton Mifflin; 2009.
- Bouchard TJ Jr, McGue M. Genetic and environmental influences on human psychological differences. *Dev Neurobio.* 2003; 54:4–45.
- Bras H, Van Bavel J, Mandemakers K. Unraveling the intergenerational transmission of fertility: Genetic and shared-environment effects during the demographic transition in the Netherlands, 1810–1910. *Hist Fam.* 2013; 18(2):116–134.
- Byars SG, Ewbank D, Govindaraju DR, Stearns SC. Natural selection in a contemporary human population. *Proc Natl Acad Sci USA.* 2010; 107(suppl 1):1787–1792. [PubMed: 19858476]
- Carlson, AC. *The fertility gap: Recrafting American population policy for a depopulating world.* Washington, DC: Family Research Council; 2003.
- Cherlin AJ. Demographic trends in the United States: A review of research in the 2000s. *J Marriage Fam.* 2010; 72:403–419. [PubMed: 22399825]
- Cleveland WS, Devlin SJ. Locally-weighted regression: An approach to regression analysis by local fitting. *J Am Stat Assoc.* 1988; 83(403):596–610.
- Coale, AJ. The demographic transition reconsidered. In: *International Union for the Scientific Study of Population.*, editor. *Proceedings of the International Population Conference 1973.* Liège: Editions Ordina; 1973. p. 53-73.
- Coale, AJ.; Watkins, SC. *The decline of fertility in Europe: The revised proceedings of a conference on the Princeton European Fertility Project.* Princeton: Princeton University Press; 1986.
- Connelly, M. *Fatal misconception: The struggle to control world population.* Cambridge, MA: Harvard University Press; 2008.
- DeLamater J. The social control of sexuality. *Annu Rev Sociol.* 1981; 7:263–290. [PubMed: 12312456]
- Goodwin P, McGill B, Chandra A. Who marries and when? Age at first marriage in the United States: 2002. *NCHS Data Brief.* 2009; (19)
- Harden KP. Genetic influences on adolescent sexual behavior: Why genes matter for environmentally oriented researchers. *Psychol Bull.* 2014; 140(2):434–465. [PubMed: 23855958]
- Heath AC, Berg K, Eaves LJ, Solaas MH, Corey LA, Sundet J, Magnus P, Nance WE. Education policy and the heritability of educational attainment. *Nature.* 1985; 314:734–736. [PubMed: 4039415]
- Hildebrandt A, Wilhelm O, Robitzsch A. Complementary and competing factor analytic approaches for the investigation of measurement invariance. *Rev Psychol.* 2009; 16(2):87–102.

- 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)
- Jokela M. Birth-cohort effects in the association between personality and fertility. *Psychol Sci.* 2012; 23(8):835–841. [PubMed: 22722269]
- Kirk KM, Blomberg S, Duffy DL, Heath AC, Owens IPF, Martin NG. Natural selection and quantitative genetics of life-history traits in western women: A twin study. *Evol.* 2001; 55(2):423–435.
- Kohler H-P, Behrman JR, Skytthe A. Partner + children = happiness? The effects of partnerships and fertility on well-being. *Pop Dev Rev.* 2005; 31(3):407–445.
- Kohler H-P, Billari FC, Ortega JA. The emergence of lowest-low fertility in Europe during the 1990s. *Popul Dev Rev.* 2002; 28:641–680.
- Kohler, H-P.; Rodgers, JL. Education, fertility, and heritability: Explaining a paradox. In: Wachter, KW.; Bulatao, RA., editors. *Offspring: Human Fertility Behavior in Biodemographic Perspective*. National Academies Press; Washington D.C.: 2003. p. 46-90.
- Kohler H-P, Rodgers JL, Christensen K. Is fertility behavior in our genes? Findings from a Danish twin study. *Popul Dev Rev.* 1999; 25(2):253–288.
- Kohler H-P, Rodgers JL, Christensen K. Between nature and nurture: The shifting determinants of female fertility in Danish twin cohorts. *Biodemography Soc Biol.* 2002; 49(3–4):218–248.
- Lee R. The demographic transition: Three centuries of fundamental change. *The Journal of Economic Perspectives.* 2003; 17:167–190.
- Lesthaeghe R. The unfolding story of the second demographic transition. *Popul Dev Rev.* 2010; 36(2): 211–251. [PubMed: 20734551]
- Lesthaeghe R, Neels K. From the first to the second demographic transition: An interpretation of demographic innovation in France, Belgium, and Switzerland. *Euro J Popul.* 2002; 18(4):325–360.
- Lesthaeghe R, Surkyn JR. Value orientations and the second demographic transition (SDT) in Northern, Western and Southern Europe: An update. *Demogr Res.* 2002; 3:45–86.
- Lesthaeghe, R.; van de Kaa, DJ. Twee demografische transitie's?. In: Lesthaeghe, RJ.; van de Kaa, DJ., editors. *Bevolking: Groei en Krimp. Mens en Maatschappij*. VanLoghum-Sluterus; Deventer: 1986. p. 9-24.
- Li, Q.; Racine, J. *Nonparametric econometrics: Theory and practice*. Princeton University Press; 2007.
- Mahajan V, Muller E, Srivastava RK. Determination of adopter categories by using innovation diffusion models. *J Mark Res.* 1990; 27:37–50.
- Marsh, LC.; Cormier, DR. *Spline regression models*. Thousand Oaks, CA: Sage Publications; 2002.
- Mathews TJ, Hamilton BE. Delayed childbearing: More women are having their first child later in life. *NCHS Data Brief.* 2009; (21)
- McGue M, Bouchard TJ Jr. Adjustment of twin data for the effects of age and sex. *Behav Genet.* 1984; 14(4):325–343. [PubMed: 6542356]
- Miller WB, Bard DE, Pasta DJ, Rodgers JL. Biodemographic modeling of the links between fertility motivation and fertility outcomes in the NLSY79. *Demography.* 2010; 47(2):393–414. [PubMed: 20608103]
- Milot E, Mayer FM, Nussey DH, Boisvert M, Pelletier F, Réale D. Evidence for evolution in response to natural selection in a contemporary human population. *Proc Natl Acad Sci USA.* 2011; 108(41): 17040–17045. [PubMed: 21969551]
- Muthén, LK.; Muthén, BO. *Mplus user's guide*. 6. Los Angeles, CA: Muthén and Muthén; 1998–2010.
- Neale, MC.; Cardon, LR. *Methodology for genetic studies of twins and families*. Dordrecht: Kluwer Academic Press; 1992.
- Nelson, SK.; Kushlev, K.; Lyubomirsky, S. The pains and pleasures of parenting: When, why, and how is parenthood associated with more or less well-being?. *Psychol Bull.* 2014 Feb 3. Advanced online publication. <http://dx.doi.org/10.1031/a0035444>
- Nísen J, Martikainen P, Kaprio J, Silventoinen K. Educational differences in completed fertility: A behavioral genetic study of Finnish male and female twins. *Demography.* 2013; 50(4):1399–1420. [PubMed: 23344794]

- Notestein, F. Population: The long view. In: Schultz, T., editor. Food for the world. Chicago, IL: University of Chicago Press; 1945. p. 36-57.
- Pettay JE, Kruuk LEB, Jokela J, Lummaa V. Heritability and genetic constraints of life-history trait evolution in preindustrial humans. *Proc Natl Acad Sci USA*. 2005; 102(8):2838–2843. [PubMed: 15701704]
- Preston, SH.; Heuveline, P.; Guillot, M. Demography: Measuring and Modeling Population Processes. Malden, MA: Blackwell Publishers; 2001.
- Purcell S. Variance component estimates for gene-environment interaction in twin analysis. *Twin Res*. 2002; 5(6):554–571. [PubMed: 12573187]
- Rodgers JL, Hughes K, Kohler H-P, Christensen K, Doughty D, Rowe DC, Miller WB. Genetic influence helps explain variation in human fertility: Evidence from recent behavioral and molecular genetic studies. *Curr Dir Psychol Sci*. 2001; 10(5):184–188.
- Rodgers JL, Bard DE, Miller WB. Multivariate cholesky models of human female fertility patterns in the NLSY. *Behav Genet*. 2007; 37(2):345–361. [PubMed: 17205393]
- Rodgers, JL.; Kohler, H-P.; Christensen, K. Genetic variance in human fertility: Biology, psychology, or both?. In: Rodgers, JL.; Kohler, H-P., editors. The Biodemography of Human Reproduction and Fertility. Kluwer; Boston: 2003. p. 229-250.
- Rodgers, JL.; Rowe, DC.; Miller, WB., editors. Genetic influences on human fertility and sexuality: Theoretical and empirical contributions from the biological and behavioral sciences. Kluwer; Boston: 2000.
- Rogers, EM. Diffusion of innovations. New York: The Free Press; 1983.
- Ryan B, Gross NC. The diffusion of hybrid seed corn in two Iowa communities. *Rural Sociol*. 1943; 8:15–24.
- Ryff, C.; Almeida, DM.; Ayanian, JS.; Carr, DS.; Cleary, PD.; Coe, C.; Williams, D. Midlife development in the United States (MIDUS II), 2004–2006. Madison, WI: University of Wisconsin, Survey Center; 2006.
- Skirbekk V, Blekesaune M. Personality traits increasingly important for male fertility: Evidence from Norway. *Eur J Pers*. 2013 Aug 5. Advanced online publication. 10.1002/per.1936
- Snyder TD, Dillow SA. Digest of education statistics, 2012. NCES 2014-015. 2013
- Stearns SC, Byars SG, Govindaraju DR, Ewbank D. Measuring selection in contemporary human populations. *Nat Rev Genet*. 2010; 11(9):611–622. [PubMed: 20680024]
- Sutton PD, Hamilton BE, Mathews TJ. Recent decline in births in the United States 2007–2009. *NCHS Data Brief*. 2011; (60)
- Thornton A. Religion and fertility: The case of Mormonism. *J Marriage Fam*. 1979; 41(1):131–142.
- Trumbetta SL, Markowitz EM, Gottesman II. Marriage and genetic variation across the lifespan: Not a steady relationship? *Behav Genet*. 2007; 37(2):362–375. [PubMed: 17165140]
- Udry JR. Biosocial models of low-fertility societies. *Pop Dev Rev*. 1996; 22:325–336.
- van de Kaa DJ. Europe's second demographic transition. *Popul Bull*. 1987; 42:1–57. [PubMed: 12268395]
- Ventura SJ. Changing patterns of nonmarital childbearing in the United States. *NCHS Data Brief*. 2009; (18)
- Zietsch BP, Kuja-Halkola R, Walum H, Verweij KJH. Perfect genetic correlation between number of offspring and grandoffspring in an industrialized human population. *Proc Natl Acad Sci USA*. 2014; 111(3):1032–1036. [PubMed: 24395780]

Figure 1.

Hypothetical decline in completed fertility from birth cohorts of 1920 to 1970 where fertility falls along a logistic curve. **A.** Expected completed fertility for the population average, late fertility adopters, and early fertility adopters. **B.** Area between the late and early adopter curve, which could potentially be related to genotypic differences between individuals.

Figure 2.

Cohort trends in full MIDUS sample. **A.** Mean and variance of completed fertility, sex adjusted. **B.** Mean and variance of age at first birth, sex adjusted. **C.** Mean and variance of completed fertility, males. **D.** Mean and variance of age at first birth, males. **E.** Mean and variance of completed fertility, females. **F.** Mean and variance of age at first birth, females.

Figure 3.

Quantitative genetic decomposition of fertility outcomes in the twin subsample of MIDUS. Unstandardized genetic and environmental influences are presented for **A.** completed fertility using LOSEM, **B.** age at first birth using LOSEM, **C.** completed fertility as a spline function of birth year, **D.** age at first birth as a spline function of birth year, **E.** completed fertility as a quartic function of birth year, and **F.** age at first birth as a quartic function of birth year. Squared ACE pathway expectations are presented.

Figure 4.

Expected sex adjusted phenotypic cohort trends based on twin subsample results assuming a population average with subgroups possessing genetic dispositions for early and late adoption of novel fertility behaviors. **A.** Phenotypic trends in completed fertility for the population average, a subgroup with genetic dispositions for late adoption (i.e., +1 SD genetic effect), and a subgroup with genetic dispositions for early adoption (i.e., -1 SD genetic effect). **B.** Phenotypic trends in age at first birth for the population average, a subgroup with genetic dispositions for late adoption (i.e., -1 SD genetic effect), and a subgroup with genetic dispositions for early adoption (i.e., +1 SD genetic effect).

Table 1
Parameter estimates for parametric behavior genetic decomposition of completed fertility and age at first birth

	Completed Fertility		Age at First Birth	
	Spline	Quartic	Spline	Quartic
<i>a</i>	.350 (.424)	.498 (.105) ***	.577 (.334) †	.606 (.077) ***
<i>a'</i>	-.032 (.014) *	.016 (.009) †	-.043 (.014) **	.022 (.005) ***
<i>a''</i>	.010 (.007)	-.001 (.00046) *	.033 (.009) ***	-.002 (.00025) ***
<i>C</i>	.889 (.168) ***	.269 (.143) †	.773 (.194) ***	.069 (.172)
<i>c'</i>	-.025 (.011) *	-.020 (.007) **	-.036 (.011) **	.021 (.005) ***
<i>c''</i>	-.004 (.017)	.000 (.000)	-.005 (.012)	.000 (.000)
<i>e</i>	1.124 (.079) ***	.761 (.034) ***	.914 (.080) ***	.913 (.038) ***
<i>e'</i>	-.014 (.003) ***	-.010 (.002) ***	.001 (.004)	-.006 (.002) **
<i>e''</i>	-.002 (.004)	.000 (.000)	-.024 (.004) ***	-.001 (.00025) ***

Notes. Standard errors reported in parentheses. In the spline specification, *a*, *c*, and *e* parameters refer to the estimate at 1920, the *a'*, *c'*, and *e'* parameters refer to the effect of the first spline variable (i.e., years before 1950), and the *a''*, *c''*, and *e''* parameters refer to the effect of the second spline variable (i.e., years after 1950). In the quartic specification, the *a*, *c*, and *e* parameters refer to the estimate at 1945, the *a'*, *c'*, and *e'* parameters refer to the effect of birth year, and the *a''*, *c''*, and *e''* parameters refer to the effect of birth year squared.

†
p < .10
*
p < .05
**
p < .01

p < .001

Table 2
Model fit statistics for multiple group behavior genetic decomposition of completed fertility and age at first birth

Panel A. Completed Fertility						
	χ^2	df	p	χ^2	df	p
1. ACE	24.080	18	.1524	4830.250	4888.246	
2. ADE	24.058	18	.1531	4830.229	4888.225	
3. Trimmed	24.084	21	.2890	.004	3	.999
				4824.254	2867.751	

Panel B. Age at First Birth						
	χ^2	df	p	χ^2	df	p
1. ACE	10.031	18	.9309	4958.359	5016.356	
2. ADE	10.022	18	.9312	4958.350	5016.346	
3. Trimmed	10.031	21	.9785	.000	3	1.000
				4952.359	4995.857	

Note. χ^2 comparisons are based on reference to the ACE model.

Table 3
Parameter estimates for multiple group behavior genetic decomposition of completed fertility and age at first birth

Panel A. Completed Fertility									
1920 to 1935 Cohort			1936 to 1955 Cohort			1956 to 1970 Cohort			
A path	C path	E path	A path	C/D path	E path	A path	C path	D path	E path
1. ACE	.000 (.445)	.574 (.107) ***	1.051 (.063) ***	.630 (.119) ***	.088 (.678) ***	.483 (.053) ***	.001 (.576) ***		.663 (.035) ***
2. ADE	.000 (.447)	.574 (.107) ***	1.052 (.063) ***	.637 (.045) ***	.003 (1.365) ***	.449 (.217) *	.187 (.568) ***		.660 (.039) ***
3. Trimmed	–	.574 (.107) ***	1.052 (.063) ***	.637 (.045) ***	–	.483 (.053) ***	–		.663 (.035) ***

Panel B. Age at First Birth									
1920 to 1935 Cohort			1936 to 1955 Cohort			1956 to 1970 Cohort			
A path	C path	E path	A path	C/D path	E path	A path	C path	D path	E path
1. ACE	.000 (.474)	.470 (.100) ***	.941 (.056) ***	.592 (.066) ***	.000 (.895) ***	.000 (.476) ***	.497 (.044) ***		.686 (.028) ***
2. ADE	.000 (.475)	.470 (.100) ***	.941 (.056) ***	.568 (.256) *	.178 (.911) ***	.000 (.475) ***	.497 (.044) ***		.686 (.028) ***
3. Trimmed	–	.470 (.100) ***	.941 (.056) ***	.592 (.066) ***	–	–	.497 (.044) ***		.686 (.028) ***

Note. In the ADE model, significant C effects were retained (e.g., for 1920 to 1935 birth cohort) Standard errors reported in parentheses.

*
p < .05

**
p < .01

p < .001