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Is the Association Between Education and Fertility Postponement Causal? The Role of Family Background Factors

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RUNNING HEAD: Education, Fertility Postponement, and Causality

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Abstract A large body of literature has demonstrated a positive relationship between education and age at first birth. However, this relationship may be partly spurious because of family background factors that cannot be controlled for in most research designs. We investigate the extent to which education is causally related to later age at first birth in a large sample of female twins from the United Kingdom (N = 2,752). We present novel estimates using within–identical twin and biometric models. Our findings show that one year of additional schooling is associated with about one-half year later age at first birth in ordinary least squares (OLS) models. This estimate reduced to only a 1.5-month later age at first birth for the within–identical twin model controlling for all shared family background factors (genetic and family environmental). Biometric analyses reveal that it is mainly influences of the family environment—not genetic factors—that cause spurious associations between education and age at first birth. Last, using data from the Office for National Statistics, we demonstrate that only 1.9 months of the 2.74 years of fertility postponement for birth cohorts 1944–1969 could be attributed to educational expansion based on these estimates. We conclude that the rise in educational attainment alone cannot explain differences in fertility timing between cohorts.

Keywords Fertility postponement; Education; Fixed effects; Twins; United Kingdom

Introduction

A large body of literature from the United States and Europe has documented a positive relationship between higher educational attainment and later age at first birth of women (e.g., Gustafsson et al. 2002; Lappegård and Rønsen 2005; Martin 2000). Researchers have argued that this relationship is causal and can account for the rise in the mean age at first birth during the educational expansion of the second half of the twentieth century in Western countries (Balbo et al. 2013; Ní Bhrolcháin and Beaujouan 2012). However, research has also cast doubt on the idea of a causal effect of education on the age at first birth, suggesting that family background characteristics (social and/or genetic factors) cause spurious associations between educational attainment and fertility timing of women (Neiss et al. 2002; Rodgers et al. 2008). Social stratification research has indicated considerable similarity between parents, children and siblings in education and socioeconomic attainment (Branigan et al. 2013; van Doorn et al. 2011) and in fertility behavior (Murphy 1999; Rijken and Liefbroer 2009). The similarity/intergenerational transmission may be due to parental socioeconomic resources and/or socialization processes (Nisén and Myrskylä 2014; Rijken and Liefbroer 2009) but also to shared genetic dispositions given the sufficient evidence for a genetic component for both outcomes (Branigan et al. 2013; Mills and Tropf 2016).

Our study contributes to the literature addressing the following three questions. First, does education indeed have a causal effect on age at first birth? Second, to what extent can the postponement of age at first birth during the second half of the twentieth century be explained by the simultaneous educational expansion? Third, to what extent are (social) environmental family background and/or genetic factors responsible for the observed relationship between education and age at first birth?

To answer these questions, we present within-twin models (with identical twins) and

engage in biometric modeling of the link between education and age at first birth in order to disentangle a possible causal relationship from genetic and environmental confounders in a large sample of twins from the UK. Next to quasi-experimental study designs, within-twin designs offer an approach to causality. Quasi-experimental designs (McCrary and Royer 2011; Skirbekk et al. 2006) use exogenous factors, such as changes in compulsory school laws, which influence education but are supposed to be independent from fertility to investigate the causal effect of education on fertility. The within-twin approach, in contrast, controls for all factors shared among siblings, including genetic material, by using identical twins as a natural experiment (Kohler et al. 2011). The experiment is that the twins differ in levels of education but not in their genetic makeup or their family environment. To the extent that the family background (social and/or genetic factors) is a common cause for education and age at first birth, standard regression models result in biased estimates, whereas within-twin estimates remain unbiased (Amin et al. 2015).

The within–identical twin approach is of particular interest for three reasons. First, as mentioned earlier, we expect that most of the unobserved factors that are important for education and fertility timing are related to the family of origin, which can be controlled for in twin studies (Branigan et al. 2013; Mills and Tropf 2016). Second, the within–identical twin design can use variation across all levels of education, whereas (for example) quasi-experimental studies on changes in school laws have mostly relied on variation at a particular (mostly lower) level of education (see also Amin et al. 2015). Third, comparing fraternal and identical twins allows a quantification of the contribution of genetic and environmental influences for a specific trait, as in classic twin and behavior genetics modeling (Rodgers et al. 2001, 2007). Kohler and colleagues (2011) recently developed a model that formally integrates the two approaches: the within–identical twin regression model and the behavior genetics model. This so-called ACE-

beta model represents a parsimonious solution to identify causality in a bivariate association and simultaneously evaluate the importance of genetics and shared environmental influences from the family.

Demographic research should strive to connect the individual and population level (Billari 2015). We therefore combine our findings at the micro level with nationally representative data from the Office for National Statistics (ONS) to evaluate whether the causal effect of education explained age at first birth trends at the population level. This goal is important, not only to understand past fertility trends, but also to assess the potential to anticipate future fertility development based on changes in educational level given that education has gained importance in fertility forecasting (Lutz et al. 2014). Furthermore, in the UK, the trend in age at first birth was U-shaped during the mid-century (Hobcraft 1996), whereas educational expansion increased steadily (Oreopoulos 2006). These differential trends motivate a closer inspection of the relationship in different birth cohorts. We therefore also present analyses separately for birth cohorts born before and after World War II (WWII).

The current study builds on and extends previous research in a number of ways. First, we apply the new ACE-beta model to study the link between education and age at first birth in a large sample of 2,752 twins from the TwinsUK, which is the largest adult twin register in the United Kingdom (Moayyeri et al. 2013), offering a larger sample size than was available to earlier research.

Second, previous investigations of the causal link between education and age at first birth in family designs showed mixed results, some confirming a causal relationship (Amin and Behrman 2014; Nisén et al. 2013) and some not (Neiss et al. 2002; Rodgers et al. 2008). However, these studies also used different analytical strategies, applying within–identical twin models (Amin and Behrman 2014), standard behavior genetics models (Nisén et al. 2013), and

complex structural equation models (SEM) (Neiss et al. 2002; Rodgers et al. 2008). To decipher this puzzle, we present within–identical twin models as well as the ACE-beta model, which simultaneously estimates the causal effect and quantifies family environmental and genetic influences on the education-fertility relationship in a parsimonious SEM (Kohler et al. 2011).

Third, the UK is a particularly interesting case to study. Comparative studies within Europe suggest that the effect of education on age at first birth is relatively strong in the UK (Gustafsson et al. 2002; Rendall et al. 2005). A recent investigation by Ní Bhrolcháin and Beaujouan (2012) showed that in the UK between 1980 and 1999, 57 % of the postponement of the mean age at first birth could be attributed to longer educational enrollment, and the remainder was due to additional, post-educational postponement effects—that is, if we endorse the said causal relationship between education and age at first birth.

Background

Education and Fertility Timing

Women's mean age at first birth steeply increased by as many as 4–5 years during the second half of the twentieth century throughout Europe and the United States, and was accompanied by an overall increase in educational attainment (Mills et al. 2011). For example, in the UK between 1980 and 2000, the average age at first birth as well as age of leaving full-time education increased by 1.4 years (Ní Bhrolcháin and Beaujouan 2012). By comparing three UK birth cohort studies (1946, 1958, 1970), Joshi (2002) showed that the percentage of women who were mothers by age 26 dropped from 81 % for those born in 1946 to 30 % for the 1970 cohort. In all three birth cohorts, women with a tertiary educational qualification were about half as likely to be mothers as women with no educational qualifications. In the most recent 1970 cohort, only 1 in 10 higher-educated women were mothers at age 26, compared with 6 in 10 for women without qualifications.

A number of causal mechanisms have been put forward to explain the association between educational level and fertility timing (for an overview, see Balbo et al. 2013; Mills et al. 2011). First, being enrolled in education itself may lead to postponement of childbearing because combining student and mother roles is difficult, given that they both entail time-intensive tasks. Women might delay childbearing because of the high costs of child rearing and limited resources while enrolled in school, and social norms might discourage parenting before the end of education (Hoem 2000; Lappegård and Rønsen 2005). Second, education may increase people's aspirations and ability to pursue a career. Thus, women might further postpone childbearing until they are well established in their careers, also implying higher opportunity costs of the transition to parenthood given their greater human capital (e.g., Liefbroer and Corijn 1999). Furthermore, education might also change values and orientations toward more individualistic lifestyles that seek fulfillment in life without children (Lesthaeghe 1995; van de Kaa 1987). Therefore, both educational enrollment and attainment may lead to postponing childbearing (and reducing fertility). Reverse causality may play a role, too, because an early age at first birth may disrupt further education, although this is presumably mainly the case for unwanted teenage childbearing (Nisén and Myrskylä 2014).

This line of reasoning along with the often-observed micro-level association led researchers to hypothesize that educational expansion in the UK explains the postponement of age at first birth (Ní Bhrolcháin and Beaujouan 2012). However, such an interpretation is not straightforward for at least three reasons. First, the observed macro-level association between education and age at first birth changed during the past century; the simultaneous rise in age when leaving education and age at first birth occurred only after WWII. Second, in addition to the overall rise in education, a number of competing explanations exist for the rise in (female) age at first birth in developed countries. Third, as we touch on earlier, the relationship between

education and age at first birth may be partly spurious. We now discuss these issues in more detail.

Changing Macro Trends

Comparative studies in Europe have indicated variability in the association between education and age at first birth (Gustafsson et al. 2002; Rendall et al. 2005). More specifically, the association in the UK between education and age at first birth at the population level changed during the past century. Age at first birth decreased after WWII, with an accompanying steep increase in fertility—the so-called Baby Boom. At the same time, educational attainment increased. The UK Education Acts lifted the school-leaving age from 12 to 14 in 1918 and then from age 14 to 15 in 1944. As shown by Oreopoulos (2006), within three years during the period 1945–1947, the percentage of 14-year-olds leaving school fell from approximately 57 % to less than 10 %. Only later that century, since the 1960s, has age at first birth increased— during the so called Baby Bust. Education further expanded during the second half of the twentieth century with higher proportions of tertiary education particularly of women and trends in age when leaving education and age at first birth ran parallel (Mills et al.2011). Given the reversing trends throughout the century, we hypothesize that a strict causal logic that longer educational enrollment leads to later age at first birth may not apply.

Fertility Postponement: Alternative Explanations

Several alternative explanations mainly related to the introduction of the Pill have been put forward to explain fertility postponement in developed countries during the twentieth century. Rising female labor force participation (e.g., Rindfuss et al. 2007), ideational shifts in norms and values in sexual behavior and family planning of the second demographic transition (Lesthaeghe 1995; van de Kaa 1987), and increasing economic uncertainty (Andersson 2000) are associated with an increasing age at first birth and might (partially) account for the postponement. The

introduction of the Pill as an effective contraception is seen as an important trigger of these mechanisms.

In the UK, especially changes in family norms and values, as well as economic uncertainty and the introduction of the Pill provide alternative explanations for fertility postponement during the last 50 years, which may operate together with or independent of the educational expansion (Hobcraft 1996; Murphy 1993). To evaluate the extent to which the educational expansion is responsible for fertility postponement and to avoid ecological fallacy, we assess the causal effect of education on age at first birth at the individual level and use these estimates for predictions at the population level on nationally representative and independently collected data.

Critique of Education and Fertility Link

The association between education and age at first birth might be in part spurious because unobservable factors influencing age at first birth may also be related to education. In particular, family background may play a pivotal role as a socializing agent, a source of resources and support, and a means for transmitting genes. Studies have repeatedly demonstrated that education (Branigan et al. 2013) and fertility (Mills and Tropf 2016) are associated with both the family environment and genetic factors. Therefore, the family environment and genetic factors may (partially) explain the observed association between education and age at first birth.

Family background—for example, the socioeconomic status (SES) of the parents—defines the resources and opportunities to remain in school longer and to financially compensate for children. The status of parents can shape consumption and status aspirations of children who aim for higher education and social status in advance of family formation (Thornton 1980). More generally, the socialized striving for autonomy might lower ambitions to build a family while increasing investments in education and a career (Rijken and Liefbroer 2009; Scott 2004).

Biological predispositions shared among family members and transmitted through genes can influence career and family trajectories. The timing of first attempts to get pregnant measured in retrospective interview is linked to one's genetic makeup (Rodgers et al. 2001). In addition, genes influence educational attainment (Branigan et al. 2013; Rietveld et al. 2013) and fertility timing (Nisén et al. 2013; Tropf et al. 2015a). However, the extent to which genetic effects for both outcomes are shared remains an open question. In the following sections, we discuss previous studies that investigated the influence of (bio-)social family factors on education and fertility.

Biometric Approaches for the Education and Age at First Birth Link

Four studies have considered both genetic and (socially) environmental family influences on the relationship between education and age at first birth. The first two investigations focused on the role of education as a possible mediator between cognitive ability and age at first birth. One used 813 pairs of relatives from the NLSY born between 1958 and 1965 (Neiss et al. 2002), and the second used 621 Danish twin pairs born between 1931 and 1952 (Rodgers et al. 2008). These two studies extended the classic twin model by introducing cognitive ability as preceding education in the causal chain at the cost of additional assumptions on shared genetic and environmental effects across traits. Both studies found that the observed mediating link of education became nonsignificant after they controlled for genetic and environmental influences from within the family. These two studies did not find genetic influences on age at first birth and thus no genetic correlation between both outcomes, in contrast with the general pattern (Mills and Tropf 2016). They concluded that education may not directly delay childbearing but rather that individual differences leading to higher cognitive ability and higher education inhibit fertility—and these differences arise between, not within, families.

The third study estimated bivariate biometric models that decomposed the (co)variance in education and age at first birth into latent genetic and environmental factors in a sample of 4,228

Finnish twins born in 1950–1957 (Nisén et al. 2013). For women, shared environmental factors of the twins as well as environmental factors unique to an individual independently explained part of the observed covariance between education and age at first birth. Influences unique to an individual can be interpreted as a causal effect (D'Onofrio et al. 2013) but might also result from third factors influencing both outcomes at the same time, such as the partner (Kohler and Rodgers 2003).

Finally, Amin and Behrman (2014) used a within–identical twin design on a sample of 628 identical twins from the Minnesota Twin Registry. They found that one year of additional education leads to fertility postponement of approximately one year. Furthermore, they did not find evidence for effects of family background on the association between education and age at first birth: that is, results in standard OLS regression models were nearly identical to the within-twin models.

In the current study, we first focus on the causal relationship between education and age at first birth and apply within-twin regression models. We present a within–identical twin estimate in a different context than Amin et al (2015) did and for a larger sample than they used. Second, we use our findings to project the trend in age at first birth at the population level and contrast the projection with the actual trend in age at first birth. Third, we extend these models according to Kohler et al. (2011) by integrating the bivariate twin model. Compared with previous mediation models (Neiss et al. 2002; Rodgers et al. 2008), the ACE-beta model needs fewer identifying assumptions, and all paths that we introduce to the model are identified and can be estimated.

Methods

Data

We used information on twins of the TwinsUK registry. TwinsUK was originally established at the St. Thomas Hospital London in 1992 and gathered information on the life course of identical (monozygotic (MZ)) and fraternal (dizygotic (DZ)) twins (Moayyeri et al. 2013). Zygosity was established using standardized questions and confirmed by DNA genotyping in uncertain cases. As of this writing, the registry contains information on approximately 12,000 individuals. We limited the analysis to same-sex female individuals in complete twin pairs because the TwinsUK contains few male twins (<15 %), making a comparable analysis for men infeasible. Furthermore, we excluded women younger than 40 at the last observation to avoid an overrepresentation of young mothers and to limit right-censoring of women who did not have children at the time of the last observation. Valid information on zygosity, fertility, and education was available for 3,856 women in 1,928 twin pairs. Because of right-censoring of one or both twins in a twin pair, the sample was further reduced to 2,752 women in 1,376 twin pairs. For robustness checks, we also applied Cox regression models that include childless women (see Online Resource 1, section S1, Tables S1 and S2).

To compare the TwinsUK sample with a representative sample and to describe trends, we used two additional sources from the ONS. For education, we used data from the General Household Survey (GHS) rounds from 2000 to 2006 (N = 35,435, birth cohorts 1931–1970). The GHS is an annual continuous survey of the population in private households in Great Britain. To describe age at first birth, we used estimates from the ONS (Office National Statistics 2013) because historical GHS fertility measures were limited to married individuals.

Age at First Birth

The measure for age at first birth was based on information from two questionnaires in the TwinsUK. First, the Main Questionnaire, which was administered between 1995 and 2001, contains an inventory of the years of birth of up to 10 children. Second, age at first birth was assessed directly with a question from the 2004 questionnaire: "How old were you when you had your first live birth?" For individuals who participated in multiple waves, we used the earliest reported age.

Education

Education was measured as the age at leaving full-time education, assessed directly with the following question: "At what age did you leave full-time education?" Most previous work has measured education in categories and imputed the corresponding years of schooling (e.g., Amin and Behrman 2014; Rodgers et al. 2008). In the UK, educational categories are difficult to order vertically because of differences in educational systems and migration across countries within the UK, especially because qualifications are so numerous and because their names and content change often (Jenkins and Sabates 2007). Therefore, both TwinsUK and the GHS provide the age when leaving full-time education as a valid and reliable measure across data sources.

Analysis

The analysis proceeded in two steps. First, we used OLS regression and subsequently within-twin models (fixed-effect models) to estimate the causal effect of education on age at first birth (AFB). Although within-twin models have a long history in economics (Rosenzweig and Wolpin 1980) and psychology/behavioral genetics (D'Onofrio et al. 2013; Neale and Cardon 1992), they have seldom been applied in demography (Amin and Behrman 2014). A comparison of the OLS and the within-twin approach indicates the extent to which family background leads to a spurious association between education and age at first birth. In a second step, we estimated the ACE-beta

model to investigate the extent to which genetic and/or environmental effects lead to a spurious association between education and age at first birth.

OLS and Fixed-Effects Models

The OLS models included birth year and birth year squared of a twin (*i*) nested in a twin pair (*j*) to allow for a curvilinear trend in fertility timing across birth cohorts. Furthermore, we controlled for zygosity measured as the expected genetic relatedness between DZ twins (0.5) and MZ twins (1) and estimated robust standard errors to correct for the dependency structure of the twins (Eq. 1). We are interested in the effect of education (β_1).

$$afb_{ij} = \beta_0 + \beta_1 \times edu_{ij} + \beta_2 \times birth\ year_j + \beta_3 \times birth\ year_j^2 + \beta_4 \times zygosity_j + \varepsilon_{ij}. \quad (1)$$

The OLS model allows a naive estimation of the association between education and age at first birth. As detailed elsewhere (Amin et al. 2013; Kohler et al. 2011), standard OLS ignores at least three additional sources of variation: (1) the shared environment in a family *j* specific for fertility (C_j), such as family norms; (2) the individual additive genetic endowment (A_{ij}); and (3) the unmeasured unique environment (E_{ij}), which includes measurement error and individual specific effects for fertility, such as the influence of partner characteristics. These effects might influence the estimation of β_1 if they are correlated with education.

The within-twin models included fixed effects per family, which capture all (observed and unobserved) factors shared among the twins. We can discriminate between fraternal (DZ) and identical (MZ) twins. DZ twins share, on average, 50 % of their segregating genetic material, and MZ twins are genetically identical. Thus, the DZ fixed-effects model controls for one-half of all additive genetic effects (similar to a sibling fixed-effects model), and the MZ fixed-effects model controls for all genetic effects. In other words, the MZ fixed-effects model controls for all factors shared amongst siblings, including birth year and zygosity, but also common (family)

environment (C_j) and genetic effects; thus, $D(C_{ij}) = 0$ *and* $D(A_{ij}) = 0$. The equation for the MZ fixed effects is as follows:

$$\Delta(afb)_{ij}^{mz} = \beta_1 \times \Delta(edu)_{ij}^{mz} + \Delta\varepsilon_{ij}^{mz}. \quad (2)$$

We applied both the OLS regression and the twin fixed-effects models to the pooled sample of twins, as well as for DZ and MZ twins separately. The comparison, particularly of the DZ and MZ models, provides the first insights as to whether differences between the naive OLS estimates and the fixed-effects estimates are due to shared environmental and/or genetic factors. If genetic effects are important, we expect a stronger reduction in the effect of education for MZ twins than for DZ twins. The remaining link between education and age at first birth can be interpreted as causal. The effect is consistent if unique environmental influences important for education are independent of age at first birth (and vice versa) (Kohler et al. 2011).

Bivariate Genetic Modeling

Twin studies are no longer uncommon in social science research (Branigan et al. 2013; Freese 2008; Kohler et al. 1999, 2011; Miller et al. 2010; Mills and Tropf 2016; Neiss et al. 2002; Nisén et al. 2013; Rodgers et al. 2001, 2008; Tropf et al. 2015a). Twin studies are based on the comparison of MZ and DZ twins to quantify genetic and environmental influences. The degree to which MZ twins are more similar than DZ twins is assumed to reflect only genetic influences. Twin models are typically SEMs that decompose the observed variance into three components: (1) additive genetic effects resulting from the sum of genetic effects from the whole genome (*A*); (2) environmental effects resulting from environmental influences shared between twins of a pair (*C*); and (3) nonshared environmental effects resulting from the unique environment of an individual (*E*) (which includes measurement error).

Three assumptions of the twin model need to be briefly addressed. The first is that MZ and DZ twins share their environment to the same extent—that is, the equal-environment assumption (EEA). Although this assumption has been criticized (e.g., Horwitz et al. 2003), empirical evidence supports the validity of this assumption (Conley et al. 2013), including fertility studies (Felson 2014). Second, the twin model assumes that there is no assortative mating within the population with respect to the outcome of interest. A violation of this assumption (Domingue et al. 2014) would result in an underestimate of genetic influences. The third assumption is that there are no nonadditive genetic effects (gene-gene, and gene-environment interaction effects).

Following the same logic as in classic twin studies, it is possible to estimate the extent to which genetic and/or environmental factors are important for the covariance between two different outcomes. If education of twin 1 correlates with age at first birth of twin 2, then part of the covariance runs in families. A higher correlation is higher among MZ twin pairs than among DZ twin pairs would indicate shared genetic effects for both outcomes. We estimated the ACE-beta model (Kohler et al. 2011), which represents an extension of a bivariate Cholesky decomposition (for details, see Loehlin 1996; Rodgers et al. 2007). The bivariate Cholesky decomposition estimates two equations: (1) one for the first variable—in our case, education (Eq. (3)); and (2) one for the second variable—in our case, age at first birth. Education of twin (*i*) is a function of

$$edu_i = a_{edu,edu}{}' \left(A_{ij}^{edu}\right) + c_{edu,edu}{}' \left(C_j^{edu}\right) + e_{edu,edu}{}' \left(E_{ij}^{edu}\right). \quad (3)$$

Indexes indicate whether the effects are on education (*edu*), on age at first birth (*afb*), or shared for both. Lower indexes indicate whether the effects are common to both twins (*j*) or different (*ij*).

The variance in education is therefore decomposed into additive genetic effects ($a_{edu,edu}$), shared environmental effects ($c_{edu,edu}$), and residual variance/measurement error ($e_{edu,edu}$).

Investigating the association between education and AFB, we formulate a second equation, which decomposes the variance in AFB into parts that are common with the genetic variance components of education: for example, $a_{edu,afb}$ ´ $\left(A_{ij}^{edu}\right)$; the shared environmental component ($c_{edu,afb}$ ´ $\left(C_{j}^{edu}\right)$); and parts that are unique to AFB, caused by A^{afb}, C^{afb}, and E^{afb}. In contrast to the classic bivariate Cholesky model, the ACE-beta model assumes that unique environmental sources of variance/measurement error for education (E_{ij}^{edu}) are uncorrelated with AFB ($e_{edu,afb} = 0$). Instead, the link between education and AFB is expressed by the causal link represented in a fixed-effects regression only within MZ twin pairs $\beta_1 \times \Delta\left(E\right)_{ij}^{mz}$ (Eq. (4)).

$$\begin{aligned} afb_{ij} = {} & \beta_1 \times \Delta\left(edu\right)_{ij}^{mz} + a_{edu,afb} \times \left(A_{ij}^{edu}\right) + c_{edu,afb} \times \left(C_{j}^{edu}\right) \\ & + a_{afb,afb} \times \left(A_{ij}^{afb}\right) + c_{afb,afb} \times \left(C_{j}^{afb}\right) + e_{afb,afb} \times \left(E_{ij}^{afb}\right). \end{aligned} \tag{4}$$

In this article, we visualize the standardized estimates in the form of a correlated factors model (Loehlin 1996; Neale and Cardon 1992) to facilitate interpretation. We present all results from these models and the standardization of the estimates in Online Resource 1. The genetic contribution to education and AFB is typically expressed as narrow-sense heritability (h^2), which is the proportion of variance due to additive genetic effects. In a parallel way, we can compute the variance component for shared (C) and unique environmental influences/measurement error (E). Importantly, we estimate the extent to which genetic and shared environmental influences that are important for education and age at first birth correlate and explain the observed correlation between education and age at first birth.

We used Stata 12 to estimate the OLS and fixed-effect models. The developers, Kohler et al. (2011), provided us with the OpenMX R-function to estimate the ACE-beta model.

Robustness

The main analysis presented in this article includes only twin pairs in which both twins had a child because the bivariate biometric models cannot deal with nonlinear outcomes. We tested the robustness of our results by including right-censored observations using Cox regression models. We present Cox regression models with and without stratification by family to replicate the OLS and within-twin models for the full sample (Allison and Christakis 2006). See Online Resource 1 for details.

Results

Descriptive Findings

Table 1 shows the descriptive statistics of the variables of interest separately for DZ and MZ twins. The twins in the sample were born on average just after WWII, mean age at first birth was almost 26, and the mean age at leaving education was approximately 17 years for both kinds of twins. Most importantly for the biometric models, there were only minor differences in average or standard deviation of the outcomes of interest between DZ and MZ twins. Also of note, only 27 individuals (~1 %) had their first birth before leaving education and only 74 (~2.7 %) had their first birth one year after leaving education, such that the temporal succession suggests that education influences age at first birth and not vice versa.

< Table 1 about here >

The two main concerns about the within-twin approaches are (1) that variation of the independent variables within twin pairs is low and largely due to measurement error, and (2) that twin data are not representative. First, Table 1 shows the mean absolute differences in education within twin pairs. These differences were substantial, at 1.33 for DZ and 0.88 for MZ twins,

suggesting that sufficient variation remained even within MZ twin pairs. The smaller differences for MZ than DZ twins suggest a genetic component underlying education. Second, TwinsUK is considered to be representative for the singleton population in the UK (Andrew et al. 2001; Moayyeri et al. 2013). Figure 1 shows the smoothed trends in age when leaving education and age at first birth for the TwinsUK sample (top panel) and the representative ONS/GHS data (lower panel). The general level and trends for the TwinsUK sample are similar to those of the ONS/GHS data, which increases our confidence in using the TwinsUK sample. However, the mean age at first birth in the TwinsUK sample (25.83) was about a year and a half later than in the ONS data (24.35). There was no difference in age at leaving education between the TwinsUK (16.97) and GHS (16.92) estimates.

Moreover, it is clear from Fig. 1 that the mean age of leaving education rose steadily throughout the twentieth century, whereas the mean age at first birth followed a U-shaped pattern in the UK. Most previous research focused on trends in both outcomes during the second half of the twentieth century, concluding that the rise in age at finishing education led to a postponement of childbearing. However, the figure reveals a discontinuity in the association, which challenges the idea of a causal relationship between education and age at first birth.

< Figure 1 about here >

The Causal Effect of Education on Age at First Birth

Our first research question asks whether education has a causal effect on age at first birth. Model 1 in Table 2 estimates the well-established naive OLS estimate of the effect of education on age at first birth at 0.44 (standard error (SE) = 0.04) in the present sample. This means that women who stayed one year longer in education had their first childbirth approximately one-half year later, on average. The effect holds independent of cohort effects given that we controlled for birth

year and its square. Models 3 and 5 repeat the same model but now for subsets of MZ and DZ twins respectively. The estimates of MZ and DZ twins are nearly identical.

Now we turn to the twin fixed-effects models to disentangle the causal effect from potential family confounders. Model 2 in Table 2 shows the pooled twin fixed-effects approach, which controls for all shared environmental factors among siblings and partly their genes. The effect of education on age at first birth falls to 0.11 (SE = 0.06) but remains significantly different from 0 at the 5 % level in a one-sided test. Model 4 depicts the MZ fixed-effects estimate (0.12, SE = 0.09), which controls for all factors that vary between twins, including all shared environmental and genetic differences (given that MZ twins are genetically identical). The twin fixed-effects estimate for DZ twins is depicted in Model 6 (0.11, SE = 0.08). This estimate controls for all factors that vary at the family level; genetic differences among DZ twins are only partially controlled for. The pooled, MZ fixed-effects, and DZ fixed-effects estimates are all similar in size (approximately 0.11), but the two twin fixed-effects analyses by zygosity are not significantly different from 0, likely due to reduced power of the twin fixed-effects approach. This suggests a reduction in the causal effect of education on age at first birth to around one quarter of the effect estimated from the OLS model (.12 / .44) to only 1.4 months (.12 × 12 = 1.44). These findings suggest that a large part of the education effect on age at first birth can be attributed to family background factors (genetic and environmental).

< Table 2 about here >

To check the robustness of these findings when also including twin pairs where one or both women did not have children at the time of last observation and those younger than 40, Table S2 in Online Resource 1 provides the results from the (stratified) Cox regression models (N = 4,398). Results follow the same pattern as those from the regression models except that the

reduction in the estimated effect of education for the within-twin analyses appears to be less strong. The probability of having a child decreases with each additional year of education by ~10 %, and this falls to ~3 % to 4 % in the stratified Cox regression models.

Our second research question asks to what extent the educational expansion during the second half of the twentieth century can explain the simultaneous overall postponement in age at first birth. Figure 2 depicts the simultaneous rise in age of leaving education (dashed line) and age at first birth (solid line) for cohorts born between 1944 and 1967. These cohorts started childbearing after the 1960s and therefore can be considered the main drivers of the fertility postponement. Age at first birth in 1967 in the UK (26.00) was approximately 2.74 years later than in 1944 (23.26). Figure 2 furthermore shows the simultaneous rise in age of leaving full-time education of approximately 1.3 years across these birth cohorts. A straightforward macro-explanation might consequently conclude that 48 % (= 1.3 / 2.74) of the 2.74 years of fertility postponement can be attributed to a rise in educational enrollment. However, as shown in the regression models, for each year of additional education, individuals postpone ~5.3 months in the OLS model and ~1.4 months in the within-twin models. Note that we use a reduced MZ estimate of 1.2 (0.10 × 12 = 1.20) months because we restrict the analysis to MZ twins born after 1944 (not listed). Across the birth cohort born between 1944 and 1967, therefore, only between 21 % (= 1.3 × 0.44 / 2.74; OLS models) and 4.8 % (= 1.3 × 0.10 / 2.74) of the observed postponement in age at first birth can be directly related to the educational expansion. Figure 2 shows the predicted average age at first birth based on the OLS (short dash-dot) and MZ twin fixed-effects results (long dash-dot), as well as the explained (dark grey) and unexplained (light grey) trends in age at first birth across the second half of twentieth century and in medium grey the difference between OLS and MZ twin fixed-effects model. The OLS specification can be considered the

upper bound of the explained trend in AFB by education, and the MZ fixed-effects model can be considered the lower bound.

< Figure 2 about here >

The estimates in Table 2 are based on birth cohorts born between 1919 and 1969. As shown in Fig. 1, the correlation of the trends in education and age at first birth is reversed at the population level and perhaps also at the individual level. Therefore, the causal effect of education might have become stronger in more recent cohorts. We additionally estimated models that allow for different educational effects for birth cohorts born before and after 1944 (the turning point in the trend in age at first birth) to investigate this issue (see Table S3 in Online Resource 1). However, we do not find such differences.

The Role of Genes and the Environment as Family Background Factors

The reduction of the estimate of the causal effect of education on age at first birth in OLS versus MZ twin fixed-effects models leaves us with the third research question, which asks to what extent this reduction is due to unobserved shared environmental influences and/or unobserved genetic endowments that are common to both outcomes. To answer this question, we present the ACE-beta model, which simultaneously estimates the direct causal link between education and age at first birth based on the MZ twin fixed-effects estimator and the genetic and shared environmental contribution. Figure 3 presents the correlated factor model (see Online Resource 1, Table S4 and section S2 for the untransformed SEM estimates and the transformation).

< Figure 3 about here >

The estimates are well in line with previous studies (Branigan et al. 2013; Mills and Tropf 2016). The heritability of education was .46, meaning that 46 % of the variance in education was associated with additive genetic differences. The shared environmental influences account also for 23 % of the variance; and the unique environment, which includes measurement error,

accounts for 31 %. The heritability of age at first birth is estimated to explain 32 % of the variance in age at first birth. Shared environmental effects are smaller (.12) but significant, and unique environmental effects/measurement error make up the largest part (.56).

All variance components are statistically significant. We also see the correlation of genetic and environmental influences across outcomes. If the genetic/environmental correlation between two traits is 1, all genetic/environmental variance in trait 1 and 2 has a common base. If the correlation is 0, the genetically/environmentally based variance between traits 1 and 2 are independent. Shared environmental effects on education and age at first birth correlate to 1, meaning that there are no shared environmental effects for age at first birth independent of shared environmental influences on education. Also a model constraining shared environmental effects for education and age at first birth to be the same has a statistical fit equal to that from the model freely estimating the parameter (not shown). Importantly, genetic effects across both outcomes do not significantly correlate (.14), implying that family background factors causing a spurious association between education and age at first birth are mainly environmental. Note that the direct effects from the MZ twin fixed-effects estimates reduces to .07 in this model specification. In the ACE-beta model, the direct link between education and age at first birth explains 16 % of the association, shared environmental influences among siblings explain 62 %, and shared genetic influences explain 22 % (see Online Resource 1, section S2 for details).

Discussion

The supposed causal effect of education on fertility postponement has become a parsimonious and powerful explanation for demographers, not only for the fact that higher-educated women have their first baby at later ages, but also for the general postponement of childbearing during the second half of the twentieth century as a result of educational expansion (Ní Bhrolcháin and Beaujouan 2012). In this study, we challenge the claim that education causally influences age at

first birth and consequently that educational expansion is the main reason for recent fertility postponement. We present within-twin (fixed-effects) and novel biometric models using a unique data set of female twins from the UK. We estimate the causal effect of education on age at first birth and the extent to which environmental and genetic factors cause a spurious association. We find a reduced effect of education on age at first birth in the within-twin design compared with standard regression models. Complementary biometric analyses reveal that the association between education and age at first birth is largely caused by social family background effects, whereas genetic inheritance plays only a small role. These results suggest that the prevailing view of a strong causal effect of education has no merit and needs to be revised.

In our study, the effect of all family factors shared among siblings explained up to two-thirds of the observed association between education and age at first birth, and only a small part of the effect is due to genetic dispositions. This finding provides a strong justification for continued research into the role of family background effects on education and fertility. However, the twin models that we present do not give insights into which family background factors are important for both education and age at first birth. Previous investigations have pointed to SES of the parents (van Doorn et al. 2011) and parental demographic behavior (e.g., Lappegård and Rønsen 2005; Marini 1985; Nisén and Myrskylä 2014; Rijken and Liefbroer 2009). Recent investigations also show that social ties, such as friends (Balbo and Barban 2014), influence fertility timing and that siblings influence each other (Lyngstad and Prskawetz 2010). Given that measured family characteristics typically account for only a small part of the explained variance (e.g., Nisén and Myrskylä 2014) and that parents are important in choosing whom to befriend and date, further study of social and network dynamics (of the family) is one way to better understand the role of the family in fertility behavior.

A central goal of this study was to link our findings at the individual level to those at the population level. An important role has been attributed to education for fertility postponement (Ní Bhrolcháin and Beaujouan 2012), and education is a core variable for fertility projections nowadays (Lutz et al. 2014). Yet, our findings contrast with this approach because we find that increasing educational attainment can explain only a fraction of the trend in age at first birth. Perhaps education serves as a proxy in many studies for other simultaneous historical developments that are not directly measured. An extensive review by Hobcraft (1996) identified changes in sexual norms and family planning as well as the dramatic economic crises throughout the 1970s and 1980s in combination with the introduction of the Pill as reasons for declining fertility levels and postponed fertility in the UK. Consequently, the role of education as a useful predictor of future fertility trends is in doubt.

Previous investigations on both the causal effect of education on age at first birth and the role of genetic versus environmental family background factors produced mixed findings, and our study may contribute to explaining these inconsistencies. First, the nuanced interpretation of the role of education fits with two previous studies. Neiss et al. (2002) and Rodgers et al. (2008) showed for the United States and Denmark respectively that the association between education and age at first birth is completely absorbed by latent family influences. Nisén and Myrskylä (2013) and Amin and Behrman (2014), however, found a significant link between both outcomes net of family influences. Our study suggests that a causal effect of education on age at first birth exists but that it is small and therefore might have remained undetected in former investigations. In the current study, the causal effect estimated from the within–identical twins model—and therefore also in the ACE-beta model—is not statistically distinguishable from zero. Only if we pool identical and fraternal twins, which have nearly identical estimates, do we detect the causal link.

Previous findings on the role of the genetic component in age at first birth in general have been mixed. Here, we find that approximately one-third of the variance in age at first birth is explained by additive genetic effects—similar to findings of studies on the United States (Byars et al. 2010), Australia (Kirk et al. 2001), and Finland (Nisén et al. 2013). However, other investigations from the United States (Neiss et al. 2002) and Denmark (Rodgers et al. 2008) found no significant genetic influences. Differences in genetic effects on fertility exist across countries and within countries over time (Tropf et al. 2016), and also within the UK (Tropf et al. 2015a), and may be due to a gene-environment interaction (Kohler et al. 2006; Mills and Tropf 2016). Further investigation into the genetic pathways to fertility is needed to better understand genetic correlations with educational attainment (Courtiol et al. 2016) and patterns of gene-environment interaction across populations, including molecular genetic information (Barban et al. 2016, Tropf et al. 2015b).

The within–(identical) twin approach that we applied forms a useful tool to establish a causal relationship between two variables but has limitations. The most critical assumption is presumably that variation in education within twin pairs is uncorrelated with variation in age at first birth. The question then arises as to what makes (identical) twins different in educational attainment. If, for example, health issues lead to early school dropout and delayed fertility, the causal effect estimated from the within twin models will be smaller than the true effect. If the presence of a partner influences career and fertility aspirations toward longer educational and later fertility, the true causal effect would be smaller than the estimate from the within-twin models. Additionally, measurement error can downwardly bias the estimates (for discussion, see Amin et al. 2013). However, conservative predictions based on estimates from the OLS models suggest that the educational expansion cannot account for a large part of observed fertility postponement. Nevertheless, we advise caution for the interpretation of our findings insofar as we

find mean differences in age at first birth across TwinsUK and the GHS data. Alternative designs can be used to validate our findings in future research. Desirable approaches include quasi-experimental designs using instrumental variables (for a discussion, see Amin et al. 2015; Boardman and Fletcher 2015).

In general, our study challenges the common approach of explaining differences and trends in fertility timing mainly by invoking educational differences. Differences between families and societal changes and upheavals across time can have a strong impact on fertility timing, which may be mistakenly attributed to the causal influence of just one factor. This study shows that fertility timing is the result of a complex interplay of environmental and genetic influences. We conclude that (the rise in) educational attainment alone cannot explain differences in fertility timing (between cohorts).

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Table 1 Summary statistics for monozygotic (MZ) and dizygotic (DZ) twins separately and within-twin absolute differences for education and age at first birth

	MZ Twins					DZ Twins				
	Mean	SD	Min.	Max.	*N*	Mean	SD	Min.	Max.	*N*
Year Born	1,945.66	10.10	1,924	1,969	1,354	1,947.02	9.37	1,919	1,969	1,398
Age at First Birth	25.87	4.44	15	44	1,354	25.79	4.66	16	44	1,398
Education (age leaving)	16.88	2.49	13	30	1,354	16.90	2.67	12	30	1,398
Within-Twin Absolute Differences										
Age at first birth	3.41	3.27	0	22	677[a]	4.21	3.75	0	20	699[a]
Education	0.88	1.80	0	15	677[a]	1.33	2.40	0	15	699[a]

Note: Education is measured as age when leaving full-time education.
Source: TwinsUK, own calculations.
[a] Refers to number of twin pairs.

Table 2 Linear ordinary least squares (OLS) and within-twin (fixed-effects) regression on age at first birth

	Full Sample		MZ Twins Only		DZ Twins Only	
	OLS	Fixed Effects	OLS	Fixed Effects	OLS	Fixed Effects
	Model 1	Model 2	Model 3	Model 4	Model 5	Model 6
Education (age leaving)	0.44***	0.11*	0.46***	0.12	0.42***	0.11
	(0.04)	(0.06)	(0.06)	(0.09)	(0.06)	(0.08)
Year Born	–3.79***		–5.19***		–2.38*	
	(0.78)		(1.13)		(1.04)	
Year Born, Squared	0.41***		0.56***		0.25*	
	(0.08)		(0.12)		(0.11)	
Zygosity	0.05					
	(0.38)					
Constant	26.81***	23.98***	29.43***	23.89***	24.09***	24.02***
	(1.92)	(0.99)	(2.74)	(1.53)	(2.54)	(1.32)
N	2,752	2,752	1,354	1,354	1,398	1,398
N pairs	1,376	1,376	677	677	699	699

Notes: Unstandardized estimates and standard errors are shown in parentheses. OLS standard errors are adjusted for nonindependence of twins.
Source: TwinsUK, own calculations.
*$p < .05$; **$p < .01$; ***$p < .001$ (one-sided)

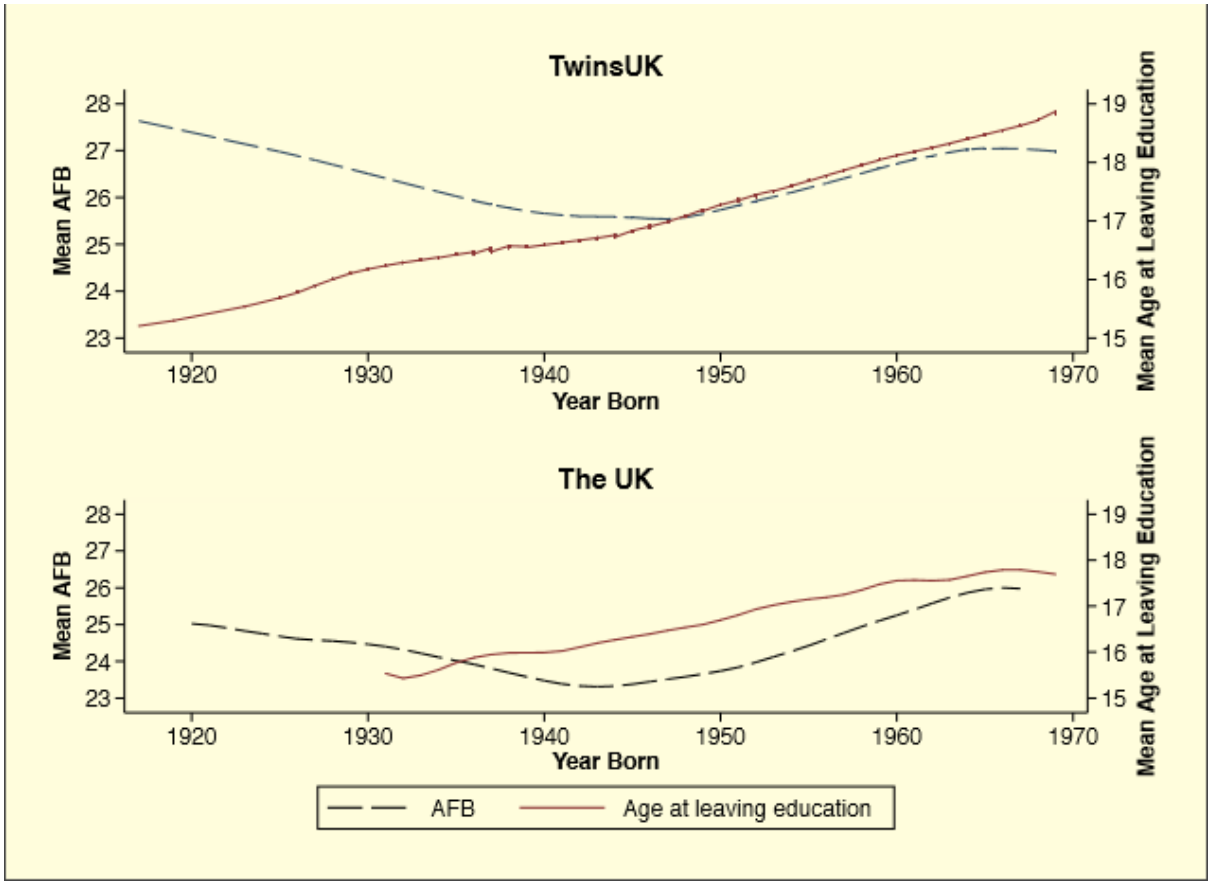

Fig. 1 Trends in mean age at first birth and mean age when leaving full-time education in the TwinsUK sample and national representative data of the United Kingdom. AFB = age at first birth The UK = estimates from large, nationally representative data from the Office for National Statistics. *Source*: TwinsUK and data from the National Office for Statistics (for details, see the Methods section)

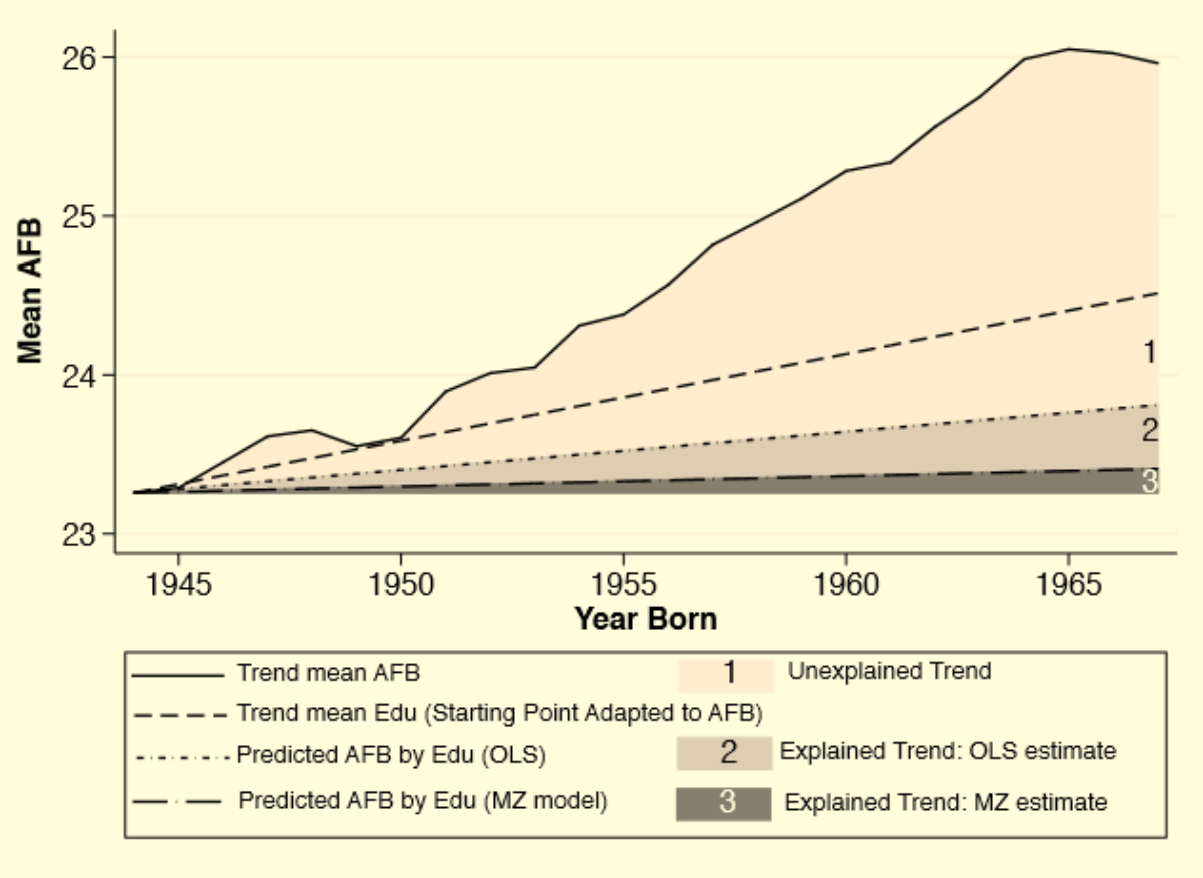

Fig. 2 Can educational expansion explain postponement of childbearing since 1945? The graph depicts the observed trends in mean age at first birth and education (age at leaving) for successive birth cohorts, and the predicted trend in age at first birth by education using the OLS and MZ estimates. The trend for educational level was rescaled to that of age at first birth. Predictions were based on estimates from Model 2, Table 2, and data from Table 1. *Source*: General trends in education were derived from British General Household Surveys (2000–2006) and age at first birth from the Office for National Statistics, Cohort fertility, Table 2 (Office National Statistics 2013)

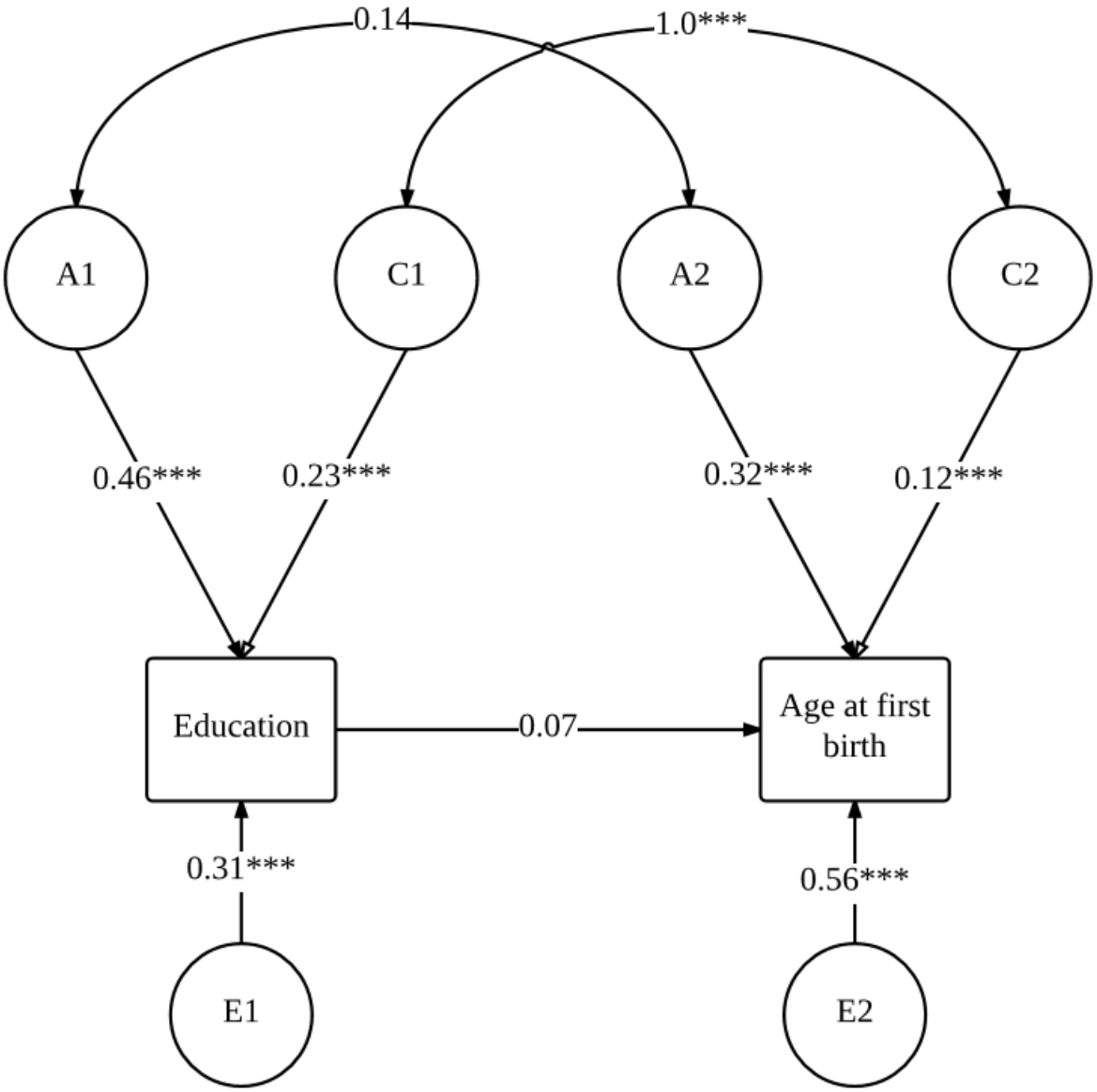

Fig. 3 Correlated factor model depicting standardized estimate of genetic (*A*) and shared environmental (*C*) influences on education and age at first birth, the correlation of these influences across traits, and a causal effect of education on the age at first birth. *Source:* TwinsUK, own calculations. *$p < .05$, **$p < .01$, ***$p < .001$ (two-sided tests)

Online Resources 1

Is the Association Between Education and Fertility Postponement Causal? The Role of Family Background Factors

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Online Resource 1

S1. (Stratified) Cox regression models

In order to draw more general conclusions about the effect of education on age at first birth (AFB) we use Cox regression models that also incorporate censored cases, namely women who have not conceived a child at the last time of observation (right-censored) as well as those younger than 40 years old at last observation (right-censored women and those with children) (*N* = 4,398). See Table S1 for descriptive statistics for this larger sample of women. For childless women we used the last age at observation as the censoring age. The Cox model estimates $\lambda_i(t)$ as the instantaneous risk of an individual i at time t to have a child in case it she did not have a child. Analogous to the linear regression model from equation (1) (main text), we fit the following Cox regression model:

$$\lambda_i(t) = \lambda_0(t) \exp\left(\beta_1(education_i) + \beta_2(birth\ year_i) + \beta_3\left(birth\ year_i^2\right) + \beta_3(zygosity_i)\right)$$

where $\lambda_0(t)$ represents an arbitrary baseline hazard and exp (β') the regression parameters. In the standard exponential form, covariates enter linearly. We will present the coefficients as the relative change in the hazard, namely the hazard ratio of a coefficient exp ($\beta_1 - 1$).

For the within-family models we estimate Cox regression models by stratifying by family. This is analogous to a fixed effects model for censored data (1)**:**

$$\lambda_{ig}(t) = \lambda_{0g}(t) \exp\left(\beta_1(education_i)\right)$$

where $\lambda_{0g}(t)$ represents an arbitrary family-specific baseline hazard. Note that variables that are constant within families are subsumed by the family stratum and therefore drop from the equation. Table S2 shows the results analogous to the OLS models in Table 2 in the main text. Results are well in line with the findings from the OLS.

Table S1: Summary statistics including censored individuals (women who were childless at last observation).

	DZ					MZ				
	Mean	SD	Min.	Max.	N	Mean	SD	Min.	Max.	N
Year born	50.02	11.91	19	83	2164	51.39	14.15	23	88	2234
Age of first birth/age at censoring	29.28	8.05	16	45	2164	29.71	7.97	15	45	2234
Status (having a child, in %)	0.78	0.41	0	1	2164	0.71	0.45	0	1	2234
Education (age when leaving)	17.38	2.94	10	30	2164	17.71	2.96	13	30	2234
Sibling differences in AFB (pairs)	6.62	6.82	0	28	1082 [a]	5.01	6.33	0	28	1117 [a]
Sibling differences in education (pairs)	1.53	2.44	0	15	1082 [a]	1.08	2.02	0	15	1117 [a]

Notes: education = age when leaving full-time education, a = number of twin pairs.
Source: UK twins

Table S2: Cox- and stratified cox-regression for monozygotic (MZ) and dizygotic (DZ) female UK twins, born 1919-88. (analogous to Table 2 in main text)

	Full sample		MZ only		DZ only	
Model	1	2	3	4	5	6
	Cox	Stratified	Cox	Stratified	Cox	Stratified
Education	-0.099***	-0.039*	-0.102***	-0.033	-0.096***	-0.042
	(0.008)	(0.018)	(0.012)	(0.029)	(0.011)	(0.023)
Year Born	0.130***		0.131***		0.128***	
	(0.012)		(0.017)		(0.018)	
Year born squared	-0.002***		-0.002***		-0.001***	
	(0.00)		(0.00)		(0.000)	
Zygosity	-0.081					
	(0.079)					
Observations	4398	4398	2234	2234	2164	2164

Notes: Education = age when leaving full-time education, year born = year born -1900, Zygosity = 0.5 for DZ and 1 for MZ twins, standard errors corrected for non-independence of twins,
* p<0.05, ** p<0.01, *** p<0.001, one-sided
Source: TwinsUK, own calculations

Table S3. Linear OLS and fixed-effects regression on MZ and DZ female UK twins, born 1919-44.

	Full sample	
	1	2
	OLS[a]	FE
Education	0.38***	0.11
	(0.06)	(0.14)
Cohort (if birth year <1945 = 0; else 1)	-1.97	
	(1.43)	
Education*Cohort	0.11	0.004
	(0.08)	(0.03)
Zygosity	0.15	
	(0.38)	
Constant	19.37***	24.14***
	(0.99)	(1.13)
Observations	2752	2752

Notes: Education = age when leaving full-time education, year born = year born -1900, Zygosity = 0.5 for DZ and 1 for MZ twins, a = OLS standard errors corrected for non-independence of twins,
* p<0.05, ** p<0.01, *** p<0.001, two-sided
Source: TwinsUK, own calculations

S2. Correlation, covariance and covariance components in biometric models

Table S4 shows the unstandardized estimates from the ACE-beta model. Figure 3 in the main text shows the transformed estimates into standardized variance components, the correlation between the variance components and we mention in the text the decomposition of the observed correlation between education and age at first birth into the contribution by additive genetic, shared environmental and a causal effect from the ACE-beta model. Here we briefly demonstrate the derivation of these quantities.

Variance components

ACE-beta model

In behavioral genetics it is standard to present the estimates of the latent factors as variance components. Additive genetic influences on education are represented as genetic variance in education a^2_{edu} over the overall variance in education (caused by all underlying factors a^2_{edu}+c^2_{edu}+e^2_{edu}).

From Table S4: $h^2_{edu} = (a^2_{edu}/(a^2_{edu}+c^2_{edu}+e^2_{edu}) = 1.77^2 /(1.77^2 + 1.24^2 + 1.44^2) = 0.46$

For heritability of age at first birth the influence of the unique environment is replaced by a direct link between education and age at first birth. It therefore consists of three elements: i) direct genetic influences unique to age at first birth, ii) direct genetic influences which are shared between education and age at first birth iii) indirect genetic influences which operate via education and can be estimate by

$$h^2_{afb} = \frac{\beta^2 a^2_{edu} + 2\beta a_{edu,afb} + a^2_{edu,afb} + a^2_{afb,afb}}{\sigma^2_{afb}}$$

where $\sigma^2_{afb} = \beta^2(\sigma^2_{edu})^2 + 2\beta(a_{edu,afb} + c_{edu,afb}) + (a^2_{afb,afb} + a^2_{edu,afb} + c^2_{afb,afb} + c^2_{edu,afb} + e^2_{afb,afb})$.

Correlation

The correlation of the latent factors in the model, for example for the shared environmental effects (parallel for genetic effects) is:

$$\mathrm{r(c)} = \frac{c_{edu} * c_{edu,afb}}{\sqrt{c^2_{edu}(c^2_{edu,afb} + c^2_{afb,afb})}}$$

A correlation of r(c) = 1 means that all shared environmental effects for fertility are associated with shared environmental effect for education, while a correlation of r(c) = 0 means that both effects are independent.

Covariance components

The overall covariance between education and age at first birth can be decomposed in parts which are due to genetic, shared environmental effects and due to the direct/causal effect. This is achieved by calculating the ratio of the respective covariance over the overall variance – the sum of the three components:

$$Cov(Age\ at\ first\ birth_{if}, education_{if}) = a_{edu}a_{edu,afb} + c_{edu}c_{edu,afb} + \beta\sigma^2\left(education_{ij}\right)$$

For example, the contribution of the genetic covariance to the overall covariance is:

$$\frac{a_{edu}a_{edu,afb}}{a_{edu}a_{edu,afb} + c_{edu}c_{edu,afb} + \beta\sigma^2\left(education_{ij}\right)} =$$

$$\frac{1.77 * 0.37}{1.77 * 0.37 + 1.24 * 1.48 + 0.07 * 6.74} = 0.22$$

This means that 22% of the correlation between education and age at first birth is due to shared genetic effects. The respective values for the shared environment of the siblings 62% and for the causal effect 16%.

Table S4. Unstandardized estimates of the ACE-beta models - *N* observations = 2,752; *N* twins = 1,376.

Model	ACE-beta	
	estimate	se
education		
a	1.77***	.12
c	1.24***	.15
e	1.44***	.04
age at first birth		
a	2.53***	.22
c	0.00	.94
e	3.42***	.09
cross-trait effects		
a	0.37	.39
c	1.48***	.31
e	-	-
Beta	0.07	.09

Notes: * p<0.05, ** p<0.01, *** p<0.005, two-sided
Source: UKtwins, own calculations

References

1. Allison PD, Christakis N (2006) Fixed-effects methods for the analysis of nonrepeated events. *Sociol Methodol* 36(1):155–172.
2. Kohler H-P, Behrman JR, Schnittker J (2011) Social science methods for twins data: Integrating causality, endowments, and heritability. *Biodemography Soc Biol* 57(1):88–141.