Genome-Wide Heritability Estimates for Family Life Course Complexity

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Abstract

Sequence analysis is an established method to study the complexity of family life courses. While both individual and societal characteristics have been linked with the complexity of family trajectories, sociologists have neglected the potential role of genetic factors for family transitions and events across the life course. We apply retrospective life history and molecular genetic data from the US Health and Retirement Study (HRS) to genome-wide complex trait analysis (GCTA). Specifically, we estimate the genetic contribution to sequence complexity and a wide range of family demographic behaviors using genomic-relatedness-based restricted maximum-likelihood (GREML) models. This innovative methodological approach allows us to estimate the heritability of a composite life course outcome – sequence complexity – for the first time. Not only do we demonstrate that a number of family demographic indicators, such as the age at first birth and first marriage as well as the number of children, marriages and divorces, are heritable, but also provide evidence that composite life course metrics can also be influenced by genetic factors. For example, our results show that 12 percent of the total variation in the complexity of differentiated family sequences is attributable to genetic influences. Interestingly, we find little evidence that the heritability of family life course complexity or any other family demographic outcomes has increased across US birth cohorts. Therefore, our results do not substantiate claims that lower normative constraints on family demographic behavior increases the role of genetic predispositions.

Keywords: *Family, Biodemography, Sequence Analysis, Heritability, Life Course, GREML*

Introduction

Scholars and the general public alike perceive modern life courses to be more complex, unstable, and unpredictable than in the early and mid-20th century (Walsh 2012; Beck 1994, 2009; Sennet 2006). Common narratives in family sociology and demography surrounding more complex family trajectories revolve around the decline of the modern nuclear family and pluralization of family forms (Bengtson 2001). Indeed, the United States experienced a decrease and postponement in marriage and marital fertility that coincided with an increase in nonmarital cohabitation and fertility as well as divorce and remarriage (Cherlin 2010). This increase in the number of family events and states that individuals experience over the course of their lives result in more complex family trajectories (Brückner and Mayer 2005). Increasing family life course complexity may have serious consequences for individuals and societies. For example, complexity generated by early nonmarital parenthood, serial cohabitation, and divorce is likely tightly intertwined with the production of social inequalities and their reproduction across generations (e.g., McLanahan and Percheski 2008).

Three meta-theoretical narratives have been applied when studying the complexity of family events and transitions within individual life courses (see Van Winkle 2018). First, the Second Demographic Transition (SDT) thesis is an ideational account, which associates more complex family life courses with a shift from materialist to post-materialist values (Van de Kaa 1987, 2001, Lesthaeghe 1995, 2010, 2014). Second, an increase in family life course complexity has been connected with increasing economic uncertainty following globalization and deindustrialization (Mills and Blossfeld 2005, 2003, 2013). Third, life course sociologists and welfare state scholars argue that labor market and family policies are related to the complexity of family lives (Esping-Andersen 1990, 1999, Mayer 1997, 2004, 2009). The first two theoretical perspectives are generally invoked to account for change over time, while the latter is more commonly used to account for cross-national differences.

However a fourth theoretical perspective on family demographic behavior is currently emerging: biodemography or genodemography (H. P. Kohler et al. 2006; Mills and Tropf 2015; Conley 2016). This approach concentrates on genetic factors that influence the components of family life course complexity. While the meta-theoretical perspectives discussed above are the most prominent in family demography and sociology, biodemographic research has gained importance in the last decades (see D'Onofrio and Lahey 2010 for a review; see Conley and Fletcher 2017 for an

introduction to socio-genomic research). Biodemography is the study of family demographic behavior that incorporates both economic and sociological theories with approaches from biology, especially behavioral and molecular genetics (H. P. Kohler et al. 2006). The first step in biodemographic research is the estimation of heritability, i.e. the proportion of variance that is attributed to genetic factors as opposed to environmental factors.

In this study, we address two research questions: First, to what extent is the complexity of family life course complexity heritable? The strength of heritability gives researchers insight on the potential importance of sociobiological explanations and the consequence of ignoring them. Discounting biological and genetic accounts may lead to grossly biased results, especially if genetic factors are confounded with associations between social factors and life course complexity (D'Onofrio and Lahey 2010). Second, has the heritability of family life course complexity changed across US birth cohorts? It has been argued in family demography that genetic influences on family demographic behavior will increase in societies with low levels of social constraints (Udry 1996; H. P. Kohler et al. 2006) because genetic predispositions can express themselves to a greater extent in contexts where variation in individual behavior is high and is less restricted by formal and informal social norms.

We apply retrospective life history and molecular genetic data from the US Health and Retirement Study (HRS) to a genome-wide complex trait analysis (GCTA). Specifically, we first reconstruct the family life courses of HRS respondents as sequences. A nuanced metric developed in sequences analysis, the sequence complexity index, is calculated for each individual trajectory (Gabadinho et al. 2010). The sequence complexity index does not just incorporate the number of life course transitions into account, but also the unpredictability across individual trajectories. Second, we estimate the heritability of sequence complexity using genomic-relatedness-based restricted maximum-likelihood (GREML) models. Most heritability estimates for family demographic behavior have used ACE decomposition models that compare monozygotic and dizygotic twins (see Boomsma, Busjahn, and Peltonen 2002 for heritability estimation with ACE models). However more precise heritability estimates can be determined using GCTA-GREML, which use genetic similarity to decompose trait variance into additive genetic and environmental components (Domingue, Wedow, et al. 2016; Yang et al. 2011, 2010).

Theoretical Background

Family Demographic Trends in 20th Century United States

In the following discussion, we briefly sketch trends in the occurrence of life course states and transitions that comprise family life course complexity: leaving the parental home, forming and dissolving unions, and entering parenthood. The transition to adulthood and family formation often begins with leaving the parental home. The age at which individuals transition out of the parental household declined in the early and mid-20th century, but has recently began to increase in the United States (Goldscheider 1997; White 1994). For example, the percentage of US women age 18-24 living in the parental home increased from 34.9 % in 1960 to 49.2 % by 2010.[1] A similar trend can be observed for men, although men tend to live in the parental home longer than women. It has become more common to return to the parental home after initially leaving, especially when experiencing economic hardship (Mitchell 2006; Stone, Berrington, and Falkingham 2014). In the United States, 36 % of young adults born 1980-1984 returned to the parental home at least once by age 27 (Sandberg-Thoma, Snyder, and Jang 2015). For more recent birth cohorts, individuals often form independent households or live in cohabiting unions after leaving the parental home rather than marrying (Evans 2013; Zorlu and Mulder 2011).

Destinations after leaving the parental home have become more diverse. For the first half of the 20th century, leaving the parental home and entering marriage were tightly coupled life course transitions (Buchmann and Kriesi 2011). In fact, only 5 % of unmarried US men and women lived outside the parental home in the early 20th century (Rosenfeld 2007). Although the median age of first marriage has increased in the United States, the median age of first union has remained constant (Manning, Brown, and Payne 2014). The stability in the age at first union is largely attributable to higher rates of cohabitation offsetting lower rates of marriage (Perelli-Harris and Lyons-Amos 2015; Kiernan 2001; Smock 2000; Goldstein and Kenney 2001). The percent of the US population living in non-marital cohabitation increased from 0.6 % in 1960 to 6 % in 2010, while the percentage living in a marital union decreased from 69 % to 54 % for men and 65 % to

[1] U.S. Census Bureau, Current Population Survey, March and Annual Social and Economic Supplements. U. S. Bureau of the Census, 1980 Census of Population, PC80-2-4B, "Persons by Family Characteristics," table 4. 1970 Census of Population, PC(2)-4B, table 2. 1960 Census of Population, PC(2)-4B, table 2.

52 % for women.[2] The median age of first marriage in 1960 was 22 for men and 20 for women, which increased to age 28 for men and 26 for women by 2010.[2]

One reason for the decrease in the crude marriage rate can be found in the increase in divorce. Between 1960 and 1980 , the crude divorce rate in the United States more than doubled from 2.2 to 5.2 (Amato 2010). It has since decreased to 3.6 divorces per 1,000 marriages by 2006. The US doesn't just exhibit high rates of marital dissolution, but cohabiting unions in the US are often short and likely to end in separation (Smock 2000). However, cohabitation is not just a life course state for young adults. Higher rates of marital separation have not only increased remarriage rates, but also rates of cohabitation later in adulthood, often as an alternative to remarriage (Coleman, Ganong, and Fine 2000; Xu, Bartkowski, and Dalton 2011).

The decline of fertility in the United States to near-replacement rates has concerned both scholars and policymakers (Balbo, Billari, and Mills 2013). Lower total fertility rates may result from delayed entry into parenthood, the tempo effect, as well as smaller family sizes and higher rates of childlessness, the quantum effect (Morgan and Taylor 2006; Zeman et al. 2018). Women's median age at first birth rose from age 23 to 25 between the 1940s and 1980s birth cohorts (Finer and Philbin 2014). Completed fertility is comparatively high in the United States: between 2.0 to 2.2 for women born in the 1960s (Zeman et al. 2018). After remaining low for mid-20th century birth cohorts, childlessness among 1970s female birth cohorts reached levels previously held by cohorts born during the 1920s: approximately 15 % (Sobotka 2017; Frejka 2017). Men and women transitioned into parenthood following their transition into marriage for most of the 20th century in the United States (Kiernan 2001). However, the percentage of nonmarital births among women under age 44 increased from 21 % in the early 1980s to 28 % in the early 1990s, only 29 % and 39 % of which occurred to cohabiting couples (Bumpass and Lu 2000).

The decoupling of parental home leaving and marriage as well as marriage and parenthood has made research on the sequencing of events during the transition to adulthood more important. While some scholars use multistate event history or life table modeling to study variation in life course origins and destinations (e.g., Schoen, Landale, and Daniels 2007; Zeng et al. 2012; Billari 2001), sequence analysis has emerged as a popular method to describe and visualize holistic life course trajectories (Abbott and Tsay 2000; MacIndoe and Abbott 2004; Aisenbrey and Fasang

[2] U.S. Census Bureau, Current Population Survey, March and Annual Social and Economic Supplements

2010). There are commonly three steps in sequence analysis: First, each individual trajectory is operationalized as a sequence by aligning the individual's life course states in chronological order with their respective durations. Second, the dissimilarity of each sequence pair is estimated using distance measures that indicate the degree of difference between any two sequences. Finally, a clustering algorithm is applied to group sequences into distinct units that are maximally homogenous.

Sequence analysis, combined with cluster analysis, reduces complexity in two ways. First, sequence analysis identifies common life course pathways within multitudes of trajectories that may warrant further study. Second, rather than analyzing numerous "point in time outcomes", such as the age of first birth, sequence analysis enables researchers to analyze life courses that amount from those outcomes, a "process outcome" (Abbott 2005). A number studies have used sequence analysis to identify life course patterns and how they vary across countries and birth cohorts (Baizan, Michielin, and Billari 2002; Bras, Liefbroer, and Elzinga 2010; Hofäcker and Chaloupková 2014; Robette 2010; Raab et al. 2014; Aisenbrey and Fasang 2017; Van Winkle 2018). Billari and Liefbroer (2010) concisely summarize the change in the transition to adulthood across birth cohorts in their study countries as a shift from an early, contracted and simple pattern to a late, protracted and complex pattern.

Previous Research on Family Life Course Complexity

The life course states within each of the dimensions discussed in the previous section – residence, unions, and parenthood – are often studied as independent spheres of the family life course (for example, see the reviews by Cherlin 2010; and Buchmann and Kriesi 2011). When two dimensions are studied simultaneously, it is generally union formation and parenthood (e.g., Thornton and Philipov 2009; Kiernan 2001; Hiekel and Castro-Martín 2014; Baizán, Aassve, and Billari 2004). However, it is important to consider the intersections of these dimensions as separate and qualitatively different life course states. For example, the social meaning and consequences of entering nonmarital parenthood within the parental home differs starkly from entering nonmarital parenthood outside the parental home (Kaphingst, Persky, and Lachance 2014). Naturally, this approach to identifying life course states increases the number of distinct states, some of which may be empirically scarce, e.g. divorced with children and in the parental home.

There is general agreement that complexity should be conceptualized in terms of life course differentiation (Mayer 1991; Mayer, Grunow, and Nitsche 2010; Elzinga and Liefbroer 2007; Biemann, Fasang, and Grunow 2011; Van Winkle and Fasang 2017; Van Winkle 2018). Brückner and Mayer (2005) define differentiation as an increase in the number of life course states experiences across the life course. Therefore, complexity has often been operationalized using a simple count measure of the number of life course states or transitions experienced across individuals' lives. However, complexity is also associated with an increase in life course uncertainty (Beck 1994, 2009; Sennett 1998; 2006; Mills and Blossfeld 2013, 2003). Composite metrics developed in sequence analysis have been developed that incorporate both the number of life course states as well as the degree of unpredictability (Elzinga and Liefbroer 2007; Gabadinho and Studer 2010; Van Winkle 2018). Sequence based complexity measures have the advantage that they can incorporate a large number life course states, i.e. the intersection of different life course dimensions, as well as a small number of simple states.

However, there is little research on the complexity of family trajectories using sequence-based complexity metrics. The bulk of studies on life course complexity are interested in the differentiation of education-work-retirement trajectories (Biemann, Fasang, and Grunow 2011; Ciganda 2015; A. Riekhoff 2016; 2018; Van Winkle and Fasang 2017). Although there are many studies that apply sequence and cluster analysis to family trajectories, only Elzing and Liefbroer (2007) studied the complexity of early family life courses in a number of countries and birth cohorts. They find that average early family life course complexity had only increased moderately across a small number of their study countries. Otherwise, average complexity had remained relatively stable. Van Winkle (2018) analyzed long-life family trajectories on a number of European countries and cohorts, and concluded that although complexity had increased across cohort, cross-national variation was much more substantial.

The Heritability of Family Life Course Complexity

The estimation of heritability is the first step to incorporate biodemography into the study of complexity. Heritability is a population level characteristic and reflects the genetic component to a trait or phenotype, here complexity. Specifically, heritability is the proportion of phenotypic variance that is attributed to genetic variance, i.e. to common genetic variants within a population,

as opposed to environmental variance (Domingue, Wedow, et al. 2016). The strength of heritability gives researchers insight on the potential importance of sociobiological explanations and the consequence of ignoring them. Note the important difference between the sociological concept of intergenerational transmission and the biodemographic concept of heritability. Intergenerational transmission denotes the similarity between parents and their children regardless whether this similarity arose through genetic or environmental factors, or a mixture of the two. Heritability in contrast is similarity between two individuals, related or not, attributable only to genetic factors.

During the 20th century, twin and family studies were the gold standard for estimating the genetic component of a given trait (Neale and Cardon 1992; Boomsma, Busjahn, and Peltonen 2002). A common analytical approach, the ACE model, compares monozygotic and dizygotic twins to decompose variance into an additive genetic component (A)[3], a component attributable to shared or common environmental factors, e.g. family background (C), and an environmental component unique to each twin (E) (see Diewald et al. 2015 for a brief discussion). Although sociologists have been mainly interested in quantifying the effects of social and family background (C), twin studies also demonstrate that many components of family life course complexity are heritable (A). The heritability of women's age at first birth has been estimated to be between 0 in Denmark to 0.3 in the UK. This means that genetic influences account for up to 30 % of the total variation of women's age at first birth in the UK, but none in the Denmark. The heritability of completed fertility has been estimated to be between 0.24 for Swedish and 0.43 for Danish women, and between 0.24 for Swedish and 0.28 for Danish men (Kohler et al. 1999; Tropf et al. 2015; Mills & Tropf 2015). Johnson and colleagues (2004) estimate large heritability in the propensity to marriage in the US: 0.72 for women and 0.66 for men. Estimates for the propensity to divorce are similarly high, between 0.52 and 0.59 for both US men and women (McGue and Lykken 1992; Jocklin, McGue, and Lykken 1996).

However, twin ACE models come with strong assumptions, which lead to biased heritability estimates if violated (Horwitz, Videon, and Schmitz 2003). For example, it is assumed that monozygotic twins do not mutually influence one another more than dizygotic twins and that dizygotic twins share an average of 50 percent of their genes (Conley et al. 2013). Further, heritability estimates from twin studies may not be generalizable if there is non-random genetic

[3] Examples of non-additive genetic effects are epistasis, i.e. interactions between genic variants, dominance deviations, suppression of genetic variants through other genetic variants, and gene-environment interactions.

stratification, e.g. genes associated with high fertility are more common among twins (Mills and Tropf 2015). Recent advances in molecular genetics and low-cost DNA sequencing methods has enabled researchers to base heritability estimates on true genetic similarity rather than assumed genetic similarity (Domingue, Wedow, et al. 2016). Molecular geneticists, as opposed to quantitative behavioral geneticists, are primarily interested in isolating and locating genetic variants associated with a trait, rather than estimating the heritability of a trait (Conley 2016). Genome-wide association studies (GWAS) are commonly used to locate single nucleotide polymorphisms (SNPs), markers of genetic variation, which are associated with a trait. Complex traits, such as fertility or cognitive ability, are polygenic traits that are affected by multiple SNPs, rather than a single gene. Following GWAS, polygenic risk scores (PGS) that estimate individuals' genetic predisposition for a certain trait are constructed. The variance that PGSs explain are often only a fraction of heritability estimates from twin models (see Eichler et al. 2010; Manolio et al. 2009 for a discussion on the problem of missing heritability).

Genome-wide complex trait analysis (GCTA) consists of several methodological approaches to estimate heritability based on all available SNPs. The genomic-relatedness-based restricted maximum-likelihood (GREML) model, utilizes genetic similarity to decompose the total variance of a trait into genetic and environmental components (Yang et al. 2011, 2010). GREML studies have been used to estimate the heritability of several anthropomorphic and some social demographic outcomes (Domingue, Wedow, et al. 2016). Tropf and colleagues (2015) recently estimated a 0.10 SNP heritability for completed fertility and a 0.15 SNP heritability for the age of first birth for a pooled sample of women from the UK and the Netherlands. SNP heritability estimates from GREML are generally larger than PGS heritability estimates, but still lower than twin based heritability estimates. A study by Yang and colleagues (2015) demonstrates that SNP heritability of height and BMI from GREML using the entire genome with imputed SNPs rather than a sample of SNPs are similar to those from twin studies. There are currently no studies that estimate SNP heritability for family demographic outcomes aside from fertility, such as marriage or divorce. However, based on the large heritability found in twin studies, it is likely that both life course states are highly heritable.

Exactly how genetic factors influence social demographic traits remains largely unclear (see Udry 1996 for an overview of early biosocial models for fertility). Kohler and colleagues (2006) argue that genetic predispositions affect fertility 1) directly through biological pathways, e.g. menarche,

2) indirectly through conscious life course decision-making, e.g. knowledge about fecundity, and 3) indirectly through subconscious life course decision-making, e.g. personality characteristics. Freese (2008) contends that any genetic effect is mediated through the body, which he terms the phenotypic bottleneck. He discusses four pathways of how genes influence outcomes through intermediate phenotypes and how sociological explanations may be affected. These biosocial pathways are displayed in Figure 1 with a stylized example. In the upper-left panel of Figure 1, genetic factors, G, partially determine the intermediate phenotype cognitive ability, Z, which affects the timing of first birth, Y (see Davies et al. 2016; Kirkpatrick et al. 2014 for the heritability of cognitive ability; see Haaga 2001 for the association between cognitive ability and fertility). Educational attainment, X, has an independent direct effect on age at 1st birth. In panel B, cognitive ability moderates the association between educational attainment and fertility, while in C cognitive ability simply precedes educational attainment. Finally, panel D demonstrates how cognitive ability is confounded with education. This is an especially worrisome case for sociological research, because effect estimations between two social phenomena, e.g. education and fertility, will often be biased if genetic factors remain unobserved.

Figure 1: Ideal Type Pathways for Genetic Effects on Sociological Relationships through Intermediate Phenotypes

GCTA-GREML approaches have estimated heritability for several sociological outcomes and anthropomorphic traits (see Domingue, Wedow, et al. 2016 for a broader overview). Height is estimated to be between 35 and 56 percent heritable, while BMI estimates are slightly lower, between 27 and 43 percent (Conley et al. 2014; Yang et al. 2015). Davies and colleagues (2016) estimate heritability of 31 percent for verbal-numeric reasoning, 5 percent for memory, and Kirkpatrick and colleagues (2014) estimate that general cognitive is 35 percent heritable. Heritability estimates for educational attainment are lie between 17 and 33 percent (Boardman, Domingue, and Daw 2015; Conley et al. 2014), and while social deprivation and household income is an estimated 21 and 11 percent heritable, respectively (Hill et al. 2016). Even socioeconomic status across the life course has been estimated to be between 18 and 19 percent heritable (Marioni et al. 2014; Trzaskowski et al. 2014). Considering the consistent findings for the relevance of genetic influences, family life course complexity are likely affected by genetic factors both directly,

e.g. biologically determined fecundity, and indirectly, e.g. phenotypes that affect family demographic decisions. Therefore, we expect that *the complexity of family trajectories will be heritable (H1)*.

The relevance of genetic factors for family life course complexity may vary across social contexts, e.g. across birth cohorts. Societal norms and institutions can change the heritability of social outcomes by influencing the relationship between intermediate phenotypes and outcomes. As an example, outside of family demography, Domingue and colleagues (2016) find evidence that the heritability of smoking in the United States increased from roughly 0.13 to 0.32 between cohorts born 1939-1945 and 1947-1959. They conclude that as evidence on the dangers of smoking emerged during the 1960's, the influence of genetic factors associated with nicotine addiction strengthened. Using historical twin data, Bras and colleagues (2013) found evidence for increasing heritability during the first demographic transition in 19th century. They argue that genetic predispositions for fertility became more important as the social control of women's fertility decreased. In these examples, environmental changes, i.e. knowledge on the effects of smoking and decreased social control, increased the effect of genetic factors, which in turn increased the heritability of smoking and fertility, respectively. As environmental forces increase or decrease the variance of behavioral outcomes, the relative importance of genes changed accordingly.

In family demography, the most common hypothesis is that the heritability of family demographic outcomes will be higher for cohorts transitioning to adulthood after the onset of the SDT. Udry (1996) proposes a multilevel biosocial model, where societal characteristics influence the relationships between genetic predispositions and outcomes. Specifically, the genetic influence on voluntary behavioral outcomes, e.g. entering parenthood and marriage, will decrease in societies with a high level of social constraints. Udry (1996, 335) argues in egalitarian and individualistic contexts behavioral variation will increase and genetic predispositions will have more opportunities to express themselves. Indeed, studies using twin data have observed higher heritability for Danish cohorts transitioning to adulthood after the onset of the SDT for fertility motivation and fertility (H. Kohler, Rodgers, and Christensen 1999, 2002; Rodgers et al. 2001). Tropf and colleagues (2015) also find that the heritability of UK women's age at first birth increased for cohorts characterized by liberalization and the sexual revolution, but that the introduction of modern contraception and economic recessions decreased heritability shortly after. Therefore, we expect

that *the heritability of family trajectory complexity will increase across birth cohorts (H2)*, especially for individuals who transitioned to adulthood after the onset of the SDT.

Data & Methods

Sample & Sequence Definition

The HRS[4] is a biennial panel study with prospective and retrospective data on family formation, which also recently collected molecular genetic information from their respondents. The original HRS cohort was first sampled in 1992 and consisted of men and women born between 1931-1941 at ages 51-61. A second study cohort, the Asset and Health Dynamics among the Oldest Old, of men and women born before 1924 was collected the following year. The two study cohorts were collected simultaneously in 1998 when the third and fourth cohorts were introduced, the Children of the Great Depression born between 1924 and 1930 and the War Babies born 1942-1947. Since then a refreshment sample has been added every three waves, i.e. six years. We restrict our analyses to respondents born between 1915 and 1965.

We estimate the heritability of a wide range of family demographic indicators that we use to construct family trajectories and calculate sequence complexity. Specifically, we analyze four fertility indicators: the age at first birth, average spacing between births in years, and the total number of biological children both including and excluding childless respondents. These variables are prepared using respondent's self-reports on the number of children and the year of their birth, but also include "best guess" information of the year of children's birth prepared by the RAND corporation. We exclude respondents from analyses on the age first birth if there is no information on the year of birth for any of their children. We prepare seven marital indicators for our analyses: whether respondents ever married, the age at first marriage, the number of marriages both including and excluding respondents who never married, whether respondents ever divorced, and the number of divorces both including all respondents who marriage and excluding respondents who never

[4] The HRS (Health and Retirement Study) is sponsored by the National Institute on Aging (grant number NIA U01AG009740) and is conducted by the University of Michigan. 1992-2014 RAND Fat Files, Cross-Wave: Polygenic Score Data, RAND HRS Longitudinal File 2014, and the RAND HRS Family Data 2014 public use datasets. Produced and distributed by the University of Michigan with funding from the National Institute on Aging (grant number NIA U01AG009740). Ann Arbor, MI, 2018. For more information, visit: http://hrsonline.isr.umich.edu/.

divorced. We exclude respondents with age at first birth, birth spacing, and age at first marriage values under the 1st percentile or above the 99th percentile from those respective analyses.

Using the information described above, we reconstruct individuals' family life courses as sequences from age 18 to 45 using 5 different sequence alphabets. The first sequence alphabet includes only information on fertility: at any given age, an individual can be childless (0C) or have any number of children (e.g. C1, C3, or C5). Similarly, our second sequence alphabet focuses on marital histories, where individuals can be single (S), in a first, second, or third marriage (M1, M2, M3), divorced (D) or widowed (W). The other three sequence specifications combine both union and fertility histories. The most differentiated alphabet simply combines the fertility and union sequences, generating states such as single with one child (SC1), married with two children (MC2), or divorced without children (DC0), or in a second marriage with five children (M2C5). We also generate sequences with a reduced alphabet, where states are only differentiated up to 3 children (e.g. MC1, MC2, MC3+), and a simple alphabet whether we only differentiate between being married or single and childless or a parent. Please note that cohabitation and residential histories have not been extensively collected by the HRS and therefore cannot be included in the sequence alphabet. However, it is unlikely that the lack of information on parental home leaving and cohabitation will bias our results. Individuals left the parental home are early ages relatively universally and cohabitation was not a common living arrangement for our study cohorts.

Estimating Genetic Similarity

Saliva samples were collected during enhanced face-to-face interviews during the 2006, 2008, 2010, and 2012 waves for 15,708 respondents. Genotyped data measuring a sample of approximately 2.4 million single nucleotide polymorphisms (SNP) was obtained from respondents' saliva samples using the Ilumina HumanOmni2.5 BeadChips.[5] SNPs are DNA nucleotide pairs that vary within a population. For example, at a single position in the genome some individuals may have the nucleotide cytosine, while others have thymine. The variants of each SNP are called alleles

[5] For detailed information on the collection and preparation on the genotypic data, see the Quality Control Report for Genotypic Data at http://hrsonline.isr.umich.edu/sitedocs/genetics/HRS2_qc_report_SEPT2013.pdf prepared by the University of Washington.

and the alleles that are the second most common in a population are termed minor alleles. Using the SNPs, genetic similarity is estimated as:

$$A_{jk} = \frac{1}{N}\sum_i \frac{(x_{ij}-2p_i)(x_{ik}-2p_i)}{2p_i(1-p_i)},$$

where N is the number of genetic markers, and x_{ij} and x_{ik} are the number of minor alleles at SNP i for individuals j and k, and p is the minor allele frequency. The matrix A containing the genetic similarity estimates between all respondent pairs is called the genetic relationship matrix (GRM). We calculate the GRMs using all autosomal[6] SNPs with a minor allele frequency above one percent in the sample to ensure that genetic similarity is based on variants that are common and not rare within a population.[7] We then prune the sample for cryptic relatedness ($A_{ij} \geq 0.025$), i.e. persons genetically comparable with second-degree cousins. It has been shown that genetically similar individuals also share similar environments, which could bias our heritability estimates by including variation attributable to shared environments. Although it has been demonstrated that the violation of this assumption does not substantially bias GREML estimates (Conley et al. 2014).

Estimating Heritability

We use GCTA-GREML to estimate the heritability of family life course complexity and its components (see Domingue, Wedow, et al. 2016 for an introduction on applications with social demographic traits; see also Yang et al. 2011). The underlying assumption of GREML is that if phenotypic variation is attributable to genetic variants, then individuals who are more similar genetically should be more similar phenotypically. GREML utilizes random effects modeling to quantify the proportion of trait variance that is attributable to genomic differences. Formally, a given phenotype, y, is modeled as:

$$y = X\beta + g + \epsilon,$$

[6] Autosomal chromosomes are non-sex chromosome pairs 1-22.

[7] GREML assume that the average effect size on the outcome of interest per standardized SNP is minute and normally distributed. The distribution assumption also assumes that SNPs with low minor allele frequencies have larger effects. Therefore, rare alleles are removed to ensure heritability estimates are not based on a rare variant found only in a select population (Yang et al. 2017, 1307–8).

where Xβ is a matrix of covariates and their coefficients, g is a vector of random effects and ϵ a vector of errors. As the name GREML indicates, the model is estimated using restricted maximum likelihood. The GRM enters the model through the variance component of the random effects:

$$g \sim Normal(0, \sigma_g^2 A).$$

This means that the individual random effects are normally distributed with a mean of zero and a variance of σ_g^2, which denotes the additive genetic variance captured by the SNPs. The GRM, here A, builds the variance-covariance structure of the random effect. Note that standard assumptions apply for GREML models, e.g. that ϵ is independent of X and g, and normally distributed. Heritability, h^2, is then defined as the proportion of total variance attributable to the variance of the random effects. Formally,

$$Var(y) = Var(g) + Var(\epsilon) = \sigma_g^2 + \sigma_\epsilon^2,$$

Where the variance of y, Var(y), is equal to the variance component of the random effect, σ_g^2, plus the residual variance, σ_ϵ^2. Subsequently, heritability is estimated as the variance component of the random effect, σ_g^2, divided by the total variance of y, $\sigma_g^2 + \sigma_\epsilon^2$:

$$h^2 = \frac{\sigma_g^2}{\sigma_g^2 + \sigma_\epsilon^2}.$$

This approach belongs to the class of mixed linear modeling, similar to multilevel modeling commonly used in social demography and sociology (e.g., Van Winkle 2018; Van Winkle and Fasang 2017 for cross-country and cross-cohort comparisons). Heritability is essentially an intraclass correlation coefficient commonly used in comparative sociological research. However, estimating country and cohort random effects to decompose the complexity variance attributable to differences across countries and cohorts, the random effect above is used to decompose the variance into a genetic and an environmental component.

We estimate SNP heritability for a pooled HRS sample as well as heritability for 25-year birth cohorts. Due to the issue of population stratification, we follow the convention in the literature and restrict our sample to self-identified Non-Black and Non-Hispanics. We use GCTA[8] to conduct all analyses (see Yang et al. 2011). We estimate both unadjusted and adjusted GREML models. In the

[8] http://cnsgenomics.com/software/gcta/#Overview

adjusted models, we include the respondents' gender and birth year as well as ten principle components to adjust for population stratification, i.e. differences in minor allele frequencies that are attributable to ancestral differences.[9] This adjustment has been criticized as it may correct for differences that are meaningful for the trait being studied and can lead to underestimated heritability estimates. All phenotypes except for whether respondents ever married or divorced are standardized by gender and birth year before the analyses.

Results

Trends in Sequence Complexity & its Components

Summary statistics for the Non-Black and Non-Hispanic HRS sample with genotyped data are displayed in Figure 2. We present both means (black lines) and standard deviations (grey lines) by birth year for all our fertility, marital, and divorce indicators as well as our sequence complexity variants. For example, the average age of first birth decreased from over 26 for the 1915 birth cohort to well under 24 for the 1940 cohort. During the same period the standard deviation in the age at first birth decreased from approximately 5 to 4.5 years. The age of first birth then increases to 25 for the 1950 birth cohort with a standard deviation of nearly 6 years. We observe a corresponding trends for birth spacing: the average number of years between births and its variance is highest for pre-1920 and post-1950 cohorts. The average number of children is highest at nearly 3.5 for the 1930 cohort with a standard deviation of 3.6, but dips well under an average of 3 children with an even lower variance for the 1950 cohort. According the Udry's (1996) proposed relationship between behavioral variation and heritability, we could expect to observe high heritability for age at first birth and birth spacing for younger cohorts and the opposite for the number of children.

[9] This is commonly called the "chopsticks problem", which arises when subgroups have different allele frequencies that coincide with a phenotype that is culturally rather than biologically determined (Hamer and Sirota 2000). For example, persons of Asian and European ancestry will systematically differ on SNP variants that would coincide with the different probability of chopstick use between the two subgroups.

Figure 2: Sample Means and Standard Deviations of Family Demographic Indicators by Birth Year

As can be seen under the marital indicators of Figure 2, the proportion of the sample that had ever married drop from nearly 100 percent to 90 percent between the 1940 and 1960 birth cohorts. During that period, the age at first marriage increased from just over 22 with a standard deviation of 4.5 to nearly 25 with a standard deviation of 6.5. The average number of marriages increased from roughly 1.3 for the 1920 cohort to 1.5 for the 1945 cohort before sinking again, likely due to higher rates of cohabitation preceding marriage and following divorce. The variance in the number of marriages, however, did not decline as steeply as the average number of marriages following the 1945 cohort. As would be expected, the propensity to divorce as well as the average number of divorces experienced and its variance increased across cohorts.

Trends in average complexity and its variation correspond to the trends above. The complexity of fertility sequences are high and vary largely for the 1930 cohort, reflecting the higher number of parity transitions and as well as the variation in the age of first birth, birth spacing, and number of children for cohorts before 1940. In contrast, the complexity of union sequences is highest for post-1950 cohorts, which matches the trend towards more variance in the propensity to marry and the number of marriages as well as a higher number of transitions into divorce. The complexity of sequences with both fertility and union statuses show differing trends. Average complexity for sequences with the most differentiated alphabet is highest and varies to a greater extent for older cohorts, which matches trajectories consisting of a larger number of fertility transitions than union transitions. The complexity of sequences with reduced and simple alphabets give somewhat more weight to union transitions. Average complexity is then higher for post-1950 cohorts, especially for sequences with the simple alphabet, and the variance of the complexity of sequences with both the reduced and simple alphabet increase across birth cohorts. Therefore, we might observe different trend for heritability depending on the sequence alphabet: decreasing along with lower variance for fertility and differentiated sequences, but increasing along with increasing variance for union sequences as well as sequences with a reduced and simple alphabet.

GREML Heritability Estimates

SNP heritability estimates for all our family demographic indicators and sequence complexity values from GREML models on the entire sample and their 95 percent confidence intervals are displayed in Figures 3 and 4. Estimates from unadjusted GREML models are displayed in Figure 3, while the estimates in Figure 4 are adjusted for 10 ancestral principal components as well as respondents' gender and birth year. The heritability estimates as well as their standard errors, p-Values, and sample sizes can be found in the first column of Table A1 for Figure 3 and Table A2 for Figure 4 in the manuscript appendix. We estimates approximate confidence intervals based on the estimated standard errors, which may be upwards biased (see Schweiger et al. 2016), making it difficult to discern which estimates are statistically different from zero. We therefore, mark heritability estimates that are statistically different from zero ($p \leq 0.05$) in black and those that are not statistically different from zero in grey.

Figure 3: Unadjusted SNP Heritability Estimates

Figure 4: Adjusted SNP Heritability Estimates

As can be seen in Figure 3, we find statistically significant heritability for most of our family demographic indicators, including the age at first birth, the number of children, the age at first marriage, the number of marriages, the propensity to divorce, and the number of divorces. Of those indicators, only the heritability estimate for the propensity to divorce becomes statistically insignificant after we adjust our GREML models. Heritability is high for both the age at first birth and first marriage: 21 percent of the variance in the age at first birth and 24 percent of the variance in the age of first marriage can be accounted for by genetic differences. Estimates are lowest for the number of children, marriages and divorces as well as the propensity to divorce, with roughly 0.15, 0.17, 0.13, and 0.09, respectively.

We also find statistically significant heritability for the complexity of fertility sequences as well as the complexity of sequences with a differentiated or simple alphabet. For example, results from unadjusted GREML models indicate that 13 percent of the complexity variance of fertility sequences is attributable to genetic factors. Common genetic differences account for 11 percent

and 10 percent of the complexity variance of sequences with differentiated and simple alphabets. Our heritability estimates for the complexity of union sequences and sequences with a reduced alphabet are smaller, roughly 0.06, and not statistically different from zero. Once our GREML models are adjusted using gender, birth year, and ancestral principal components, our heritability estimate for the complexity of sequences with a simple alphabet is no longer statistically different from zero. In contrast, the heritability estimates for the complexity of fertility and differentiated sequences increase slightly to 15 percent and 12 percent, respectively. In sum, we find partial support for our first hypothesis that the complexity of family trajectories will be heritable.

GREML Heritability Estimates across Birth Cohorts

SNP heritability estimates for our family demographic indicators and sequence complexity measures and their 95 percent confidence intervals are displayed by birth cohort in Figures 5 and 6. Estimates from unadjusted GREML models are depicted in Figure 5 and estimates from adjusted GREML models are shown in Figure 6. Each estimate is based on a 26-year birth cohort. For example, the 1930 heritability estimates are based on individuals born between 1917 to 1943. Cohort heritability is estimated using 26-year cohorts to ensure a sufficiently large sample to identify moderately large heritability ($N \approx 5{,}000$ for $h^2 \approx 0.2$) (see Visscher et al. 2014). These figures allow us to gage whether the heritability of family demographic behavior is changing across birth cohorts. Similar to Figures 3 and 4, heritability estimates that are statistically different from zero ($p \leq 0.05$) are black and those that are not statistically different from zero are grey. All heritability estimates as well as their standard errors, p-Values, and sample sizes can be found in Tables A1 and A2 for Figure 5 and 6, respectively (see manuscript appendix).

Figure 5: Unadjusted SNP Heritability Estimates by 25-Year Birth Cohorts

Fertility Indicators

Figure 6: Adjusted SNP Heritability Estimates by 25-Year Birth Cohorts

Fertility Indicators

Broadly speaking, we find little evidence that the heritability of family demographic behavior changed across US cohorts born between 1915 and 1965. For example, the unadjusted heritability of the age at first birth fluctuates between roughly 36 percent for the 1928 birth cohort and 26 percent for the 1946 cohort (see Figure 5). However, the estimates are not statistically different from one another. The adjusted heritability estimates for the age at first birth shown in Figure 6 also do not statistically differ across birth cohorts, although the estimates vary from 14 percent for the 1953 cohort and 29 percent for the 1928 cohort. Similar trends – relatively constant non-zero heritability estimates – can be found for the age at first marriage and the number of marriages. Heritability estimates for the propensity to divorce and the number of divorces are statistically different from zero for older cohorts, but become statistically insignificant for post-1945 cohorts. We do find more considerable and statistically significant differences between the heritability of the number of children between younger and older birth cohorts. However, rather than increasing heritability, our results show that heritability drops from 31 percent for the 1928 cohort to zero for the post-1950 cohorts.

Similar to the overall estimates, we find statically significant and non-zero heritability for the complexity of fertility sequences and sequences with differentiated or simple alphabet from unadjusted GREML models. Our results also indicate that like the cohort trends for the propensity to divorce, the number of divorces and the number of children, the non-zero heritability estimates for complexity become statistically insignificant for younger birth cohorts. Cohort-specific estimates from adjusted GREML models demonstrate that only the complexity of fertility and differentiated sequences are statistically significant for pre-1940 birth cohorts. For example, nearly 22 percent of the complexity variance of sequences with a differentiated alphabet can be attributed to genetic factors for the 1928 cohort, but none for post-1940 cohorts. In sum, our results do not support our second hypothesis that the heritability of family trajectory complexity will increase across birth cohorts.

Discussion

In this study, we applied for the first time a biodemographic approach to the study of family life course complexity using methods developed in molecular genetics. Specifically, we estimated the

heritability of family life course complexity as well as a wide range of family demographic indicators using GCTA-GREML on data from the HRS. Based on findings from behavioral genetics research on twins, we hypothesized that family life course complexity as well as its components will be moderately heritable (H1). Further, based on arguments by Udry (1996; see also H. P. Kohler et al. 2006) that heritability increases in contexts characterized by fewer social constraints, we hypothesized that the heritability of family life course complexity and its components will increase across birth cohorts in the United States (H2). While we do indeed find moderate heritability for the complexity of fertility and differentiated sequences as well as other family demographic indicators (H1), we find no evidence for increasing heritability (H2).

Our results support micro-biosocial models that family demographic (e.g. H. P. Kohler et al. 2006; Udry 1996) and sociological (Freese 2008) outcomes are heritable. On account of the composite nature of family life course complexity – incorporating both fertility, union formation, and union dissolution – it seems plausible that genetic factors influence complexity directly through biological mechanisms and indirectly through intermediate phenotypes, such as personality. Our SNP heritability estimates for fertility are slightly higher than those estimated by Tropf and colleagues (2015) on a pooled sample of UK and Dutch women. As would be expected, SNP heritabilities for marriage and divorce are somewhat lower than earlier estimates using twin data (Johnson et al. 2004 for marriage; Jocklin, McGue, and Lykken 1996; McGue and Lykken 1992 for divorce). In sum, our analyses corroborate previous literature that family demographic behavior and demonstrate that even composite life course outcomes are heritable.

However, our results do not systematically validate Udry's (1996) hypothesis that the genetic influence on voluntary behavioral outcomes, e.g. entering parenthood and marriage, will increase in societies characterized by the SDT. A possible explanation might lie in additional hypothesis made by Udry (1996, 328) that has received less attention: "The more similar the social constraints faced by individuals in a society (…) the higher the proportion of variance in their behavior that is controlled by biological differences in the population". While shifts in value orientations and legal opportunities for divorce may be similar between the US and many Northwestern European countries, the universality of those constraints may be higher in generous European welfare states that are secularized to a higher degree. Further analyses, given adequate sample sizes, should estimate heritability separately by educational group or socioeconomic status.

There are limitations to the estimation of heritability with GREML and using SNPs (see Krishna Kumar et al. 2016 for an overview). The GCTA-GREML approach is only valid for traits with genetic variants located throughout the entire genome that are weakly associated with that trait (Domingue, Wedow, et al. 2016). This is because the GRM only approximates genome-wide genetic similarity if the sample of SNPs and their association with a trait are a reasonably good proxy for the entire genome. The SNP heritability estimate may be biased if a genetic variant with an exceptionally large association is missing. However, it is unlikely that our heritability estimate for sequence complexity, which is per definition highly polygenic due to its composite nature, is biased in such a manner.

A further limitation that GCTA shares with other estimation methods for heritability, is the "black box" nature of heritability. Without knowing the loci associated with complexity and the phenotypic effects of those loci, it is not possible to follow the causal biological and social pathways connecting genetic factors with complexity. A possible first step would be to estimate chromosome-specific SNP heritability, because molecular geneticists have identified specific chromosomes that are associated with phenotypes to a greater or lesser degree. Future research could use bivariate GREML models to test the genetic correlation between two phenotypes, such as complexity and personality (Deary et al. 2012; Lee et al. 2012). This would allow scholars to inch towards untangling the causal mechanisms involved in the heritability of family life course complexity. Similarly, analysis of specific genetic associations—top hits—from a GWAS of sequence complexity would allow for functional analysis of the particular roles of those genes that exert the most influence on the outcome.

What role can family, labor market, and welfare policy play in societies where social demographic outcomes are heritable? Murry & Herrnstein's (1994) deeply controversial book "The Bell Curve" argued that the United States had become a "genotocracy" (term applied later by Conley and Fletcher 2017): a society where social stratification is based on genetic differences and social policy only could alleviate the consequences of those differences. Recently, Conley & Fletcher (2017) published a systematic critique of the perspective that stratification patterns in western societies are genetically based and unalterable. Even if heritability is extremely high, it does not indicate genetic determinism. The impact of genetic factors on sociological and demographic outcomes is dependent on the social environment and must be contextualized. Although we do not find evidence for cross-temporal change in heritability, a comparison of our estimates with findings using

European data (e.g. Tropf, Stulp, et al. 2015) indicates strong gene-environment interactions (see also Tropf et al. 2017). The reaction of individuals with similar genotypes likely varies across social contexts, e.g. when institutions effect the relationship between intermediate phenotypes and social processes. There is also increasing evidence in the area of epigenetics that environments also triggers or suppresses the manifestation of genetic factors at a molecular level (Landecker and Panofsky 2013). Our results in no way support arguments that stratification patterns are static due to genetic influences and cannot be altered by social policy.

Our results demonstrate that holistic life course outcomes can also be heritable and that life course sociologists should begin to incorporate biodemography into their research. A first starting point is to estimate heritability for more life course outcomes that are of interest. For example, the heritability of life course patterns derived from cluster analysis and the heritability of deviations from those patterns would be an obvious next step. Depending on the outcomes of those analyses, life course sociologists may need to add existing educational and fertility polygenic risk scores into their analyses that estimate the association between individual characteristics and life course patterns. This could give sociologists leverage on whether educational attainment is causally associated with the choice to follow a certain life course path or whether unobserved intermediate phenotypes are confounding our analyses.

Works Cited

Abbott, Andrew. 2005. "Process and Temporality in Sociology: The Idea of Outcome in U.S. Sociology." In *The Politics of Method in the Human Sciences: Positivism and Its Epistemological Others*, edited by G Steinmetz, 393–426. Durham, NC: Duke University Press.

Abbott, Andrew, and Angela Tsay. 2000. "Sequence Analysis and Optimal Matching Methods in Sociology: Review and Prospect." *Sociological Methods & Research* 29 (1): 3–33. https://doi.org/10.1177/0049124100029001001.

Aisenbrey, Silke, and Anette Fasang. 2017. "The Interplay of Work and Family Trajectories over the Life Course: Germany and the United States in Comparison." *American Journal of Sociology* 122 (5): 1448–84. https://doi.org/10.1086/691128.

Aisenbrey, Silke, and Anette Eva Fasang. 2010. "New Life for Old Ideas: The 'Second Wave' of Sequence Analysis Bringing the 'Course' Back Into the Life Course." *Sociological Methods & Research* 38 (3): 420–62. https://doi.org/10.1177/0049124109357532.

Amato, Paul. 2010. "Research on Divorce: Continuing Trends and New Developments." *Journal of Marriage and Family* 72 (3): 650–66. https://doi.org/10.1111/j.1741-3737.2010.00723.x.

Baizán, Pau, Arnstein Aassve, and Francesco Billari. 2004. "The Interrelations between Cohabitation, Marriage and First Birth in Germany and Sweden." *Population and Environment* 25 (6): 531–61. https://doi.org/10.1023/B:POEN.0000039064.65655.3b.

Baizan, Pau, Francesca Michielin, and Francesco Billari. 2002. "Political Economy and Life Course Patterns: The Heterogeneity of Occupational, Family and Household Trajectories of Young Spaniards." *Demographic Research* 6: 191–240.

Balbo, Nicoletta, Francesco Billari, and Melinda Mills. 2013. "Fertility in Advanced Societies: A Review of Research." *European Journal of Population* 29 (1): 1–38. https://doi.org/10.1007/s10680-012-9277-y.

Beck, Ulrich. 1994. *Risk Society: Towards a New Modernity*. London: Sage Publications. https://doi.org/10.2307/2579937.

———. 2009. *World at Risk*. Cambridge: Polity Press.

Bengtson, Vern L. 2001. “Beyond the Nuclear Namily: The Increasing Importance of Multigenerational Bonds.” *Journal of Marriage and Family* 63 (1): 1–16. https://doi.org/10.1111/j.1741-3737.2001.00001.x.

Biemann, Torsten, Anette Eva Fasang, and Daniela Grunow. 2011. “Do Economic Globalization and Industry Growth Destabilize Careers? An Analysis of Career Complexity and Career Patterns Over Time.” *Organization Studies* 32 (12): 1639–63. https://doi.org/10.1177/0170840611421246.

Billari, Francesco. 2001. “The Analysis of Early Life Courses: Complex Descriptions of the Transition to Adulthood.” *Journal of Population Research* 18 (2): 119–42. https://doi.org/10.1007/BF03031885.

Billari, Francesco, and Aart Liefbroer. 2010. “Towards a New Pattern of Transition to Adulthood?” *Advances in Life Course Research* 15 (2–3): 59–75. http://linkinghub.elsevier.com/retrieve/pii/S1040260810000407.

Boardman, Jason D, Benjamin Domingue, and Jonathan Daw. 2015. “What Can Genes Tell Us about the Relationship between Education and Health?” *Social Science & Medicine* 127: 171–80. https://doi.org/https://doi.org/10.1016/j.socscimed.2014.08.001.

Boomsma, Dorret, Andreas Busjahn, and Leena Peltonen. 2002. “Classical Twin Studies and Beyond.” *Nature Reviews Genetics* 3 (11): 872–82. https://doi.org/10.1038/nrg932.

Bras, Hilde, Jan Van Bavel, and Kees Mandemakers. 2013. “Unraveling the Intergenerational Transmission of Fertility: Genetic and Shared-Environment Effects during the Demographic Transition in the Netherlands, 1810–1910.” *The History of the Family* 18 (2): 116–34. https://doi.org/10.1080/1081602X.2013.803491.

Bras, Hilde, Aart C. Liefbroer, and Cees H. Elzinga. 2010. “Standardization of Pathways to Adulthood? An Analysis of Dutch Cohorts Born between 1850 and 1900.” *Demography* 47 (4): 1013–34. https://doi.org/10.1007/BF03213737.

Brückner, Hannah, and Karl Ulrich Mayer. 2005. “The De-Standardization of the Life Course: What It Might Mean and If It Means Anything Whether It Actually Took Place.” In *The Structure of the Life Course. Standardized? Individualized? Differentiated?*, edited by Ross Macmillan, 27–54. Amsterdam: Elsevier.

Buchmann, Marlis C., and Irene Kriesi. 2011. “Transition to Adulthood in Europe.” *Annual Review of Sociology* 37: 481–503.

Bumpass, Larry, and Hsien Hen Lu. 2000. “Trends in Cohabitation and Implications for Children’s Family Contexts in the United States.” *Population Studies* 54 (1): 29–41. https://doi.org/10.1080/713779060.

Cherlin, Andrew. 2010. “Demographic Trends in the United States: A Review of Research in the 2000s.” *Journal of Marriage and the Family* 72 (3): 403–19. https://doi.org/10.1111/j.1741-3737.2010.00710.x.

Ciganda, Daniel. 2015. “Unstable Work Histories and Fertility in France: An Adaptation of Sequence Complexity Measures to Employment Trajectories.” *Demographic Research* 32 (1): 843–76. https://doi.org/10.4054/DemRes.2015.32.28.

Coleman, Marilyn, Lawrence Ganong, and Mark Fine. 2000. “Reinvestigating Remarriage: Another Decade of Progress.” *Journal of Marriage and Family* 62 (4): 1288–1307. https://doi.org/10.1111/j.1741-3737.2000.01288.x.

Conley, Dalton. 2016. “Socio-Genomic Research Using Genome-Wide Molecular Data.” *Annual Review of Sociology* 42 (1): 275–99. https://doi.org/10.1146/annurev-soc-081715-074316.

Conley, Dalton, and Jason Fletcher. 2017. *The Genome Factor: What the Social Genomics Revolution Reveals about Ourselves, Our History, and the Future*. Princeton, NJ: Princeton University Press.

Conley, Dalton, Emily Rauscher, Christopher Dawes, Patrik K.E. Magnusson, and Mark L. Siegal. 2013. “Heritability and the Equal Environments Assumption: Evidence from Multiple Samples of Misclassified Twins.” *Behavior Genetics* 43 (5): 415–26. https://doi.org/10.1007/s10519-013-9602-1.

Conley, Dalton, Mark L Siegal, Benjamin Domingue, Kathleen Mullan Harris, Matthew B McQueen, and Jason D Boardman. 2014. “Testing the Key Assumption of Heritability Estimates Based on Genome-Wide Genetic Relatedness.” *Journal Of Human Genetics* 59 (March): 342. http://dx.doi.org/10.1038/jhg.2014.14.

D’Onofrio, Brian M., and Benjamin B. Lahey. 2010. “Biosocial Influences on the Family: A

Decade Review." *Journal of Marriage and Family* 72 (3): 762–82. https://doi.org/10.1111/j.1741-3737.2010.00729.x.

Davies, G., R. E. Marioni, D. C. Liewald, W. D. Hill, S. P. Hagenaars, S. E. Harris, S. J. Ritchie, et al. 2016. "Genome-Wide Association Study of Cognitive Functions and Educational Attainment in UK Biobank (N=112 151)." *Molecular Psychiatry* 21 (6): 758–67. https://doi.org/10.1038/mp.2016.45.

Deary, Ian J, Jian Yang, Gail Davies, Sarah E Harris, Albert Tenesa, David Liewald, Michelle Luciano, et al. 2012. "Genetic Contributions to Stability and Change in Intelligence from Childhood to Old Age." *Nature*. Department of Psychology, University of Edinburgh, 7 George Square, Edinburgh EH8 9JZ, UK. i.deary@ed.ac.uk. https://doi.org/10.1038/nature10781.

Diewald, Martin, Tina Baier, Wiebke Schulz, and Reinhard Schunck. 2015. "Status Attainment and Social Mobility." *KZfSS Kölner Zeitschrift Für Soziologie Und Sozialpsychologie* 67 (S1): 371–95. https://doi.org/10.1007/s11577-015-0317-6.

Domingue, Benjamin, Dalton Conley, Jason Fletcher, and Jason D Boardman. 2016. "Cohort Effects in the Genetic Influence on Smoking." *Behavior Genetics* 46 (1): 31–42. https://doi.org/10.1007/s10519-015-9731-9.

Domingue, Benjamin, Robbee Wedow, Dalton Conley, Matt McQueen, Thomas Hoffmann, and Jason Boardman. 2016. "Genome-Wide Estimates of Heritability for Social Demographic Outcomes." *Biodemography and Social Biology* 62 (1): 1–18. https://doi.org/10.1002/adma.201403943.Evaluating.

Eichler, Evan E., Jonathan Flint, Greg Gibson, Augustine Kong, Suzanne M. Leal, Jason H. Moore, and Joseph H. Nadeau. 2010. "Missing Heritability and Strategies for Finding the Underlying Causes of Complex Disease." *Nature Reviews Genetics* 11 (6): 446–50. https://doi.org/10.1038/nrg2809.

Elzinga, Cees H., and Aart C. Liefbroer. 2007. "De-Standardization of Family-Life Trajectories of Young Adults: A Cross-National Comparison Using Sequence Analysis." *European Journal of Population / Revue Européenne de Démographie* 23 (3–4): 225–50. https://doi.org/10.1007/s10680-007-9133-7.

Esping-Andersen, Gøsta. 1990. *The Three Worlds of Welfare Capitalism*. Princeton, NJ: Princeton University Press.

———. 1999. *Social Foundations of Postindustrial Economies*. Oxford: Oxford University Press. https://doi.org/10.1093/0198742002.001.0001.

Evans, Ann. 2013. "Generational Change in Leaving the Parental Home." In *Negotiating the Life Course: Stability and Change in Life Pathways*, edited by Ann Evans and Janeen Baxter, 53–67. Dordrecht: Springer Netherlands. https://doi.org/10.1007/978-90-481-8912-0_4.

Finer, Lawrence B., and Jesse M. Philbin. 2014. "Trends in Ages at Key Reproductive Transitions in the United States, 1951-2010." *Women's Health Issues* 24 (3): e271–79. https://doi.org/10.1016/j.whi.2014.02.002.

Freese, Jeremy. 2008. "Genetics and the Social Science Explanation of Individual Outcomes." *American Journal of Sociology* 114 (S1): 1–35.

Frejka, Tomas. 2017. "Childlessness in the United States." In *Childlessness in Europe: Contexts, Causes, and Consequences*, edited by Michaela Kreyenfeld and Dirk Konietzka, 159–79. Cham: Springer International Publishing. https://doi.org/10.1007/978-3-319-44667-7_8.

Gabadinho, Alexis, Gilbert Ritschard, Matthias Studer, and Nicolas S. Müller. 2010. "Indice de Complexité Pour Le Tri et La Comparaison de Séquences Catégorielles." *Revue Des Nouvelles Technologies de l'information RNTI E-19*, no. September 2015: 61–66.

Gabadinho, Alexis, and Matthias Studer. 2010. "Indice de Complexité Pour Le Tri et La Comparaison de Séquences Catégorielles." *Revue Des Nouvelles Technologies de l'information RNTI E-19*, no. September 2015: 61–66.

Goldscheider, Frances. 1997. "Recent Changes in U.S. Young Adult Living Arrangements in Comparative Perspective." *Journal of Family Issues* 18 (6): 708–24. https://doi.org/10.1177/019251397018006008.

Goldstein, JR, and CT Kenney. 2001. "Marriage Delayed or Marriage Forgone? New Cohort Forecasts of First Marriage for US Women." *American Sociological Review* 66 (4): 506–19.

Haaga, John G. 2001. "Comment : The Pace of Fertility Decline and the Utility of Evolutionary Approaches." *Population and Development Review* 27 (2001): 53–59.

Hamer, D. H., and L. Sirota. 2000. “Beware the Chopsticks Gene.” *Molecular Psychiatry* 5 (1): 11–13. https://doi.org/10.1038/sj.mp.4000662.

Hiekel, Nicole, and Teresa Castro-Martín. 2014. “Grasping the Diversity of Cohabitation: Fertility Intentions among Cohabiters across Europe.” *Journal of Marriage and Family* 76 (3): 489–505. https://doi.org/10.1111/jomf.12112.

Hill, W David, Saskia P Hagenaars, Riccardo E Marioni, Sarah E Harris, David C M Liewald, Gail Davies, Aysu Okbay, Andrew M McIntosh, Catharine R Gale, and Ian J Deary. 2016. “Molecular Genetic Contributions to Social Deprivation and Household Income in UK Biobank.” *Current Biology* 26 (22): 3083–89. https://doi.org/https://doi.org/10.1016/j.cub.2016.09.035.

Hofäcker, Dirk, and Jana Chaloupková. 2014. “Patterns of Family Life Courses in Europe – between Standardisation and Diversity A Cross-National Comparison of Family Trajectories and Life Course Norms in European Countries.” *Comparative Population Studies* 39 (3): 559–86. https://doi.org/10.12765/CPoS-2014-11en.

Horwitz, Allan, Tami Videon, and Mark F Schmitz. 2003. “Rethinking Twins and Environments : Possible Social Sources for Assumed Genetic Influences in Twin Research.” *Journal of Health and Social Behavior* 44 (2): 111–29.

Jocklin, Victor, Matt McGue, and David T. Lykken. 1996. “Personality and Divorce: A Genetic Analysis.” *Journal of Personality and Social Psychology* 71 (2): 288–99. https://doi.org/10.1037/0022-3514.71.2.288.

Johnson, Wendy, Matt McGue, Robert F. Krueger, and Thomas J. Bouchard. 2004. “Marriage and Personality: A Genetic Analysis.” *Journal of Personality and Social Psychology* 86 (2): 285–94. https://doi.org/10.1037/0022-3514.86.2.285.

Kaa, D. J. Van de. 1987. “Europe’s Second Demographic Transition.” *Population Bulletin*, Population bulletin, 42 (1).

———. 2001. “Postmodern Fertility Preferences: From Changing Value Orientation to New Behavior.” *Population and Development Review*, 290–331.

Kaphingst, Kimberly A, Susan Persky, and Christina Lachance. 2014. “Parenthood and Leaving

Home in Young Adulthood." *Population Research and Policy Review* 33 (6): 771–96. https://doi.org/10.1080/10810730902873927.Testing.

Kiernan, K. 2001. "The Rise of Cohabitation and Childbearing Outside Marriage in Western Europe." *International Journal of Law, Policy and the Family* 15 (1): 1–21.

Kirkpatrick, Robert M., Matt Mcgue, William G. Iacono, Michael B. Miller, and Saonli Basu. 2014. "Results of a '"GWAS plus:"' General Cognitive Ability Is Substantially Heritable and Massively Polygenic." *PLoS ONE* 9 (11). https://doi.org/10.1371/journal.pone.0112390.

Kohler, Hans-Peter, Joseph L Rodgers, and Kaare Christensen. 1999. "Is Fertility Behavior in Our Genes? Findings from a Danish Twin Study." *Population and Development Review* 25 (2): 253–88.

Kohler, Hans-Peter, Joseph Lee Rodgers, and Kaare Christensen. 2002. "Between Nurture and Nature: The Shifting Determinants of Female Fertility in Danish Twin Cohorts." *Social Biology* 49 (3–4): 218–48. https://doi.org/10.1080/19485565.2002.9989060.

Kohler, Hans Peter, Joseph Lee Rodgers, Warren B. Miller, Axel Skytthe, and Kaare Christensen. 2006. "Bio-Social Determinants of Fertility." *International Journal of Andrology* 29 (1): 46–53. https://doi.org/10.1111/j.1365-2605.2005.00606.x.

Koops, Judith C., Aart C. Liefbroer, and Anne H. Gauthier. 2017. "The Influence of Parental Educational Attainment on the Partnership Context at First Birth in 16 Western Societies." *European Journal of Population* 33 (4): 533–57. https://doi.org/10.1007/s10680-017-9421-9.

Krishna Kumar, Siddharth, Marcus W. Feldman, David H. Rehkopf, and Shripad Tuljapurkar. 2016. "Limitations of GCTA as a Solution to the Missing Heritability Problem." *Proceedings of the National Academy of Sciences* 113 (1): E61–70. https://doi.org/10.1073/pnas.1520109113.

Landecker, Hannah, and Aaron Panofsky. 2013. "From Social Structure to Gene Regulation, and Back: A Critical Introduction to Environmental Epigenetics for Sociology." *Annual Review of Sociology* 39 (1): 333–57. https://doi.org/10.1146/annurev-soc-071312-145707.

Lee, S. H., J. Yang, M. E. Goddard, P. M. Visscher, and N. R. Wray. 2012. "Estimation of

Pleiotropy between Complex Diseases Using Single-Nucleotide Polymorphism-Derived Genomic Relationships and Restricted Maximum Likelihood." *Bioinformatics* 28 (19): 2540–42. https://doi.org/10.1093/bioinformatics/bts474.

Lesthaeghe, Ron. 1995. "The Second Demographic Transition: An Interpretation." In *Gender and Family Change in Industrialized Countries*, edited by K. O. Mason and A.-M. Jensen. Oxford: Oxford University Press.

———. 2010. "The Unfolding Story of the Second Demographic Transition." *Population and Development Review* 36 (2): 211–51. https://doi.org/10.1111/j.1728-4457.2010.00328.x.

———. 2014. "The Second Demographic Transition: A Concise Overview of Its Development." *Proceedings of the National Academy of Sciences* 111 (51): 18112–15. https://doi.org/10.1073/pnas.1420441111.

MacIndoe, Heather, and Andrew Abbott. 2004. "Sequence Analysis and Optimal Matching Techniques for Social Science Data." In *Handbook of Data Analysis*, edited by Melissa Hardy and Alan Bryman, 387–406. Sage Publications.

Manning, Wendy D., Susan L. Brown, and Krista K. Payne. 2014. "Two Decades of Stability and Change in Age at First Union Formation." *Journal of Marriage and Family* 76 (2): 247–60. https://doi.org/10.1111/jomf.12090.

Manolio, Teri A, Francis S Collins, Nancy J Cox, David B Goldstein, Lucia A Hindorff, David J Hunter, Mark I McCarthy, et al. 2009. "Finding the Missing Heritability of Complex Diseases." *Nature* 461 (October): 747. https://doi.org/10.1038/nature08494.

Marioni, Riccardo E, Gail Davies, Caroline Hayward, Dave Liewald, Shona M Kerr, Archie Campbell, Michelle Luciano, et al. 2014. "Molecular Genetic Contributions to Socioeconomic Status and Intelligence." *Intelligence* 44: 26–32. https://doi.org/https://doi.org/10.1016/j.intell.2014.02.006.

Mayer, Karl Ulrich. 1991. "Soziale Ungleichheit Und Die Differenzierung von Lebensverläufen." In *Die Modernisierung Moderner Gesellschaften*, edited by Wolfgang Zapf, 667–87. Frankfurt/Main: Campus Verlag.

———. 1997. "Notes on a Comparative Political Economy of Life Courses." *Comparative Social*

Research 16: 203–26.

———. 2004. “Whose Lives? How History, Societies, and Institutions Define and Shape Life Courses.” *Research in Human Development* 1 (3): 161–87. https://doi.org/10.1207/s15427617rhd0103_3.

———. 2009. “New Directions in Life Course Research.” *Annual Review of Sociology* 35: 413–33.

Mayer, Karl Ulrich, Daniela Grunow, and Natalie Nitsche. 2010. “Mythos Flexibilisierung? Wie Instabil Sind Berufsbiografien Wirklich Und Als Wie Instabil Werden Sie Wahrgenommen?” *Kolner Zeitschrift Fur Soziologie Und Sozialpsychologie* 62: 369–402. https://doi.org/10.1007/s11577-010-0106-1.

McGue, M, and DT Lykken. 1992. “Genetic Influence on Risk of Divorce.” *Psychological Science* 3 (6): 368–73. http://pss.sagepub.com/content/3/6/368.short.

McLanahan, Sara, and Christine Percheski. 2008. “Family Structure and the Reproduction of Inequalities.” *Annual Review of Sociology* 34 (1): 257–76. https://doi.org/10.1146/annurev.soc.34.040507.134549.

Mills, Melinda, and Hans-peter Blossfeld. 2003. “Globalization , Uncertainty and Changes in Early Life Courses.” *Zeitschrift Für Erziehungswissenschaft* 6 (2): 188–218.

Mills, Melinda, and Hans-Peter Blossfeld. 2005. “Globalization, Uncertainty and the Early Life Course. A Theoretical Framework.” In *Globalization, Uncertainty and Youth in Society*, edited by Hans-Peter Blossfeld, E Klijzing, Melinda Mills, and Karin Kurz, 1–24. New York: Routledge.

———. 2013. “The Second Demographic Transition Meets Globalization: A Comprehensive Theory to Understand Changes in Family Formation in an Era of Rising Uncertainty.” In *Negotiating the Life Course: Stability and Change in Life Pathways*, edited by Ann Evans and Janeen Baxter, 9–33. Dordrecht: Springer Netherlands. https://doi.org/10.1007/978-90-481-8912-0_2.

Mills, Melinda, and Felix Tropf. 2015. “The Biodemography of Fertility: A Review and Future Research Frontiers.” *KZfSS Kölner Zeitschrift Für Soziologie Und Sozialpsychologie* 67

(S1): 397–424. https://doi.org/10.1007/s11577-015-0319-4.

Mitchell, B. 2006. *The Boomerage Age*. New York, NY: Routledge.

Morgan, S. Philip, and Miles G. Taylor. 2006. "Low Fertility at the Turn of the Twenty-First Century." *Annual Review of Sociology* 32 (1): 375–99. https://doi.org/10.1146/annurev.soc.31.041304.122220.

Murry, C, and RJ Herrnstein. 1994. *The Bell Curve: The Reshaping of American Life by Differences in Intelligence*. New York: Free Press.

Neale, Michael C., and Lon R. Cardon. 1992. *Methodology for Genetic Studies of Twins and Families*. Dordrecht: Springer Netherlands. https://doi.org/10.1007/978-94-015-8018-2.

Perelli-Harris, Brienna, and Mark Lyons-Amos. 2015. "Changes in Partnership Patterns across the Life Course: An Examination of 14 Countries in Europe and the United States." *Demographic Research* 33 (1): 145–78. https://doi.org/10.4054/DemRes.2015.33.6.

Power, R A, and M Pluess. 2015. "Heritability Estimates of the Big Five Personality Traits Based on Common Genetic Variants." *Translational Psychiatry* 5 (July): e604. http://dx.doi.org/10.1038/tp.2015.96.

Raab, Marcel, Anette Eva Fasang, Aleksi Karhula, and Jani Erola. 2014. "Sibling Similarity in Family Formation." *Demography* 51 (6): 2127–54. https://doi.org/10.1007/s13524-014-0341-6.

Riekhoff, Aart-jan. 2016. "De-Standardisation and Differentiation of Retirement Trajectories in the Context of Extended Working Lives in the Netherlands." *Economic and Industrial Democracy*, 1–28. https://doi.org/10.1177/0143831X16669593.

Riekhoff, Aart-Jan. 2018. "Extended Working Lives and Late-Career Destabilisation: A Longitudinal Study of Finnish Register Data." *Advances in Life Course Research* 35 (June 2017): 114–25. https://doi.org/10.1016/j.alcr.2018.01.007.

Robette, Nicolas. 2010. "The Diversity of Pathways to Adulthood in France: Evidence from a Holistic Approach." *Advances in Life Course Research* 15 (2–3): 89–96. https://doi.org/http://dx.doi.org/10.1016/j.alcr.2010.04.002.

Rodgers, Joseph Lee, Hans-Peter Kohler, Kirsten O H M Kyvik, and Kaare Christensen. 2001. "Behavior Genetic Modeling of Human Fertility: Findings from a Contemporary Danish Twin Study." *Demography* 38 (1): 29–42.

Rosenfeld, Michael J. 2007. *The Age of Independence: Interracial Unions, Same-Sex Unions, and the Changing American Family*. Harvard University Press.

Sandberg-Thoma, Sara E., Anastasia R. Snyder, and Bohyun Joy Jang. 2015. "Exiting and Returning to the Parental Home for Boomerang Kids." *Journal of Marriage and Family* 77 (3): 806–18. https://doi.org/10.1111/jomf.12183.

Schoen, Robert., Nancy S. Landale, and Kimberly. Daniels. 2007. "Family Transitions In Young Adulthood." *Demography* 44 (4): 807–20. https://doi.org/10.1353/dem.2007.0044.

Schweiger, Regev, Shachar Kaufman, Reijo Laaksonen, Marcus E. Kleber, Winfried März, Eleazar Eskin, Saharon Rosset, and Eran Halperin. 2016. "Fast and Accurate Construction of Confidence Intervals for Heritability." *American Journal of Human Genetics* 98 (6): 1181–92. https://doi.org/10.1016/j.ajhg.2016.04.016.

Sennet, Richard. 2006. *The Culture of the New Capitalism*. New Haven: Yale University Press.

Sennett, Richard. 1998. *Corrosion of Character. The Personal Consequences of Work in the New Capitalism*. New York: W.W. Norton.

Smock, Pamela J. 2000. "Cohabitation in the United States: An Appraisal of Research Themes, Findings, and Implications." *Annual Review of Sociology* 26: 1–20. https://doi.org/10.1146/annurev.polisci.6.121901.085546.

Sobotka, Tomáš. 2017. "Childlessness in Europe: Reconstructing Long-Term Trends Among Women Born in 1900–1972." In *Childlessness in Europe: Contexts, Causes, and Consequences*, edited by Michaela Kreyenfeld and Dirk Konietzka, 17–53. Frankfurt/Main: Springer International Publishing. https://doi.org/10.1007/978-3-319-44667-7_2.

Stone, Juliet, Ann Berrington, and Jane Falkingham. 2014. "Gender, Turning Points, and Boomerangs: Returning Home in Young Adulthood in Great Britain." *Demography* 51 (1): 257–76. https://doi.org/10.1007/s13524-013-0247-8.

Thornton, Arland, and Dimiter Philipov. 2009. "Sweeping Changes in Marriage, Cohabitation

and Childbearing in Central and Eastern Europe: New Insights from the Developmental Idealism Framework." *European Journal of Population* 25 (2): 123–56. https://doi.org/10.1007/s10680-009-9181-2.

Tropf, Felix, Nicola Barban, Melinda Mills, Harold Snieder, and Jornt J Mandemakers. 2015. "Genetic Influence on Age at First Birth of Female Twins Born in the UK, 1919–68." *Population Studies* 69 (2): 129–45.

Tropf, Felix, Gert Stulp, Nicola Barban, Peter M. Visscher, Jian Yang, Harold Snieder, and Melinda Mills. 2015. "Human Fertility, Molecular Genetics, and Natural Selection in Modern Societies." *PLoS ONE* 10 (6): 1–14. https://doi.org/10.1371/journal.pone.0126821.

Tropf, Felix, Renske M Verweij, Peter J van der Most, Gert Stulp, Andrew Bakshi, Daniel A Briley, Matthew Robinson, et al. 2017. "Hidden Heritability Due to Heterogeneity across Seven Populations." *Nature Human Behaviour* 1 (10): 757. http://biorxiv.org/content/early/2016/04/18/049163.abstract.

Trzaskowski, Maciej, Nicole Harlaar, Rosalind Arden, Eva Krapohl, Kaili Rimfeld, Andrew McMillan, Philip S Dale, and Robert Plomin. 2014. "Genetic Influence on Family Socioeconomic Status and Children's Intelligence." *Intelligence* 42: 83–88. https://doi.org/https://doi.org/10.1016/j.intell.2013.11.002.

Udry, Richard. 1996. "Biosocial Models of Low-Fertility Societies." *Population and Development Review* 22: 325–36.

Visscher, Peter M., Gibran Hemani, Anna A E Vinkhuyzen, Guo Bo Chen, Sang Hong Lee, Naomi R. Wray, Michael E. Goddard, and Jian Yang. 2014. "Statistical Power to Detect Genetic (Co)Variance of Complex Traits Using SNP Data in Unrelated Samples." *PLoS Genetics* 10 (4). https://doi.org/10.1371/journal.pgen.1004269.

Walsh, Froma. 2012. "The New Normal: Diversity and Complexity in 21st-Century Families." In *Normal Family Processes: Growing Diversity and Complexity*, edited by Froma Walsh, 4th ed., 3–28. Guilford Press.

White, Lynn. 1994. "Coresidence and Leaving Home: Young Adults and Their Parents." *Annual Review of Sociology* 20: 81–102. https://doi.org/10.1146/annurev.soc.20.1.81.

Winkle, Zachary Van. 2018. “Family Trajectories Across Time and Space: Increasing Complexity in Family Life Courses in Europe?” *Demography* 55 (1): 135–64. https://doi.org/10.1007/s13524-017-0628-5.

Winkle, Zachary Van, and Anette Fasang. 2017. “Complexity in Employment Life Courses in Europe in the Twentieth Century—Large Cross-National Differences but Little Change across Birth Cohorts.” *Social Forces* 96 (1): 1–30. https://doi.org/10.1093/sf/sox032.

Xu, Xiaohe, John P. Bartkowski, and Kimberly A. Dalton. 2011. “The Role of Cohabitation in Remarriage: A Replication.” *International Review of Sociology* 21 (3): 549–64. https://doi.org/10.1080/03906701.2011.625664.

Yang, Jian, Andrew Bakshi, Zhihong Zhu, Gibran Hemani, Anna A.E. Vinkhuyzen, Sang Hong Lee, Matthew R. Robinson, et al. 2015. “Genetic Variance Estimation with Imputed Variants Finds Negligible Missing Heritability for Human Height and Body Mass Index.” *Nature Genetics* 47 (10): 1114–20. https://doi.org/10.1038/ng.3390.

Yang, Jian, Beben Benyamin, Brian P McEvoy, Scott Gordon, Anjali K Henders, Dale R Nyholt, Pamela A Madden, et al. 2010. “Common SNPs Explain a Large Proportion of Heritability for Human Height.” *Nature Genetics* 42 (7): 565–69. https://doi.org/10.1038/ng.608.Common.

Yang, Jian, S. Hong Lee, Michael E. Goddard, and Peter M. Visscher. 2011. “GCTA: A Tool for Genome-Wide Complex Trait Analysis.” *American Journal of Human Genetics* 88 (1): 76–82. https://doi.org/10.1016/j.ajhg.2010.11.011.

Yang, Jian, Jian Zeng, Michael E. Goddard, Naomi R. Wray, and Peter M. Visscher. 2017. “Concepts, Estimation and Interpretation of SNP-Based Heritability.” *Nature Genetics* 49 (9): 1304–10. https://doi.org/10.1038/ng.3941.

Zeman, Kryštof, Éva Beaujouan, Zuzanna Brzozowska, and Tomáš Sobotka. 2018. “Cohort Fertility Decline in Low Fertility Countries: Decomposition Using Parity Progression Ratios.” *Demographic Research* 38 (1): 651–90. https://doi.org/10.4054/DemRes.2018.38.25.

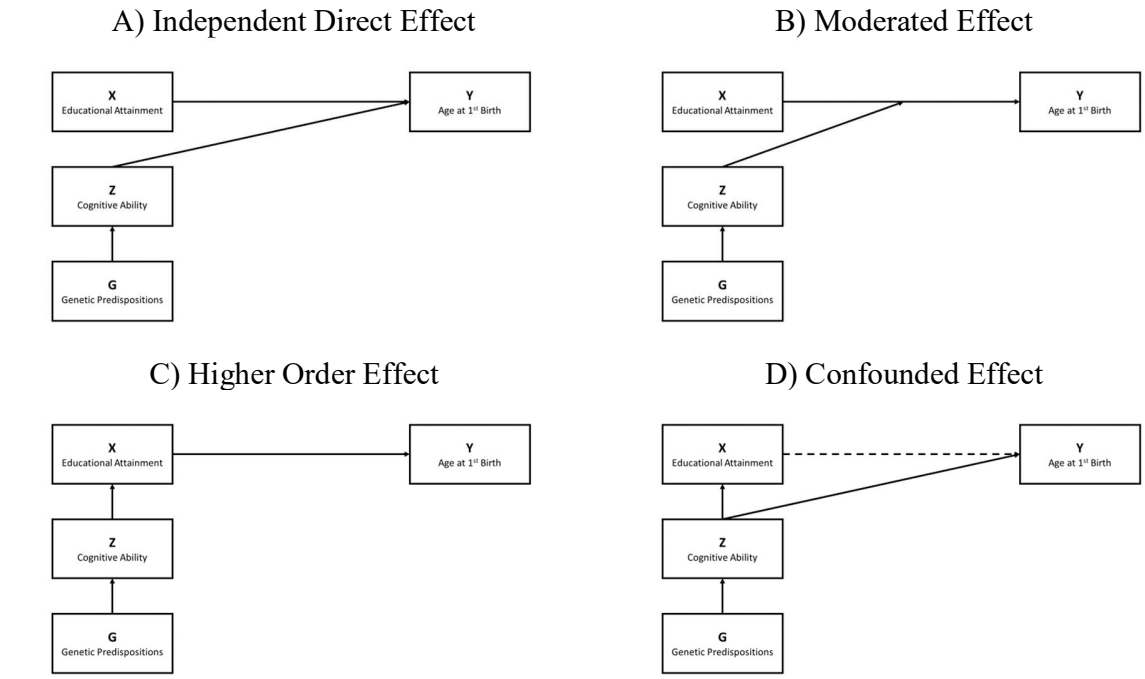
Zeng, Yi, S Philip Morgan, Zhenglian Wang, Danan Gu, and Chingli Yang. 2012. “A Multistate Life Table Analysis of Union Regimes in the United States: Trends and Racial Differentials,

1970–2002." *Population Research and Policy Review* 31 (2): 207–34. https://doi.org/10.1007/s11113-011-9217-2.

Zorlu, Aslan, and Clara H. Mulder. 2011. "Ethnic Differences in Leaving Home: Timing and Pathways." *Demography* 48 (1): 49–72. https://doi.org/10.1007/s13524-010-0012-1.

Figures

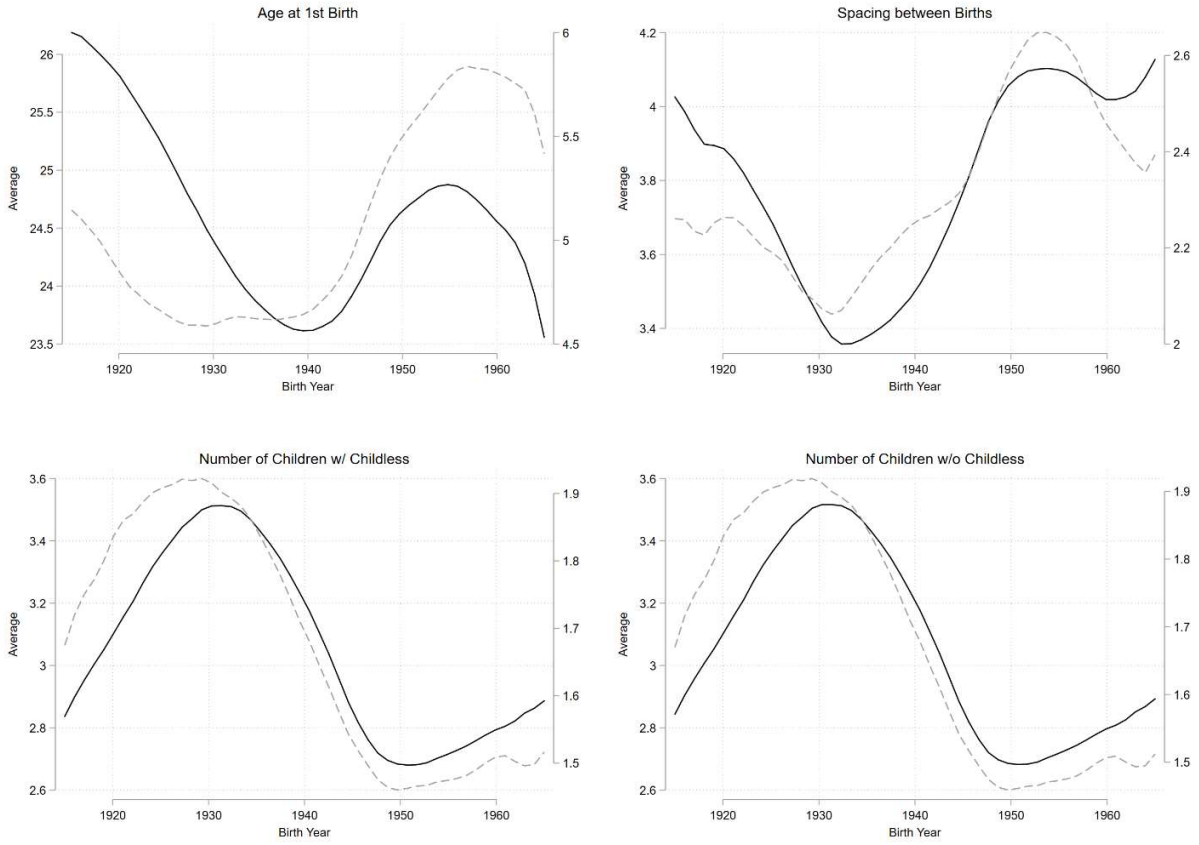
Figure 1: Ideal Type Pathways for Genetic Effects on Sociological Relationships through Intermediate Phenotypes



Note: Figure adapted from Freese (2008)

Figure 2: Sample Means and Standard Deviations of Family Demographic Indicators by Birth Year

Fertility Indicators



Note: Averages in black and standard deviations in grey displayed by birth year using local polynomial smoothing.

Figure 2 continued: Marital Indicators

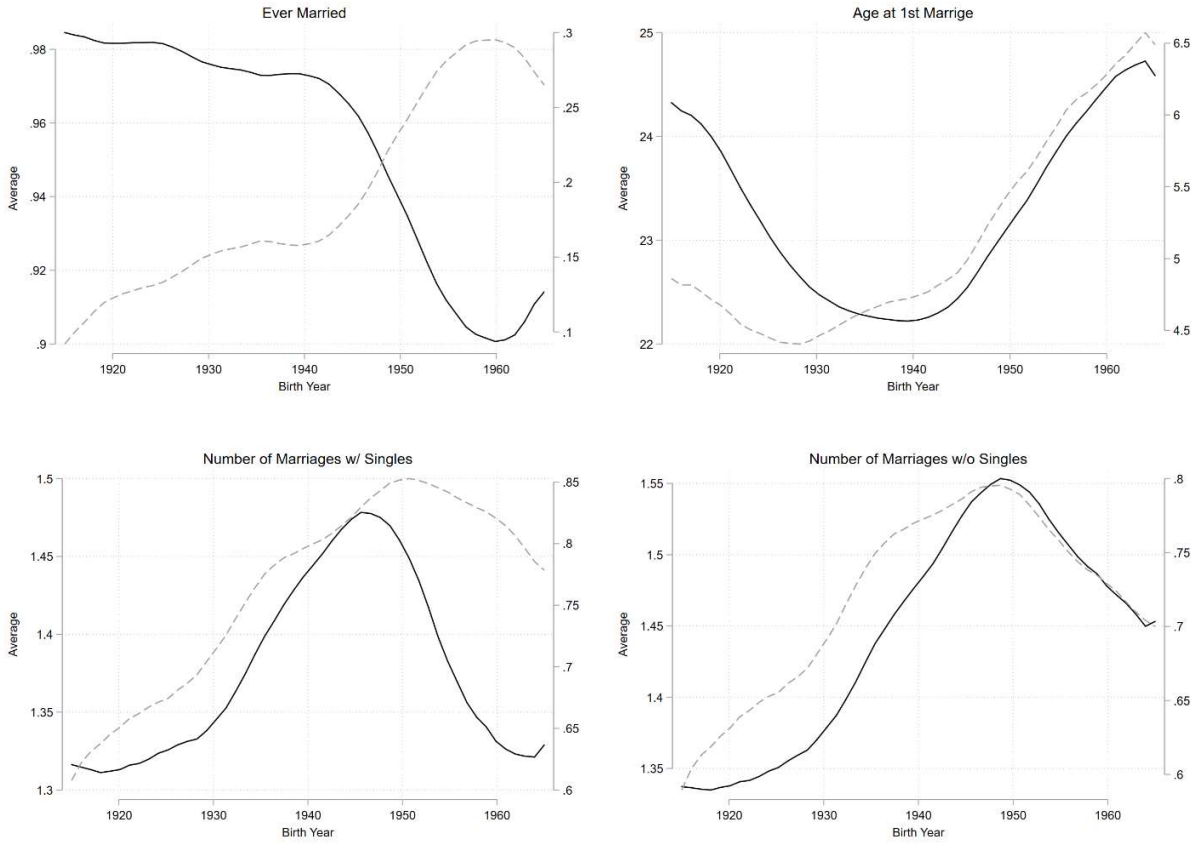


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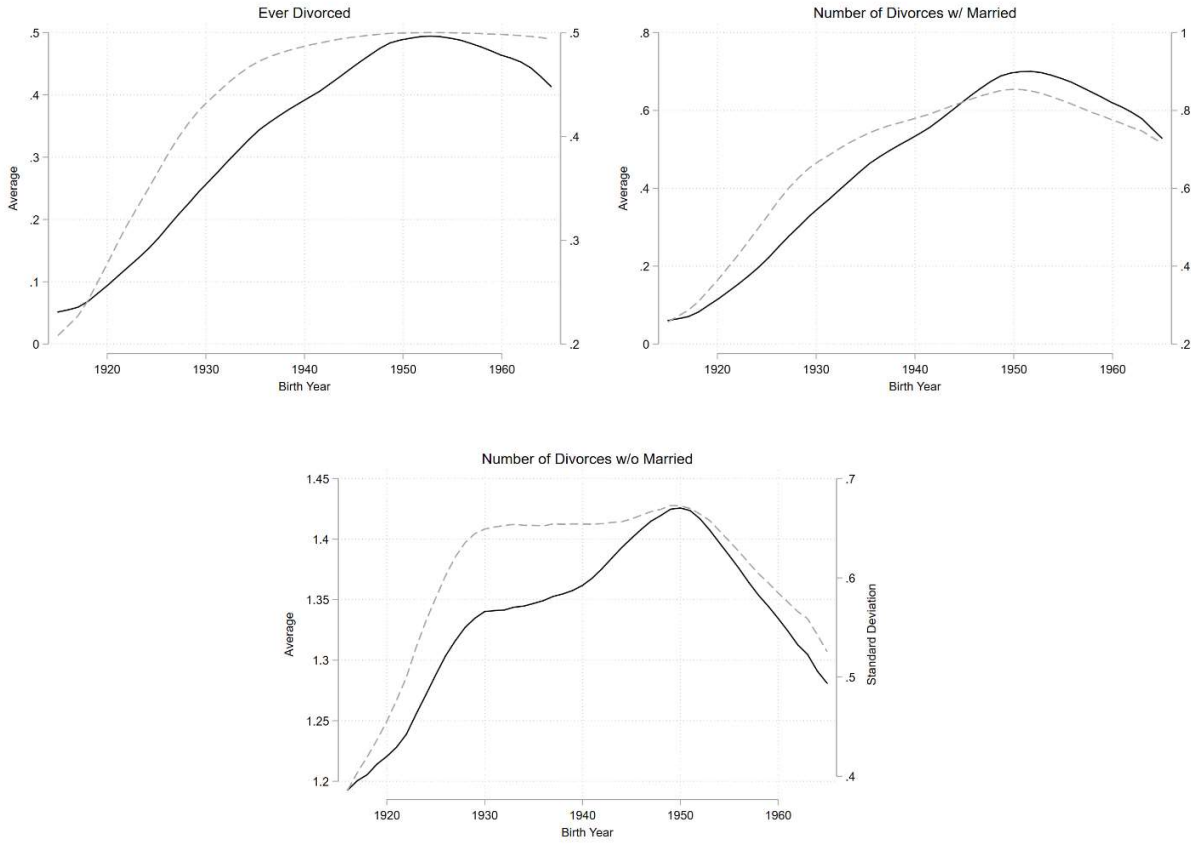
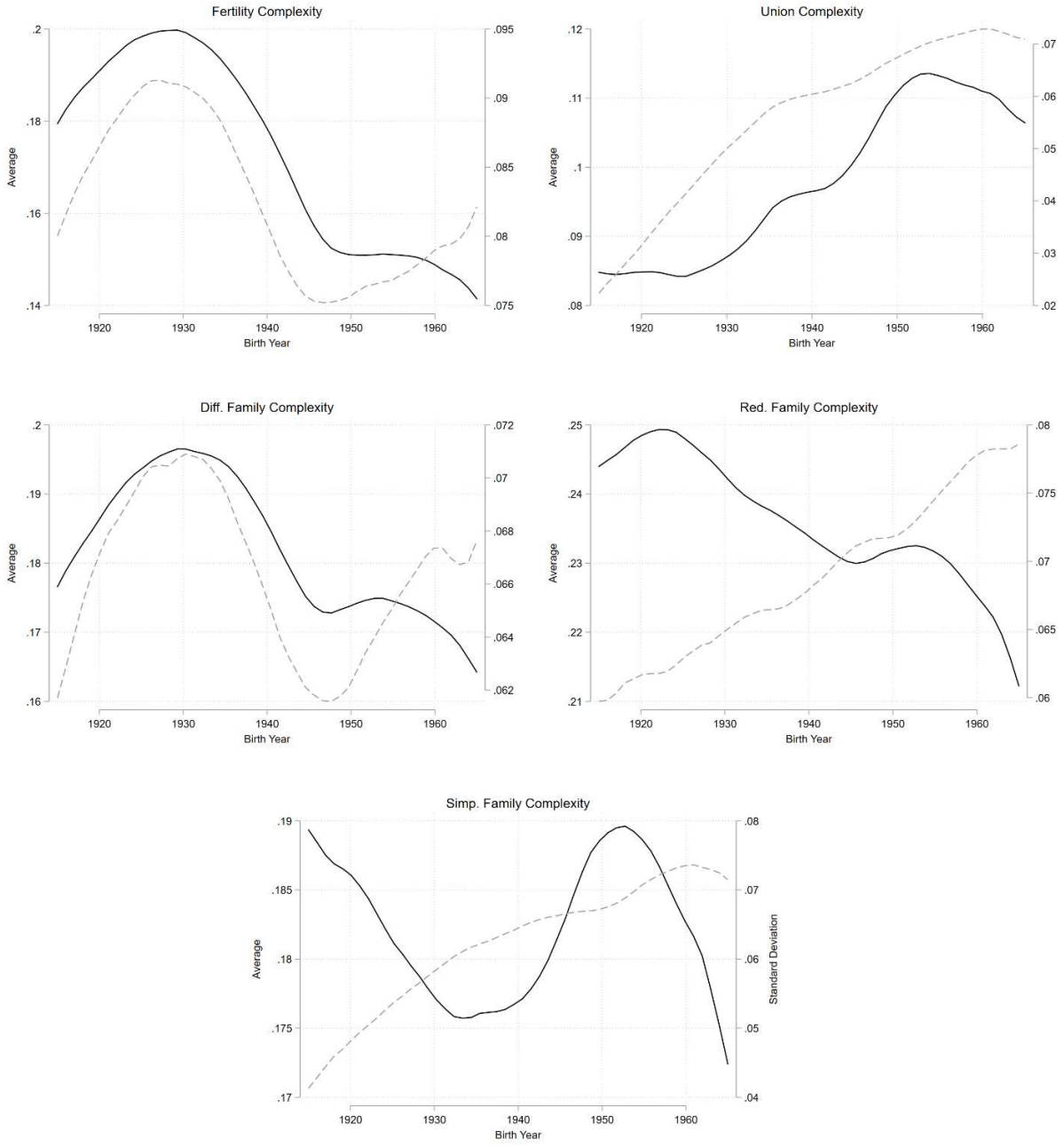
Figure 2 continued: Divorce Indicators



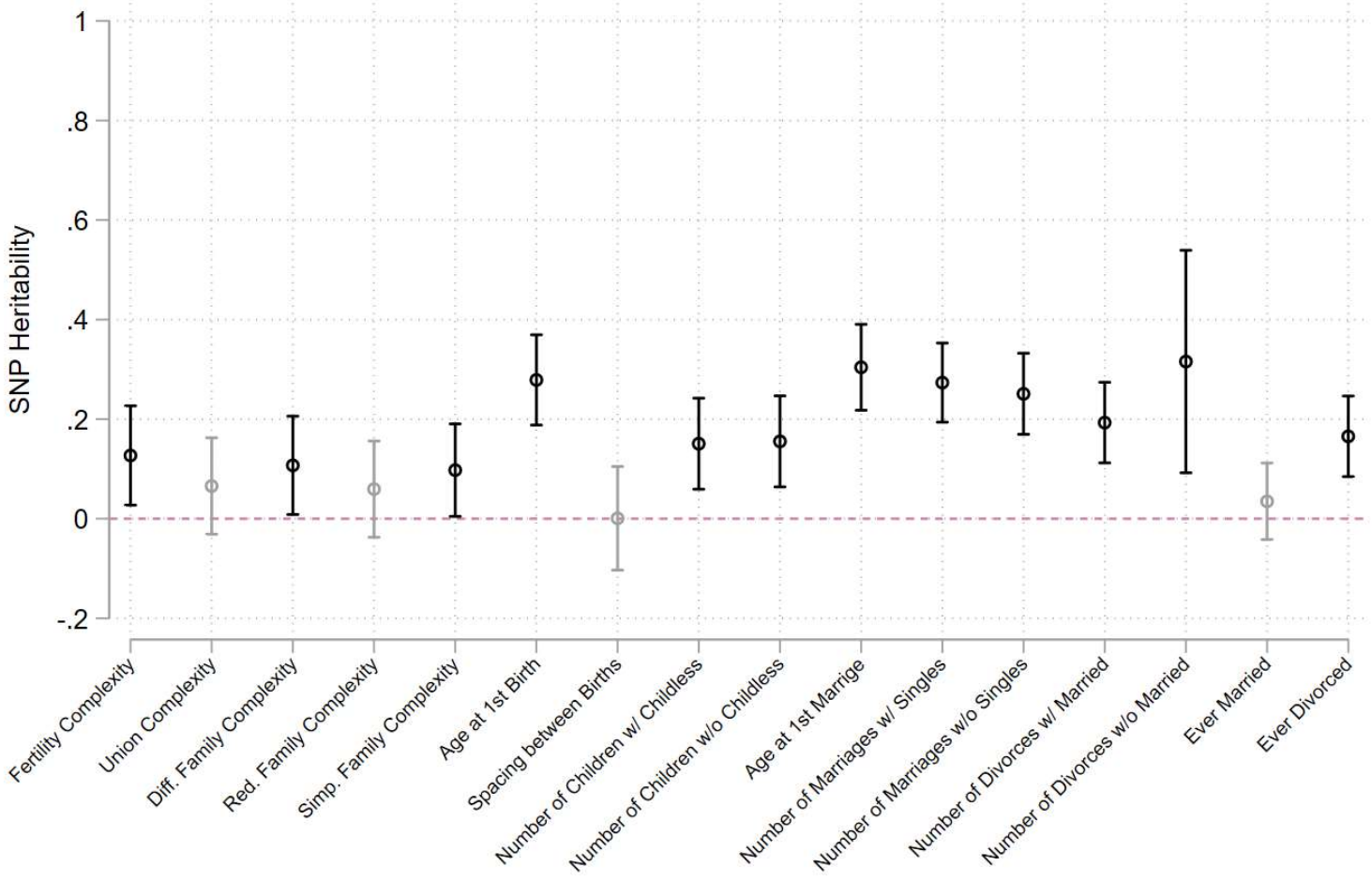
Note: Averages in black and standard deviations in grey displayed by birth year using local polynomial smoothing.

Figure 2 continued: Sequence Complexity



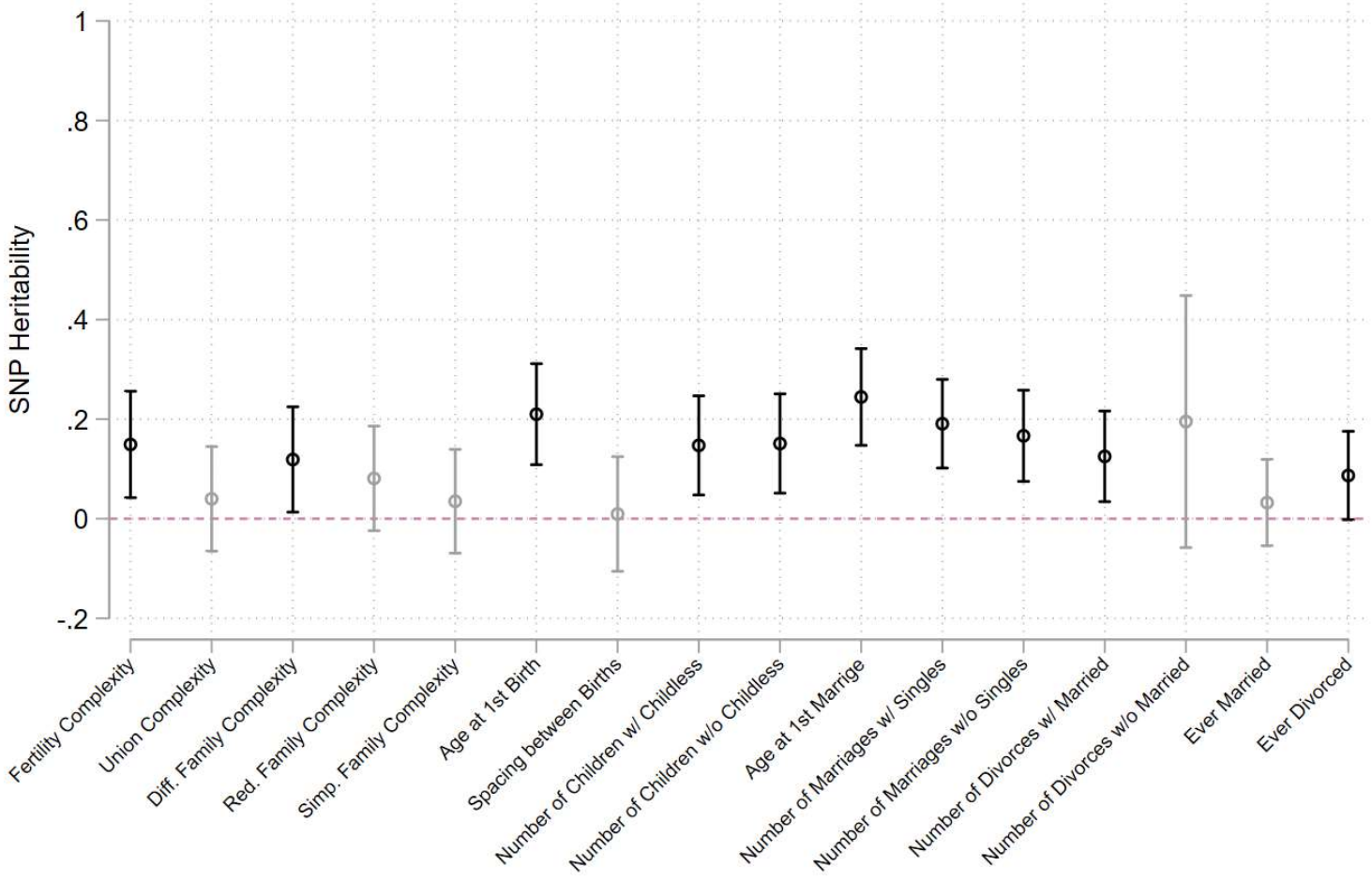
Note: Averages in black and standard deviations in grey displayed by birth year using local polynomial smoothing.

Figure 3: Unadjusted SNP Heritability Estimates



Note: Heritability estimates and 95% confidence intervals from unadjusted GREML models displayed. See Table A1 for details.

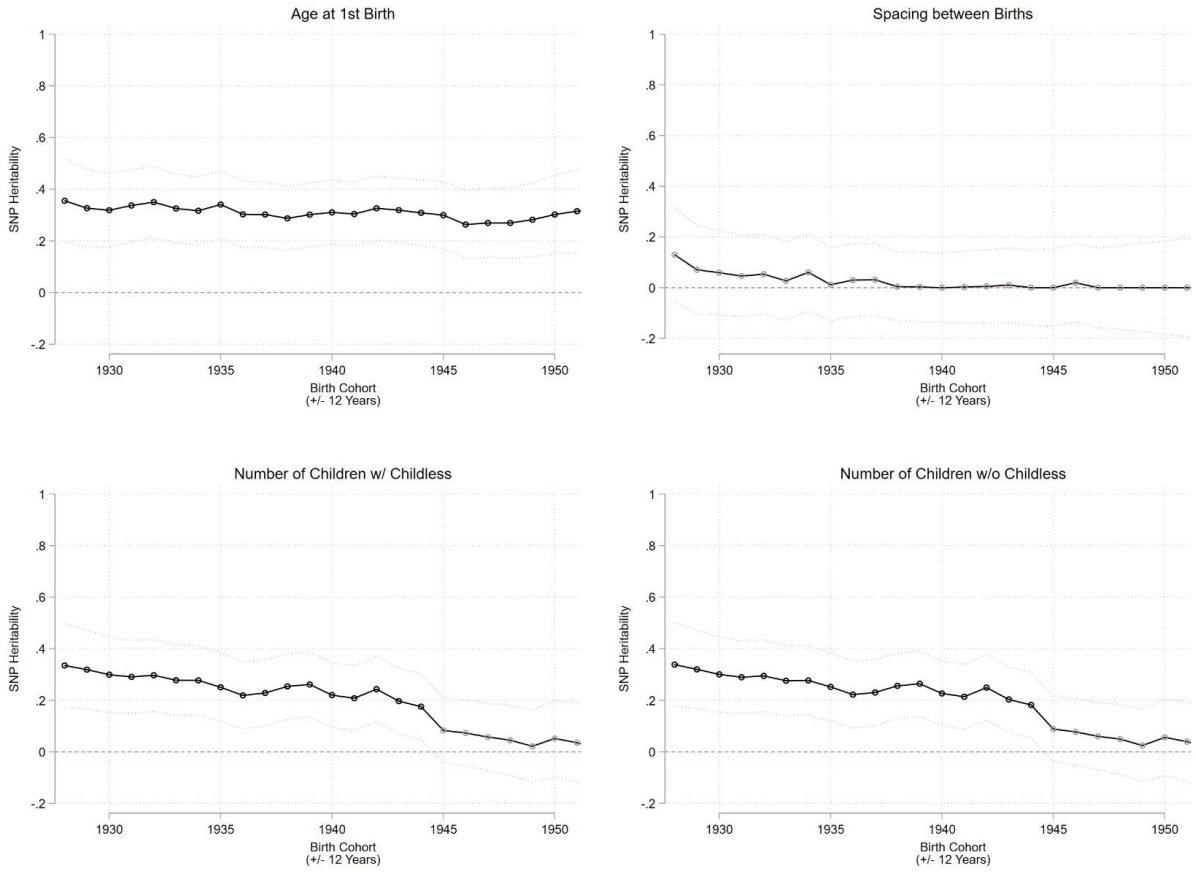
Figure 4: Adjusted SNP Heritability Estimates



Note: Heritability estimates and 95% confidence intervals from GREML models adjusted for 10 ancestral principle components, gender, and birth year displayed. See Table A2 for details.

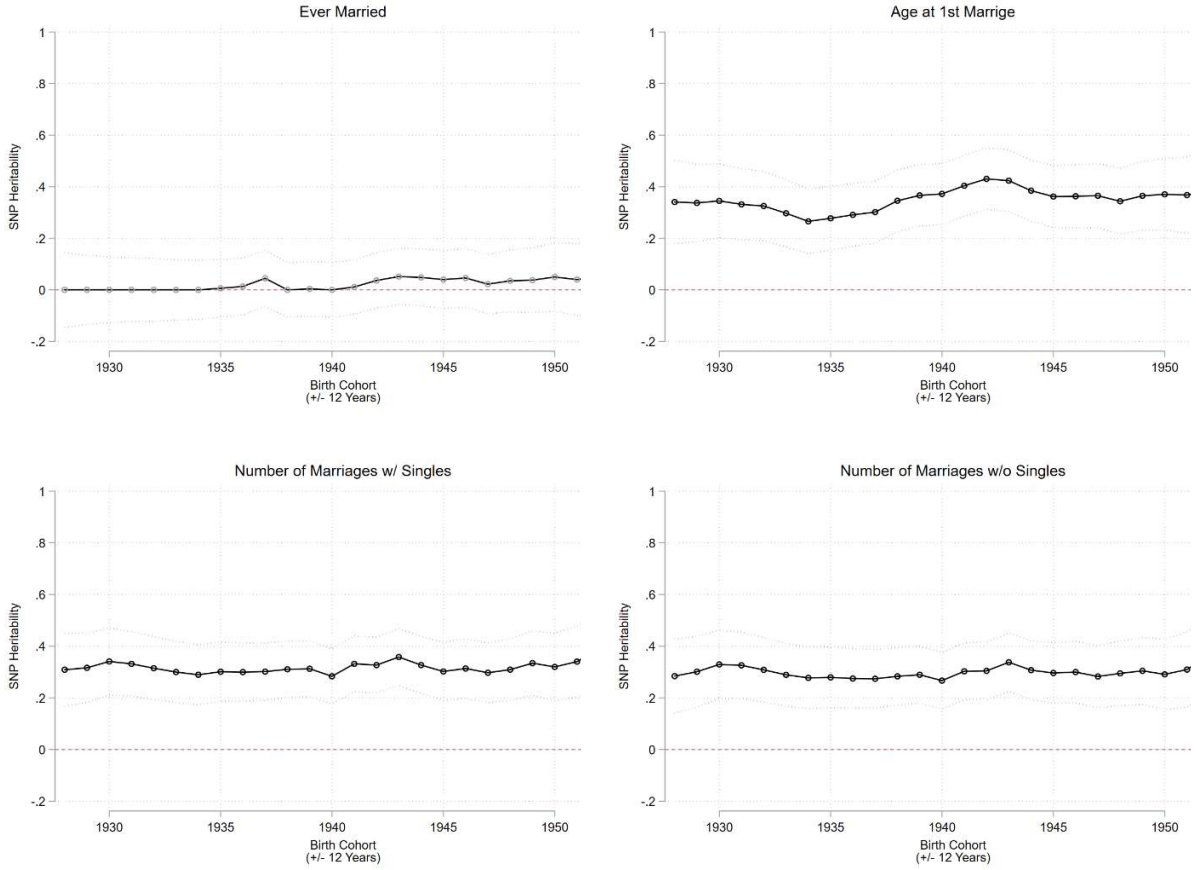
Figure 5: Unadjusted SNP Heritability Estimates by 25-Year Birth Cohorts

Fertility Indicators



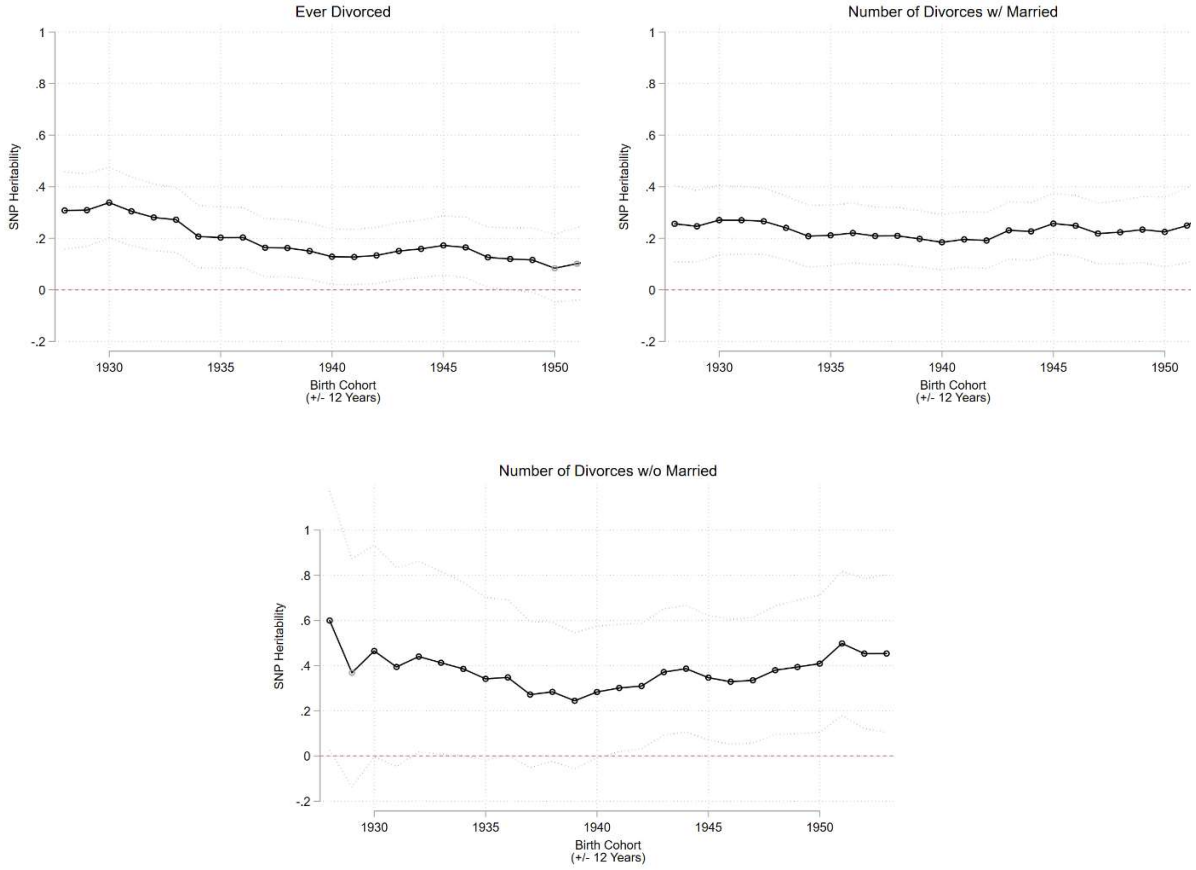
Note: Heritability estimates and 95% confidence intervals from unadjusted GREML models displayed from 25-year birth cohorts. Black markers indicate that heritability estimates are statistically different from zero ($p < 0.05$).

Figure 5 continued: Marital Indicators



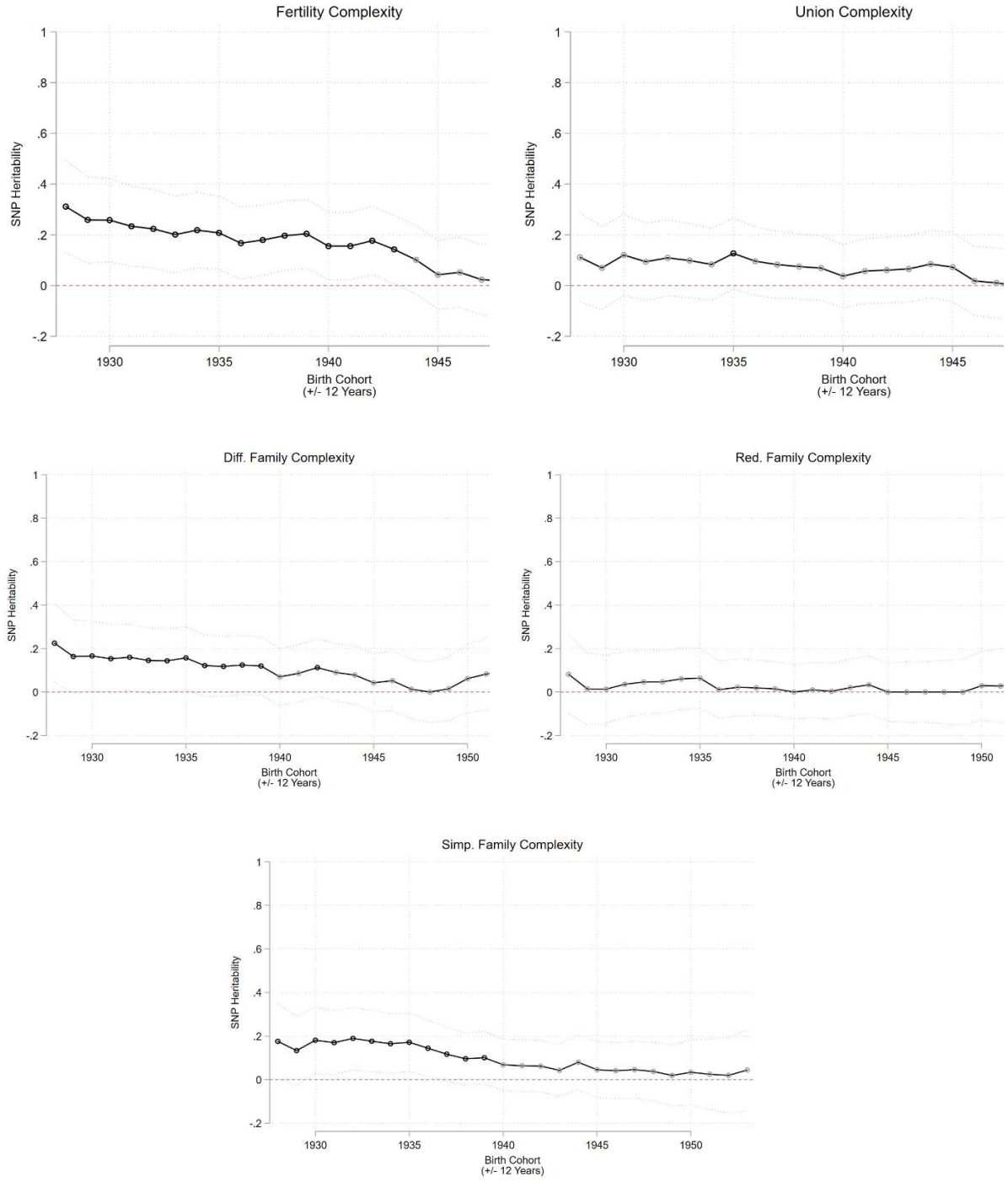
Note: Heritability estimates and 95% confidence intervals from unadjusted GREML models displayed from 25-year birth cohorts. Black markers indicate that heritability estimates are statistically different from zero ($p < 0.05$).

Figure 5 continued: Divorce Indicators



Note: Heritability estimates and 95% confidence intervals from unadjusted GREML models displayed from 25-year birth cohorts. Black markers indicate that heritability estimates are statistically different from zero ($p < 0.05$).

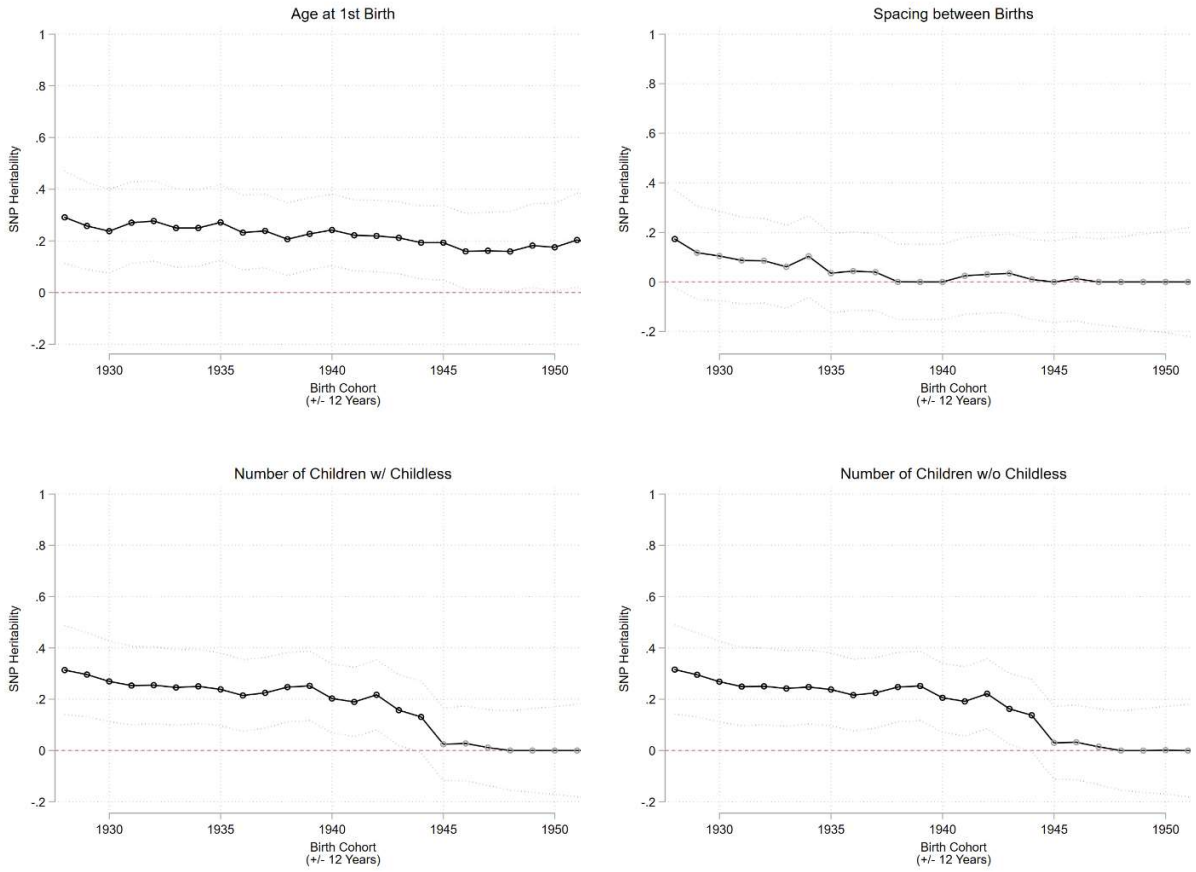
Figure 5 continued: Sequence Complexity



Note: Heritability estimates and 95% confidence intervals from unadjusted GREML models displayed from 25-year birth cohorts. Black markers indicate that heritability estimates are statistically different from zero ($p < 0.05$).

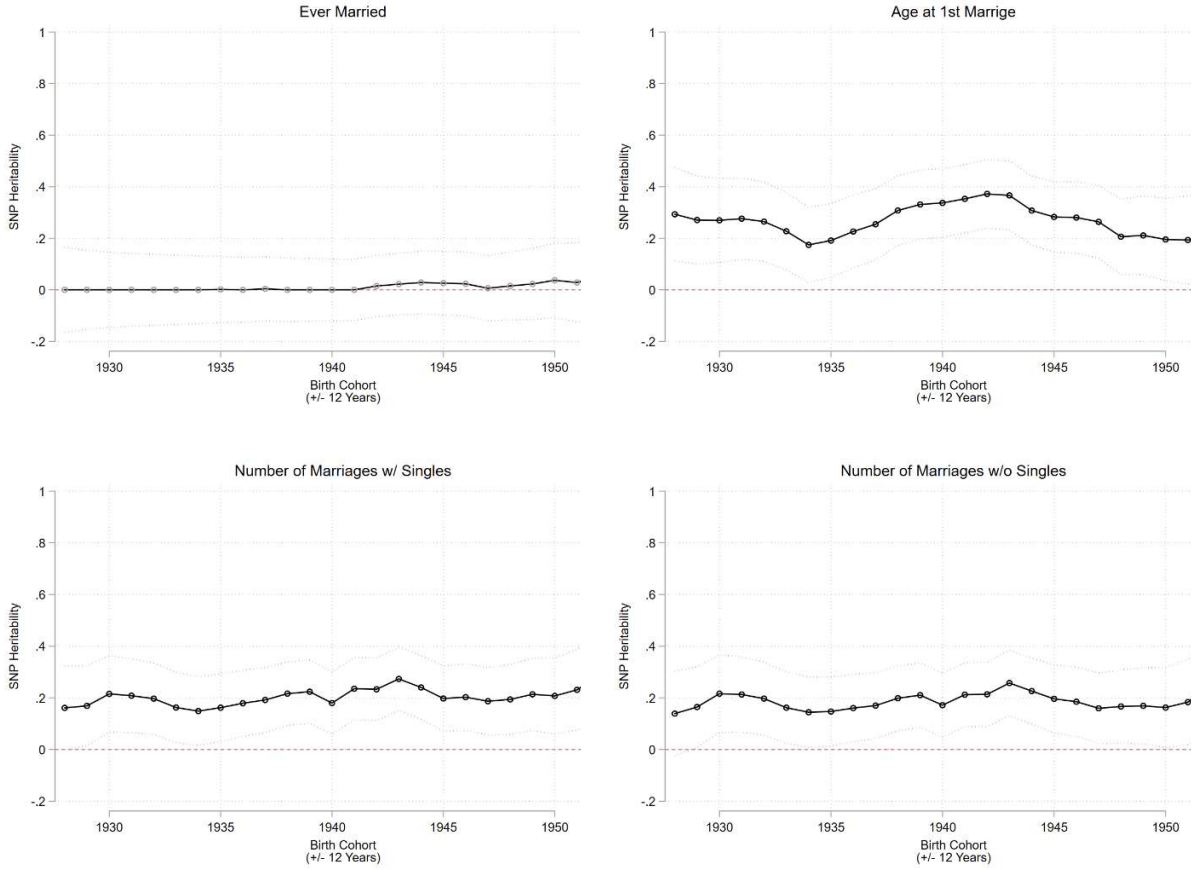
Figure 6: Adjusted SNP Heritability Estimates by 25-Year Birth Cohorts

Fertility Indicators



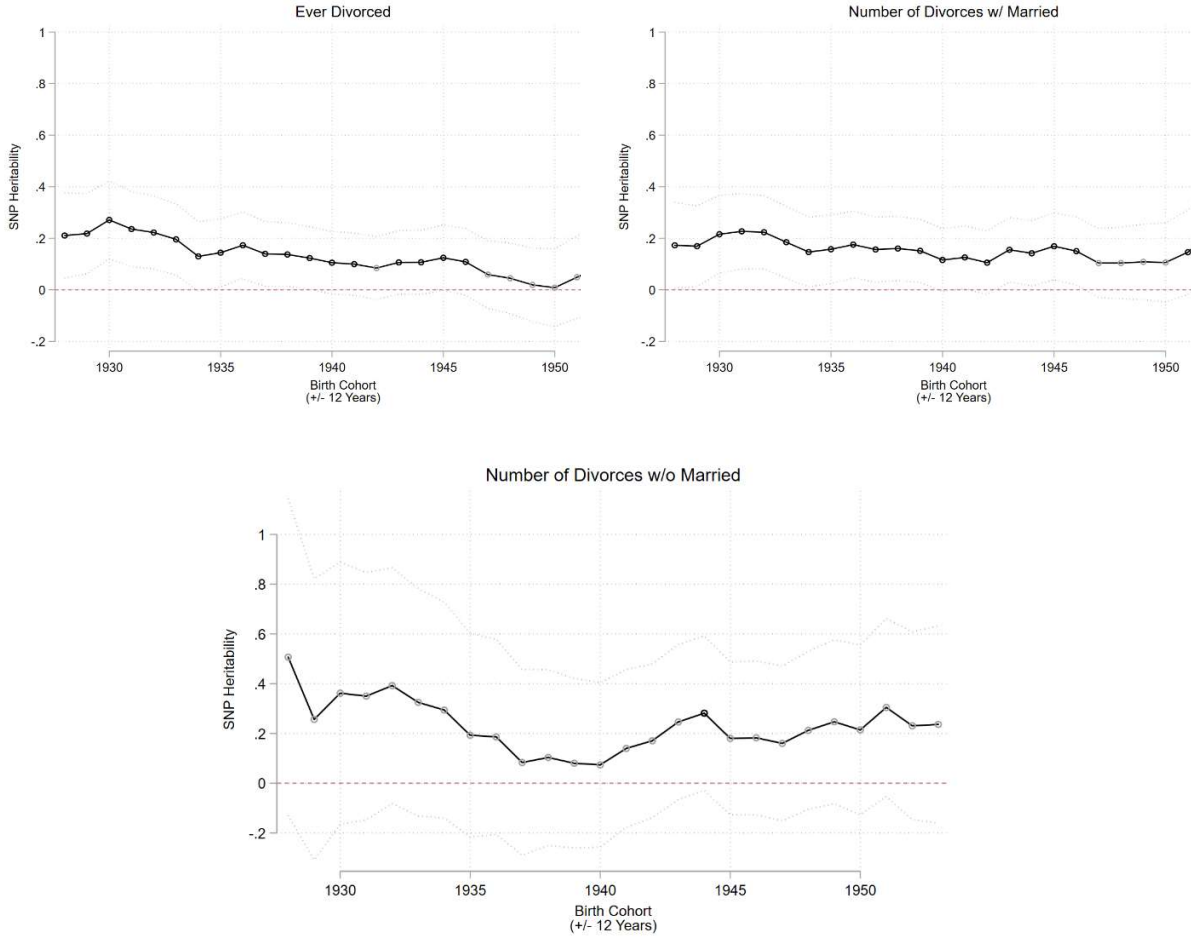
Note: Heritability estimates and 95% confidence intervals from GREML models adjusted for 10 ancestral principal components, gender, and birth year displayed from 25-year birth cohorts. Black markers indicate that heritability estimates are statistically different from zero ($p < 0.05$).

Figure 6 continued: Marital Indicators



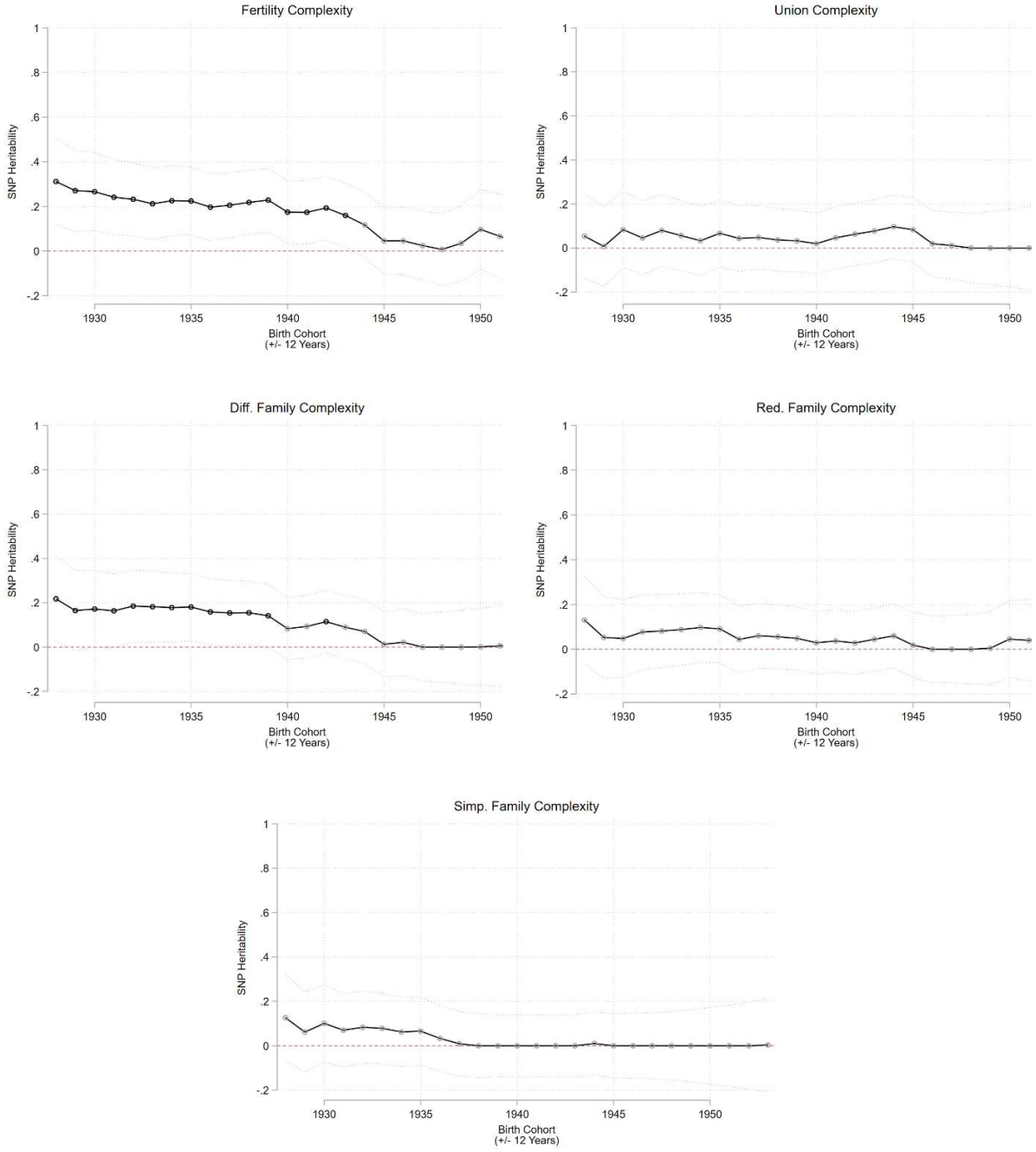
Note: Heritability estimates and 95% confidence intervals from GREML models adjusted for 10 ancestral principal components, gender, and birth year displayed from 25-year birth cohorts. Black markers indicate that heritability estimates are statistically different from zero ($p < 0.05$).

Figure 6 continued: Divorce Indicators



Note: Heritability estimates and 95% confidence intervals from GREML models adjusted for 10 ancestral principal components, gender, and birth year displayed from 25-year birth cohorts. Black markers indicate that heritability estimates are statistically different from zero ($p < 0.05$).

Figure 6 continued: Sequence Complexity



Note: Heritability estimates and 95% confidence intervals from GREML models adjusted for 10 ancestral principal components, gender, and birth year displayed from 25-year birth cohorts. Black markers indicate that heritability estimates are statistically different from zero ($p < 0.05$).