Education, fertility postponement and causality: the role of family background factors

Abstract

A large body of literature demonstrates a positive relationship between education and age at first birth. However, this relationship may in part be spurious due to family background factors that cannot be controlled for in most research designs. We investigate to what extend higher education is causally related to later age at first birth timing in a large sample of female twins from the UK (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 half a year later age at first birth in standard models. This reduced to only 1.5 months for the within-identical twin model that control for all shared family background factors (genetic and family environmental). Biometric analyses reveal that mainly influences of the family environment – not genetic factors – cause spurious associations between education and age at first birth. Lastly, we demonstrate using data from the Office for National Statistics that only 1.9 months of the 2.4 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: Age at first birth; Education; Fertility postponement; Twins; fixed-effects; Genetics; UK

Introduction

A large body of literature from the USA and Europe demonstrates a positive relationship between higher educational attainment and later age at first birth (e. g. Blossfeld and Jaenichen 1992; Gustafsson et al. 2002; Lappegård and Rønsen 2005; Marini 1984, 1985; Martin 2000; Rendall et al. 2005; Rindfuss and St. John 1983). It has been argued that this relationship is causal and that it can account for the rise in the mean age at first birth during the educational expansion of the second half of the 20th century in Western countries (Balbo et al. 2013; Bhrolcháin and Beaujouan 2012; Mills et al. 2011). However, research also casts doubt on the idea of a causal effect of education on fertility timing, suggesting that family background characteristics ([social] environmental and genetic) cause spurious associations between educational attainment and fertility timing of women (Neiss et al. 2002; Rodgers et al. 2008).

Our study contributes to the existing 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-identical twin estimates and engage in biometric modeling of the link between education and age at first birth in order to disentangle the causal relationship from genetic and environmental confounders. Next to quasi-experimental study designs, within-twin designs offer an approach to causality. Quasi-experimental designs (McCrary and Royer 2011; Skirbekk and Prskawetz 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 amongst siblings including genetic material by using identical twins as a natural experiment (Kohler et al. 2011; Nisén and Myrskylä 2014). Although more widely used in economics (e. g. Amin et al. 2013), to our knowledge, we are the first to present within-identical twin estimates of the effect of education on age at first birth.

This approach is of particular interest for two reasons. First, it has been shown that within-twin variation arises across all levels of education, while for example compulsory school laws mainly cause variation on a particular level of education (Amin et al. 2015). Second, we expect that most of the third factors important for education and fertility timing are related to the family of origin. Research into social stratification has demonstrated considerable similarity between parents-children and siblings in education and socio-economic attainment (Jæger 2012; van Doorn et al. 2011). Equally, fertility behavior is transmitted across generations (Murphy 1999; Rijken and Liefbroer 2009; Steenhof and Liefbroer 2008). In families, not only parental socio-economic status or socialization are important for education and fertility (Marini 1984; Nisén and Myrskylä 2014; Rijken and Liefbroer 2009; Rindfuss et al. 1984), but also genetic dispositions (Branigan et al. 2013; Mills and Tropf 2015). Importantly, Kohler and colleagues (2011) recently developed a model integrating the biometric and the within-identical twin approaches. Their approach allows to disentangle the causal effect of education on age at first birth from family environmental and genetic effects as a common influence on both outcomes.

We applied standard models as well as the advanced modeling approach from Kohler and colleagues (2011) to a unique data source of female identical and fraternal twins from TwinsUK. The TwinsUK is the largest adult twin register in the United Kingdom (Moayyeri et al. 2013). 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 stronger in the UK than in other European countries such as France, Germany, Norway or the Netherlands (Gustafsson et al. 2002; Rendall et al. 2005). A recent investigation from the UK by Bhrolcháin &

Beaujouan (2012) showed that between 1980-1999, 57 per cent of the postponement of the mean age at first birth can be attributed to longer educational enrolment while the remainder would be due to additional, post-educational postponement effects – if we endorse the said causal relationship between education and age at first birth.

Previous demographic research has not sufficiently related the individual and population level (Billari 2015). We therefore combined our findings at the micro-level with nationally representative data from the Office for National Statistics to evaluate whether the causal effect of education explained age at first birth trends at the population level. This was an important goal, not only in order to understanding past fertility trends, but also to assess the potential to anticipate future fertility development based on changes in the educational level - as education has gained importance in fertility forecasting (Lutz et al. 2014). In the UK we observed furthermore that the trend in age at first birth was U-shaped during the mid-century in the UK (Hobcraft 1996), whereas educational expansion increased steadily (Oreopoulos 2006). These differential trends motivated a closer inspection of the relationship in different birth cohorts. We therefore also present analyses separately for birth cohorts born before and after the Second World War. In the following, we first discuss the previous (bio-)demographic literature, continue by introducing the TwinsUK dataset and our methods, followed by a presentation and discussion of the central findings.

Background

Education and fertility timing

The mean age at first birth of women steeply increased by up to 4-5 years during the second half of the twentieth century all over Europe and the US and was accompanied by an overall increase in educational attainment (Mills et al. 2011). In the UK, between 1980 and 2000, for example, the average age at first birth as well as age of leaving full-time education increased by 1.4 years (Bhrolcháin and Beaujouan 2012). Joshi (2002) showed by comparing three UK birth cohort studies (1946, 1958, 1970) that the percentage of women who were already mothers by age 26 dropped from 81 per cent for those born in 1946 to 30 per cent for the 1970 cohort. In all three birth cohorts, women with tertiary educational qualification were about half as likely to be mothers than women with no educational qualifications. In the most recent 1970 cohort, only 1 in 10 higher educated women were mothers at age 26 compared to 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 (Balbo et al. 2013; Bhrolcháin and Beaujouan 2012; Mills et al. 2011; Nisén and Myrskylä 2014). First, being enrolled in education itself may postpone childbearing, as it is difficult to combine the student role with the mother role, since both imply time intense tasks. Women might delay childbearing due to high costs of children and few resources during the time of their studies or social norms might control parenting before the end of education (Bhrolcháin and Beaujouan 2012; Blossfeld and Huinink 1991; Hoem 2000; Lappegård and Rønsen 2005). Second, education may increase people's aspirations and ability to pursue a career. So, 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 larger human capital (Amuedo-Dorantes and Kimmel 2005; Happel et al. 1984; Liefbroer and Corijn 1999). Furthermore, education might also change values and orientations towards more individualistic lifestyles that seek fulfillment in life without children (de Kaa 1987; Lesthaeghe 1995). Therefore, both educational enrolment and attainment may lead to postponing childbearing (and reducing fertility). It is worth to mention also a reverse causal mechanism because an early age at first birth may disrupt further education (Cohen 2011; Hoffman et al. 1993). This is presumably mainly the case for unwanted teenage childbearing (Nisén and Myrskylä 2014).

Reasoning along these lines and noting the often observed micro-level association led to the hypothesis that educational expansion in the UK directly explains up to 57 per cent of the postponement of age at first birth at the population level (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 across the past century; the coinciding trends only hold for after WWII. Second, besides the overall rise in education, there are a number of competing explanations for the rise in (female) age at first birth in developed countries, which we will briefly discuss below. Third, the supposed causal relationship between education and age at first birth at the micro-level is not without its critics. We will discuss these issues in turn in more detail.

Changing macro trends

Comparative studies within Europe demonstrated variability in the association between education and age at first birth (Gustafsson et al. 2002; Rendall et al. 2005) as well as educational specific birth timing (Rendall et al. 2010) across countries. Also within the UK the observed association between education and age at first birth on the population level changed across the past century. Earlier in the 20th century, age at first birth decreased in periods after the Second World War accompanied with a steep increase in fertility - the so-called baby boom. At the same time educational attainment increased. The UK's Education Acts from 1918 and 1944 lifted the school leaving age from 12 to 14 and later from age 14 to 15. As shown by Oreopoulos (2006), within three years during the period of 1945-1947 the fraction of 14-year-olds leaving schools fell from about 57 percent to less than 10 percent. Only later that century, since the 1960s age at first birth increased – the so called baby-bust – and education further expanded and trends in the age when leaving education and age at first birth ran parallel. Given the reversing trends throughout the century, a strict causal logic that longer educational enrollment leads to later age at first birth may not apply.

Fertility postponement: Alternative explanations

There are several alternative explanations important for fertility postponement in most developed countries during the 20th century - mainly surrounding the introduction of the 'pill'. It has been shown that rising female labor force participation (A. R. Miller 2011; Oppenheimer 1994; Rindfuss et al. 2007), ideational shifts in norms and values in sexual behavior and family planning (de Kaa 1987; Lesthaeghe 1995) and economic uncertainty (Andersson 2000; Hoem 2000; Kravdal 2002) are associated with an increasing age at first birth and might (partly) account for the postponement. The introduction of the 'pill' as an effective contraception is seen as an important trigger of these mechanisms (Mills et al. 2011).

In the United Kingdom, the contraceptive pill was introduced in 1961 and broadly available to married adults by contraceptive services at the end of the 1960s. According to Hobcraft (1996), more than half the fertility decline in the 1960s may be attributed to a reduction of unwanted children outside of marriage. Hobcraft identifies the sexual revolution of the 1960s as a strong factor leading to both fertility postponement and lower overall fertility.

A large body of literature furthermore linked economic uncertainty on the individual and aggregated level to first birth postponement. Most central, unemployment or unstable labor market situations prevent from making long-term binding decisions such as having children (for review see Mills et al. 2011). Throughout the 1970s and in the beginning of the 1980s, the UK had to face a major economic crisis. The proportion of married women who had ever used the pill during the first 5 years of marriage increased from less than one-third in 1967 to around 80 per cent in 1976 (Murphy 1993) and England and Wales reached a historical low point in total fertility in 1977 (Hobcraft 1996).

Especially change 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 or independent of the educational expansion (Hobcraft 1996; Murphy 1993). To evaluate to what extent the educational expansion can be held responsible for fertility postponement and avoid ecologic fallacy, we assess the causal effect of education on age at first birth on the individual level and use these estimates for predictions on the population level.

Critique of education and fertility link

The association between education and age at first birth might be in part spurious because third factors influencing age at first birth, such as economic uncertainty or one's genetic make-up, may also be related to education. Especially the family background may be important, as many studies repeatedly demonstrated that education and fertility are associated with both the family environment and genetic factors. Both, socio-economic characteristics (Jæger 2012; van Doorn et al. 2011) and fertility behavior (Murphy 1999; Rijken and Liefbroer 2009; Steenhof and Liefbroer 2008) show intergenerational resemblance. The family background plays a pivotal role as a socializing agent, a source of resources and support, and for transmitting genes. Therefore the family environment and genetic factors may explain the observed association between for education and age at first birth.

Family background, for example the socio-economic status 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 and increase 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. It has been shown that the timing of first attempts to get pregnant measured in retrospective interview is linked to one's genetic make-up (Rodgers et al. 2001). It is also established that genes influence educational attainment (Branigan et al. 2013; Rietveld et al. 2013), and fertility timing (Nisén et al. 2013; Tropf et al. 2015). However, the question remains to what extent genetic effects for both outcomes are shared. In the following, 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

To our knowledge three previous twin studies applied biometric models to investigate genetic and (socially) environmental influences on the relationship between education and age at first birth. The first, a study of Finnish twins born in 1950-1957 (Nisén et al. 2013) estimated bivariate biometric models which decomposed the (co)variance in education and age at first birth into latent genetic and environmental factors. For women they report that genetic, shared environmental factors of the twins as well as environmental factors unique to an individual independently explain part of the observed covariance between education and age at first birth. Influences unique to an individual, which both lead to lower/higher education and earlier/later age at first birth 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).

The second study used the NLSY siblings born between 1958-65 (Neiss et al. 2002) and the third the Danish twins born between 1931-52 (Rodgers et al. 2008). Both investigations focus on the role of education as a mediator between cognitive ability and age at first birth. They extend the bivariate biometric model by introducing cognitive ability as a third variable and allow for causal paths between cognitive ability and education, and between education and age at first birth. Modeling the causal pathways directly is important to take the dependency of confounding factors and causal influences into account. It is for example possible that genetic influences on age at first birth operate via education only. Mediation models do consider this possibility and therefore genetic and shared environmental influences important for both outcomes can be considered as common causes and independent of a mediating effect (Kohler et al. 2011). Both studies find that the observed mediating link of education turns non-significant when controlling for genetic and environmental influences from within the family. In contrast to the Finish study, these investigations find no genetic influences on age at first birth and thus no genetic correlation between both outcomes. Still, they conclude that education may not directly delay childbearing, but individual differences which lead to higher education and higher cognitive ability inhibit fertility and those differences arise between families, but not within them.

In this study, we followed a different strategy than the previous studies. First, we focus on the causal relationship between education and age at first birth and apply within-twin regression models, including within identical twin regression (Amin et al. 2013). Similar to the bivariate modeling approach (Nisén et al. 2013), this gives an estimate of the causal effect of education on age at first birth controlling for all shared factors amongst the twins, including their genetic material. However, the with-twin regression is statistically more powerful and we conduct our analyses (also) on identical and fraternal twins separately, which has the advantage that it relaxes assumptions about the extent to which twins share environmental factors within families (Amin et al. 2015). Second, we extend these models according to Kohler et al. (2011) by integrating the bivariate twin model in order to estimate the direct effect of education on age at first birth as well as the effect of genes and the shared environment as common causes for the observed association between both outcomes.

Compared to previous approaches (Neiss et al. 2002; Rodgers et al. 2008), this mediation model needs fewer identifying assumptions and all paths we introduce to the model are identified and can be estimated. We compare this approach with standard bivariate twin models as in Nisén et al. (2013), which will give insights in how far genetic and environmental influences on education and fertility are common causes or mediated by a causal effect of education on fertility.

Methods

Data

We used information on twins of the TwinsUK registry. The TwinsUK was originally established at the St. Thomas Hospital London in 1992 and gathered information on the life course of identical or monozygotic (MZ) and fraternal or dizygotic (DZ) twins (Moayyeri et al. 2013). Zygosity was established using standardized questions and confirmed by DNA genotyping in cases of uncertainty. Currently it contains information on about 12,000 individuals. We limited the analysis to same sex female individuals in complete twin pairs because the TwinsUK dataset contains very few male twins (<15 per cent) so that a comparable analysis for men was not feasible. We furthermore excluded women younger than 40 years old to avoid an over-representation of young mothers and limit right-censoring of women who did not have children at the time of last observation. Valid information on zygosity, fertility and education was available for 3,856 women in 1,928 twin pairs. The sample is further reduced due to right-censoring of one or both twins in a twin pair to 2,752 women in 1,376 twin pairs.

In order to compare the TwinsUK sample to a representative national sample and to describe trends in education on the population level we pooled data from the Office for National Statistics. 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, carried out by the Office for National Statistics. It collects a range of socio-economic information on household members. To describe age at first birth we used estimates from the National Statistics (Office National Statistics 2013) because 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, it contains an inventory of the years of birth of up to ten children. Second, age at first birth was assessed directly with the question: “How old were you when you had your first live birth?” in the 2004 questionnaire. In case individuals participated in multiple waves, these two measures can differ by one year, because the former is constructed based on year born and year of first childbirth only. If the difference across measures was one year (~2.5 percent of the cases), we used the earliest reported age at first birth. In less than 2 percent of all reports, there was a discrepancy across the wave of more than one year and we excluded those cases.

Education

A distinct feature to our study is that we measured education as the age at leaving full-time education. This was assessed directly with the question: “At what age did you leave full-time education?” Three questionnaires contained this information: the Main Questionnaire, the Twins Questionnaire from 1999-2000 and the TwinsUK Baseline Health Questionnaire from 2004-2008. This may be a better measure of the supposed causal mechanisms underlying the education effect than using the highest attained educational level because the causal reasoning for an education effect rests in part on direct time competition during studies and a waiting period after finishing school to start a career.

Methods

The analysis proceeded in two steps. First, we present ordinary least square regression (OLS) and subsequently within-twin fixed effect models (FE) to estimate the effect of education on age at first birth. A comparison of the results from these two methods shows to what extent the family background leads to a spurious association between education and age at first birth. The OLS-models include birth year and birth year squared to allow for a curvilinear trend in fertility timing across birth cohorts. We furthermore control for zygosity measured as the expected genetic relatedness between DZ twins (0.5) and MZ twins (1) and estimate robust standard errors to correct for the dependency structure of the twins. The OLS model estimates the 'naive' association between education and age at first birth. As detailed elsewhere (Amin et al. 2013; Kohler et al. 2011), standard OLS ignores the shared environment in a family, such as family norms and genetic endowments.

The within-twin models include fixed effects per family, which capture all (observed and unobserved) factors shared among the twins. In addition, within-twin models also allow to control for unobserved genetic differences. We can discriminate between DZ and MZ twins. DZ twins share on average 50% of their segregating genetic material, MZ twins are genetically identical, so that a within-DZ model controls for half of all additive genetic effects and the within-MZ model controls for all genetic effects.

We applied both the OLS regression and the within-twin design to the pooled sample of siblings as well as DZ und MZ twins separately. The comparison, particularly of the DZ and MZ models gives us first insights whether differences between the OLS and the fixed effects models are due to shared environmental 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).

Genetic and environmental factors

In a second step, we estimated bivariate biometric models to quantify to what extent genetic and/or environmental effects lead to a spurious association between education and age at first birth. Twin models are no longer uncommon in social science (Branigan et al. 2013; Freese 2008; Kohler et al. 1999, 2011; W. B. Miller et al. 2010; Mills and Tropf 2015; Neiss et al. 2002; Nisén et al. 2013; Rodgers et al. 2001, 2008; Tropf et al. 2015). Briefly: Twin models facilitate the comparisons between identical or monozygotic (MZ) and fraternal or dizygotic (DZ) twins, in order to quantify genetic and non-genetic environmental influences. The siblings are assumed to share common environmental influences such as their parents, the neighborhood they grew up in and other related aspects. While MZ twins are genetically identical (i.e., share all their genotypes), DZ twins are akin to full siblings and thus share on average only around 50 per cent of additive genetic effects. Similar to parent–offspring correlations, the correlation among DZ twins therefore reflects the importance of both environmental and genetic effects in families. The degree, however, to which MZ twins have a higher correlation in the trait of interest than DZ twins, reflects the fact that they are genetically more similar.

The comparison of twin correlations thus already makes it possible to quantify genetic and shared environmental effects related to a particular trait (Boomsma et al., 2002; Snieder et al., 2010). As has become standard, we apply structural equation models using the R software package OpenMx (Boker et al. 2011; Neale and Cardon 1992) to decompose the observed variance for the outcomes into three components: (a) additive genetic effects resulting from the sum of genetic effects from the whole genome (A), (c) environmental effects resulting from environmental influences shared between twins of a pair (C) and (e) non-shared environmental effects resulting from the unique environment of an individual (E) (which includes measurement error).

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. We estimate two bivariate twin models, first the 'bivariate ACE' model which estimates not only the three variance components (A, C, and E) for both outcomes, but also the correlation of these latent factors across outcomes.

Three assumptions of the standard behavioral genetics model need to be briefly addressed: The first is that MZ and DZ twins share their environment to the same extent (the equal-environment assumption (EEA)). This assumption has repeatedly been criticized (e.g., Horwitz et al. 2003), though evidence of it not being made is rare in studies (Conley et al. 2013), including fertility studies (Felson 2014). Second, it is assumed that there is no assortative mating within the population with respect to the outcome of interest. A violation of this assumption (Domingue and Fletcher 2014) would result is an underestimate of genetic influences on an outcome. The third assumption is that there are no non-additive genetic effects (dominant, epistatic and gene-environment interaction effects).

We then extend the bivariate model and estimate the recently developed 'ACE-beta' model. This model integrates the within-MZ estimation into the structural equation model by assuming that the correlation of the unique environment of the individuals for both outcomes is zero. The difference between both modeling approaches is that the bivariate ACE model treats all three components as independent, whereas the ACE-beta model allows the direct link between education and age at first birth to mediate genetic (A) and shared environmental influences (C) on education (see Figure S1 and S2 in the supplementary material for a graphical comparison of both approaches). The estimated genetic and environmental correlation in the ACE-beta model therefore can be interpreted as common influences on both outcomes and the comparison of both modeling approaches gives insights in how far shared influence on both outcomes are mediated through the causal effect of education on age at first birth.

We used Stata 12 to estimate the OLS and fixed effect models, the OpenMx package in R for the bivariate ACE models (Boker et al. 2011) and we gratuitously received the OpenMX R-function to estimate the 'ACE-beta' model from the developers Kohler et al. (2011).

Robustness

The main analysis presented in this paper included only twin pairs for whom both twins had a child and who were both 40 at last point of observation because the bivariate biometric models cannot deal with non-linear outcomes (see Tropf, et al. 2015 for age at first birth using a Tobit model). We tested the robustness of our results by repeating the analysis using Cox-regression models that can handle right-censored observations. In the supplementary material, table S1 presents summary statistics and table S2 presents 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).

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 about 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. It should also be noted that only 27 individuals (~1 per cent) had their first birth before leaving education and only 74 (~2.7 per cent) until one year after leaving education so that the temporal succession suggests that education influences age at first birth and not vice versa.

< Table 1 about here >

There are two main concerns about the within-twin approaches: first that variation of the independent variables within twin pairs is low and largely due to measurement error and second that twin data are not representative for the general population. To address these concerns, first, table 1 shows the mean absolute differences in education within twin pairs. These differences were substantial with 1.33 for DZ and 0.88 for MZ, which suggests that sufficient variation remained even within MZ twin pairs. The fact that they were smaller in MZ twins than in DZ twins suggests a genetic component underlying education. Unfortunately, we were limited in our ability to address measurement error as the available TwinsUK dataset does not offer independent report for outcomes of interest. Second, TwinsUK, is considered to be representative for the singleton population in the UK (Moayyeri et al. 2013). Figure 1 shows the smoothed trends in age when leaving education and age at first birth for the general population (top panel) and the TwinsUK sample (lower panel). The general level and trends for the TwinsUK sample were very similar to that of the general population, which increases our confidence in using the TwinsUK sample.

Moreover, it is clear from Figure 1 that the mean age of leaving education rose steadily throughout the 20th 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 20th century, concluding that the rise in age at finishing education lead to a postponement of childbearing. However, the figure reveals the previously described discontinuity in the association, which challenges the idea of a causal relationship between education and age at first birth.

< Figure 1 about here >

< Table 2 about here >

The causal effect of education on age at first birth

Our first research question asks: Is there a causal effect of education on age at first birth? Model 1 in Table 2 shows the well-established naïve OLS estimate of the effect of education on age at first birth of .44 (standard error (SE) 0.04). In this sample, women who stayed one year longer in education had their first childbirth about half a year later. This holds independent of cohort effects as we controlled for birth year and its square. Model 3 and model 5 repeat the same model but now for subsets of MZ and DZ twins separately. The estimates of MZ and DZ twins were nearly identical.

Now we turn to the within-twin models (FE) to tease out the causal effect. Model 2 in Table 2 shows the pooled within-twin approach, controlling for all shared environmental factors among siblings. The effect of education on age at first birth reduced to .11 (SE 0.06), but remained significantly different from 0 at the 5% level. The within-twin estimate for the

DZ twins is depicted in model 6, this estimate controlled for all factors that vary at the family level, only genetic differences among DZ twins are not controlled for. The within-MZ estimate in model 4 additionally controlled for all genetic differences, as MZ twins are genetically identical. The within MZ estimate was similar to that of DZ twins and the pooled estimate. The estimates from the within-twin MZ and DZ analyses were not significantly different from 0. This was probably due to reduced power of the within-twin approach, as the estimates were all about the same size. This suggests a reduction in the causal effect of education on age at first birth with about three quarters (1-.12/.44) to about 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).

In order to check for 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, Supplementary Table S2 provides the results from the (stratified) Cox regression models (*N* = 4,398). Results followed the same pattern as the regression models, except that standard errors were generally smaller due to the increased sample size and the reduction in the estimated effect of education for the within-twin analyses appeared to be less strong. The probability to have a child decreased with each additional year of education by ~10% and this reduced to ~3 to 4% in the stratified Cox regression models.

Our second research question asked, to what extent the educational expansion during the second half of the twentieth century is able to explain the simultaneous overall postponement in age at first birth. Figure 2 depicts the simultaneous rise in age of leaving education and age at first birth for cohorts born between 1944-1969. These cohorts started childbearing in time periods since the 1960s and therefore can be considered the main drivers of the fertility postponement (Mills et al. 2011). Age at first birth in 1969 in the UK (28.15) was about 2.4 years later than in 1944 (25.75). Figure 2 furthermore shows the simultaneous rise in age of leaving full-time education from around 16.5 year to 18 (a difference of 1.3

years) across these birth cohorts. A straightforward macro-explanation might consequently conclude that 54 per cent (=1.3/2.4) of the 2.4 year of fertility postponement can be attributed to a rise in educational enrolment. However, as shown in the within-twin regression, for each year of additional education, individuals postpone ~1.4 months. Across birth cohort born between 1944-69 therefore only around 6.5 per cent (=1.3*0.12/2.4) 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 for subsequent birth cohorts based using the within-MZ estimate, as well as the explained (green) and unexplained (red) age at first birth trend across the second half of 20th century.

The estimates in Table 2 are based on birth cohorts born between 1919-1969. As shown in Figure 1, the association between education and age at first birth reversed on the population level and it is possible that the same applies to 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 age at first birth trend) to investigate this issue (see Table S3 in the suppl. material). However, we do not find such differences.

< Table 3 about here >

The role of genes and the environment as family background factors

The three fourth reduction of the estimate of the causal effect of education on age at first birth in OLS versus within-MZ models leaves us with the third research question to what extent this is due to unobserved shared environmental influences and/or unobserved genetic endowments that are common to both outcomes. Table 3 shows the results of the two biometric models, the standard bivariate ACE model and the extended ACE-beta model, which includes the causal effect of education on age at first birth, to answer this question. We present results as standardized variance components and correlations between these latent factors. Please see table S4 in the supplementary material for unstandardized estimates.

In both models, the heritability of age at first birth is estimated to be 0.32, meaning that 32 per cent of the variance in age at first birth was associated with additive genetic differences. Shared environmental effects were smaller (0.12) but significant and unique environmental effects, which includes measurement error, made up the largest part (0.56). For education, heritability was higher than for age at first birth (0.46) and the shared environmental influences account also for a larger part (0.23) of the variance, while the unique environment was smaller than for age at first birth (0.31).

Both the bivariate ACE model and the extended ACE-beta model provide insight in the relationship between education and age at first birth. We first discuss the bivariate ACE-model. In the bivariate ACE model, the correlation of genetic factors for education and age at first birth was 0.19, which was significant at the 5% level. Shared environmental influences correlated to 1 across outcomes. This means that there were no shared environmental influences unique to age at first birth, i.e. those latent shared environmental influences that pertain to education also pertain to age at first birth. Unique environmental influences showed no significant correlation across outcomes. Based on the bivariate ACE-model 29 per cent of the association between education and age at first birth can be attributed to common additive genetic effects, 66 per cent to common shared environmental effects amongst siblings and 5 per cent to unique environmental factors.

The second model specification, the ACE-beta model, included a direct link between both traits estimated from the within-MZ model instead of the correlation of unique environmental effects (estimated slightly lower than in Table 2 with beta 0.08, SE 0.09). This suggests that the direct link between education and age at first birth explained 17 percent of the association, 62 percent can be attributed to shared environmental influences amongst siblings and 21 percent to shared genetic influences. Comparing the bivariate ACE and ACE-beta model, the decrease in the contributions of the latent family environmental factors in the ACE-beta model indicates that education mediated the effect of the latent influence on age at first birth, and genetic influences shared independent of the causal effect turn non-significant.

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 babies at later ages, but also for the general postponement of childbearing during the second half of the 20th century due to the educational expansion (Bhrolcháin and Beaujouan 2012). In this study we challenge the claim that education causally influences the age at first birth and consequently that the educational expansion is mainly responsible for recent fertility postponement. We present within-twin and novel biometric models using a unique dataset of female twins from the UK to 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 to standard regression models. Complementary biometric analyses reveal that the association between education and age at first birth is to a large extent 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 merit but needs to be nuanced.

In our study we quantify the effect of all family factors shared among siblings and observe that up to two thirds of the observed association between education and age at first birth is due to the (unobserved) family environment, only a small part is due to genetic dispositions. This finding forms a strong motivation for continued research into the role of family effects on education and fertility. At least two major challenges are first that the twin model we present does not give insights which family background factors are important for both education and age at first birth in the UK. Previous investigations have pointed to socio-economic status of the parents (Jæger 2012; 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, however, also show that social ties such as friends (Balbo and Barban 2014) influence fertility, just siblings influence each other in fertility behavior (Lyngstad and Prskawetz 2010). Given that measured family characteristics typically only account for a small part of the explained variance (e. g. Nisén and Myrskylä 2014) a potential direction to better understand fertility behavior lies in the social and network dynamics (of the family) – also in order to explain trends on the population level (Kohler et al. 2002) . Second, shared environmental influences of siblings represent a rather diffuse concept in twin models. We know for example the same parental influences can have different effects on children's development (Pike and Kretschmer 2009). and therefore, models not considering the family structure itself remain unable to entirely control for this statistical component.

A central goal of this study was to link our findings on the individual level to the population level because the explanation of macro phenomena such as fertility postponement is one of the central enterprises in demography (Billari 2015). An important role has been attributed to education for fertility postponement (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, as we find that increasing educational attainment can only explain a fraction of the trend in age at first. 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) nominated changes in sexual norms and family planning as well as the dramatic economic crises throughout the 1970s and 80s in combination with the introduction of the 'pill' as reasons for depressing fertility levels and postponed fertility in the UK. Consequently, the role of education as a useful predictor of future fertility trends is in doubt.

This nuanced interpretation of the role of education fits with a number of previous investigations on the relationship between education and age at first birth. A twin study from Finland (Nisén et al. 2013) similarly only finds evidence for a (small) causal effect of education on age at first birth in the within-twin models and studies by Neiss et al. (2002) and Rodgers et al. (2008) show for the US and Denmark that the entire association between education and age at first birth is absorbed by latent family influences. In our study, the effect size is small with only around 1.5 months birth postponement per additional year of education. Additionally, the structural equation models (SEM) applied in this study and previous work show larger standard errors than regression models which may have contributed to the null-findings in previous SEM models (Neiss et al. 2002; Rodgers et al. 2008). Overall, replication of these findings in larger datasets is desirable and differences across studies are possibly due to true heterogeneity in the causal effect between education and age at first birth across countries. Yet, even our finding of a causal relationship shows in line with previous studies within families (Neiss et al. 2002; Nisén and Myrskylä 2014; Rodgers et al. 2008) that that the causal effect of education on age at first birth is smaller than suggested in the past.

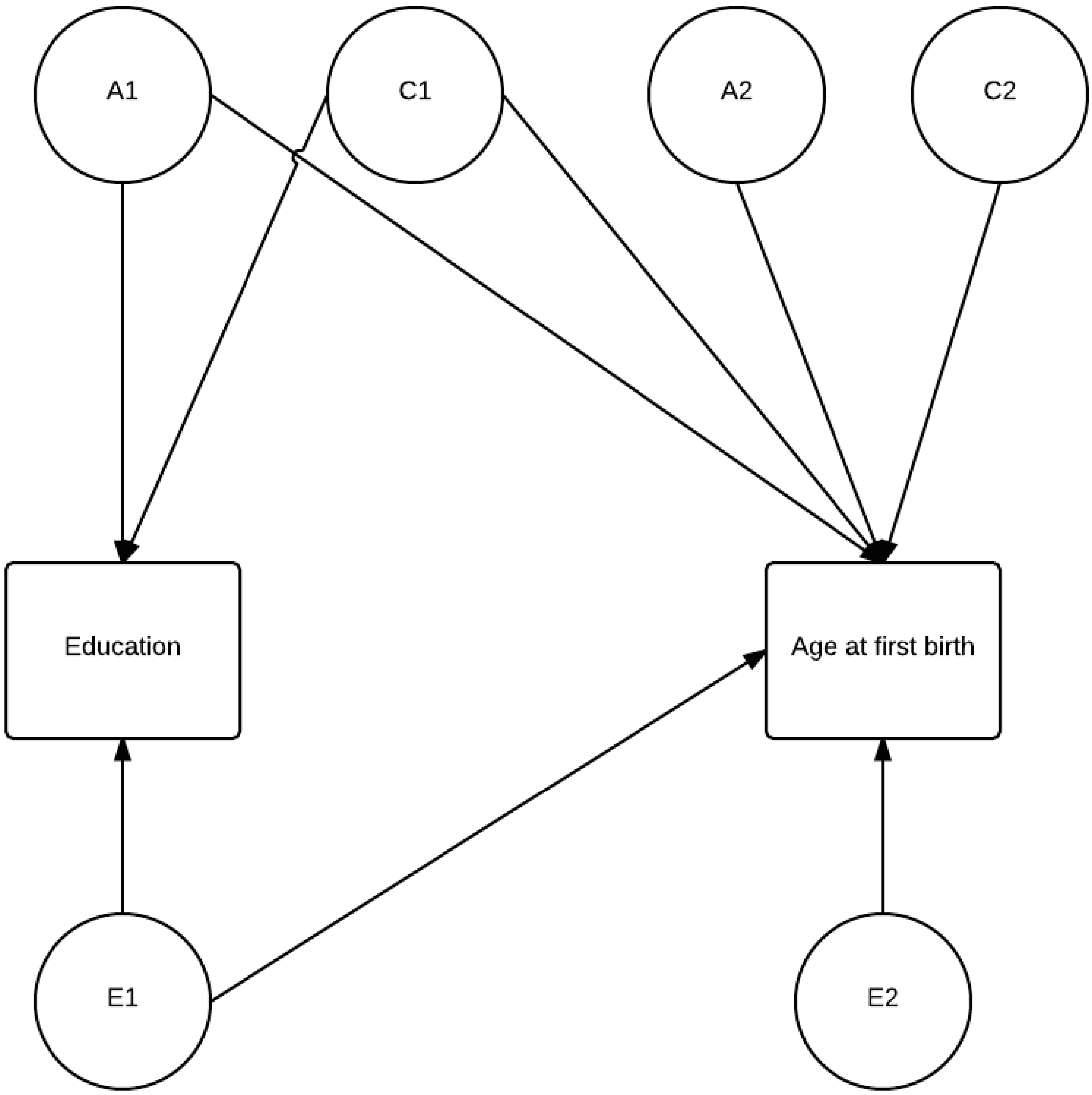
The within (identical) twin approach we applied forms a useful tool to establish the causal relationship between two variables yet it has its limitations. The most critical assumption is presumably that variation in education within twin pairs is uncorrelated with variation in age at first birth. The question arises, what makes (identical) twins different in their 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 towards longer educational and later fertility, the true causal effect would be smaller than the estimate from the within-twin models. Alternative designs can validate our findings in future research. Desirable approaches are quasi-experimental designs using instrument variables (IV), such as the aforementioned educational acts in Great Britain (GIVE THE REFERENCE). However, note that such approaches also require large sample sizes and have critical assumptions of their own (for a recent discussion see Amin et al. 2015; Boardman and Fletcher 2015).

Finally, complementary biometric analyses revealed that the association between education and age at first birth is to a large extent caused by social family background effects, whereas genetic inheritance plays only a small role - which fits with previous studies (Neiss et al. 2002; Nisén et al. 2013; Rodgers et al. 2008). It needs to be mentioned that findings on the role of the genetic component in age at first birth are mixed in the literature. We find that around one third of the variance in age at first birth is explained by additive genetic effects. This is similar to studies from the US (Byars et al. 2010), Australia (Kirk et al. 2001) or Finland (Nisén et al. 2013). However, other investigations from the US (Neiss et al. 2002) and Denmark (Rodgers et al. 2008) find no significant genetic influences. Differences in genetic effects on fertility across countries and within countries over time exist – also within the UK (Tropf et al. 2015) - and may be due to gene-environment interaction (Kohler et al. 2006; Mills and Tropf 2015). A previous study suggests that genetic effect on education and age at first birth were shared (Nisén et al. 2013). Our results, in contrast, reveal that the genetic effect on age at first birth may be partly mediated by the causal effect of education. Genetic correlations do therefore not entirely represent genetic influences as a common cause for both outcomes, but genes may only influence age at first birth via education. Further investigation into the genetic pathways to fertility is needed, also in order to better understand patterns of gene-environment interaction across populations.

In general our study challenges the common approach to explain differences and trends in fertility timing mainly by 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).

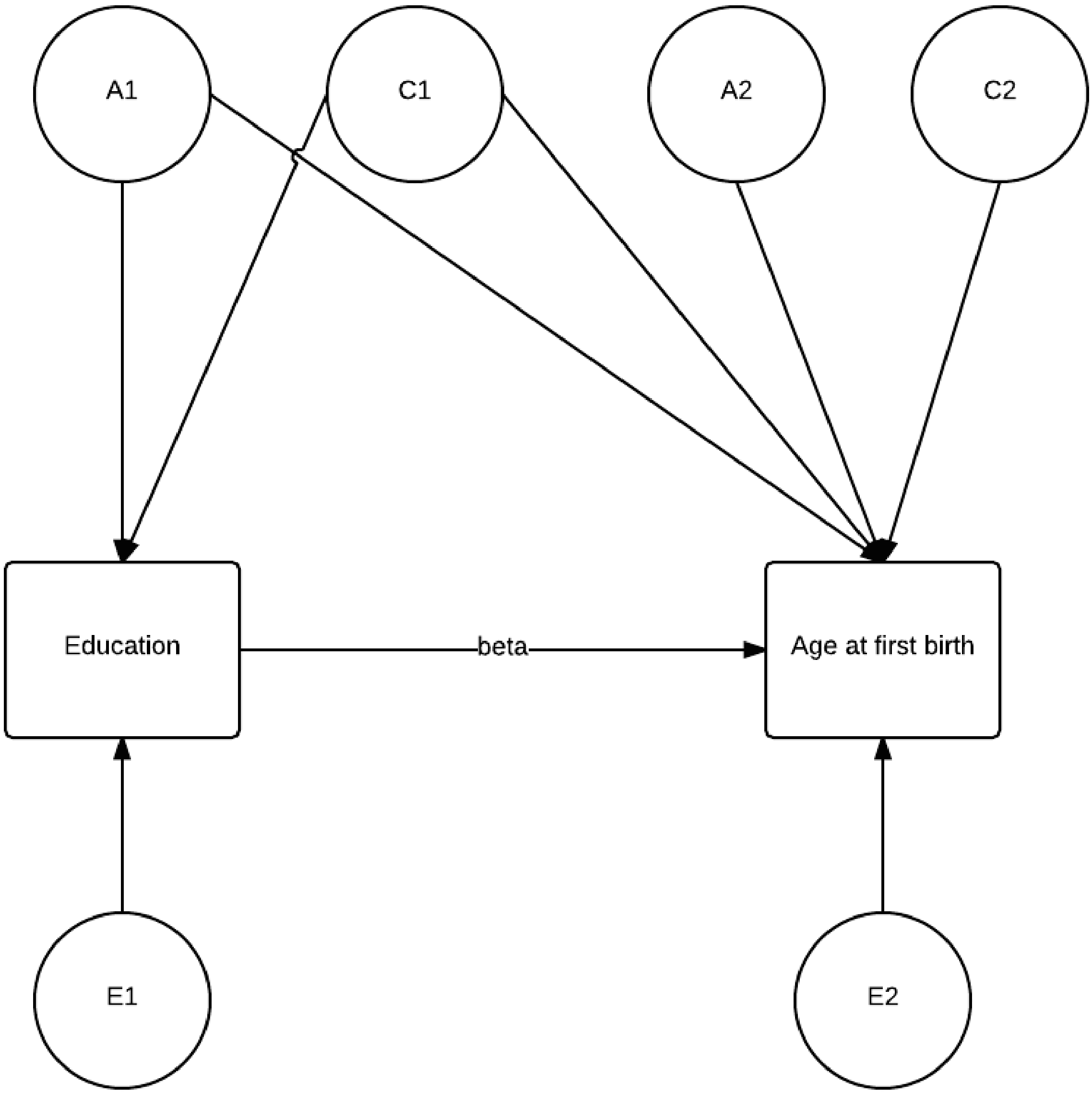
SUPPLEMENTARY MATERIAL

Figure S1 Bivariate ACE model.



Note: A1 = first genetic factor, A2 = second genetic factor, C1 = first shared environmental factors, C2 second shared environmental factors, E1 = first unique environmental factors, E2 = second unique environmental factor

Figure S2 Beta-ACE model.



A1 = first genetic factor, A2 = second genetic factor, C1 = first shared environmental factors, C2 second shared environmental factors, E1 = first unique environmental factors, E2 = second unique environmental factor, beta = causal effect of education on age at first birth

(Stratified) Cox regression models

In order to draw more general conclusions about the effect of education on 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 (Allison and Christakis 2006)**:**

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

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	1950.02	11.91	1919	1983	2164	1951.39	14.15	1923	1988	2234
Age of first birth/age at censoring	31.53	13.37	16	85	2164	31.77	12.89	15	86	2234
Status (having a child, in %)	78	--	0	1	2164	71	--	0	1	2234
Age at last observation (if censored)	51.11	15.58	21	85	476	45.71	15.88	20	86	644
Education	17.38	2.94	10	30	2164	17.71	2.96	13	30	2234
Sibling differences in AFB (pairs)	9.57	12.99	0	60	1082[a]	7.27	11.87	0	60	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 = refers to 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***	0.08
	(0.06)	(0.13)
Cohort (if birth year <1945 = 0; else 1)	-1.97	
	(1.43)	
Education*Cohort	0.11	0.04
	(0.08)	(0.14)
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
Source: TwinsUK, own calculations

Correlation, covariance and covariance components in biometric models

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

Variance components

Bivariate ACE model

The estimates of the latent factors are consistent across models. 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_{edu}^2 over the overall variance in education (caused by all underlying factors a_{edu}^2+c_{edu}^2+e_{edu}^2).

Model 1: $h_{edu}^2 \ = (a_{edu}^2/(a_{edu}^2+c_{edu}^2+e_{edu}^2)$ = $1.173^2\ /(1.173^2 + 1.244^2 + 1.436^2) = \ 0.46$

where h^2 or narrow-sense heritability represents the standardized additive genetic variance. For age at first birth the additive genetic variance consists of both, additive genetic variance shared between education and age at first birth ($a_{edu,afb}^2$) and genetic variance unique to age at first birth ($a_{afb,afb}^2$). The same applies to the environmental variance components:

$$h_{afb}^2 = (a_{afb,afb}^2 + \ a_{edu,afb}^2)/\ (a_{afb,afb}^2 + \ a_{edu,afb}^2+c_{afb,afb}^2 + \ c_{edu,afb}^2+e_{afb,afb}^2 + \ e_{edu,afb}^2)$$

= $(2.525^2 + \ 0.495^2)/((2.525^2 + \ 0.495^2 + 0^2 + 1.568^2 + 3.416^2 + 0.106^2$ = 0.32

Shared and unique environmental variance components have been calculated in the same way.

*ACE-beta model ((*Kohler et al. 2011)*)*

The estimate of heritability for education is the same as in the ACE model, however, for heritability of age at first birth it changes because 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.

$$h_{afb}^2 = \frac{\beta^2 a_{edu}^2 + 2\beta a_{edu} a_{edu,afb} + a_{edu,afb}^2 + a_{afb,afb}^2}{\sigma_{afb}^2}$$

where $\sigma_{afb}^2 = \beta^2(\sigma_{edu}^2)^2 + 2\beta(a_{edu}a_{edu,afb} + c_{edu,afb} + c_{afb,afb}) + (a_{afb,afb}^2 + a_{edu,afb}^2 + c_{afb,afb}^2 + c_{edu,afb}^2 + e_{afb,afb}^2)$.

Correlation, Covariance and Covariance Components

ACE-model

The correlation of the latent factors in the ACE model, for example for the shared environmental effects is:

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

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.

If we relate the observed covariance of shared environmental factors across outcomes to the overall observed covariance, we find the estimated contribution of shared environmental factors to the observed correlation, which is $c_{edu}c_{edu,afb}/(a_{edu}a_{edu,afb} + c_{edu}c_{edu,afb} + e_{edu}e_{edu,afb})$ = 0.73, so that 73% of the observed correlation is due to shared environmental factors.

ACE-beta model

In the ACE-beta model, again the fixed-effect estimate instead of the covariance of unique environmental influences contribute to the overall covariance.

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

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

Model	1		2	
	Bivariate ACE		ACE-beta	
	estimate	se	estimate	se
education				
a	1.176***	.115	-1.176***	.115
c	-1.244***	.148	-1.244***	.147
e	-1.436***	.039	-1.436***	.039
age at first birth				
a	2.525***	.224	2.525***	.221
c	0.000	1.152	0.000	.943
e	3.416***	.087	3.416***	.087
cross-trait effects				
a	-0.495*	.293	-0.365	.392
c	-1.568***	.293	1.477***	.314
e	-0.106	.129	-	-
Beta	-		0.074	.089

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

References

Allison, P. D., & Christakis, N. (2006). Fixed-effects methods for the analysis of nonrepeated events. *Sociological Methodology*, *36*(1), 155–172. doi:10.1111/j.1467-9531.2006.00177.x

Amin, V., Behrman, J. R., Kohler, H.-P., Xiong, Y., & Zhang, J. (2015). Causal inferences: Identical twins help and clarity about necessary assumptions is critical. *Social Science & Medicine*, *127*, 201–202. doi:10.1016/j.socscimed.2014.12.012

Amin, V., Behrman, J. R., & Spector, T. D. (2013). Does more schooling improve health outcomes and health related behaviors? Evidence from UK twins. *Economics of education review*, *35*, 134–148.

Amuedo-Dorantes, C., & Kimmel, J. (2005). The motherhood wage gap for women in the United States: The importance of college and fertility delay. *Review of Economics of the Household*, *3*(1), 17–48.

Andersson, G. (2000). The impact of labour-force participation on childbearing behaviour: pro-cyclical fertility in Sweden during the 1980s and the 1990s. *European Journal of Population/Revue européenne de …*.

Balbo, N., & Barban, N. (2014). Does fertility behavior spread among friends? *American Sociological Review*, *79*(3), 412–431.

Balbo, N., Billari, F. C., & Mills, M. C. (2013). Fertility in advanced societies: A review of research. *European Journal of Population/Revue Européenne de Démographie*, *29*(1), 1–38.

Bhrolcháin, M. N., & Beaujouan, É. (2012). Fertility postponement is largely due to rising educational enrolment. *Population Studies*, *66*(3), 311–327.

Billari, F. (2015). Integrating macro-and micro-level approaches in the explanation of population change. *Population studies*.

Blossfeld, H.-P., & Huinink, J. (1991). Human capital investments or norms of role transition? How women's schooling and career affect the process of family formation. *American journal of Sociology*.

Blossfeld, H.-P., & Jaenichen, U. (1992). Educational expansion and changes in women's entry into marriage and motherhood in the Federal Republic of Germany. *Journal of Marriage and the Family*, 302–315.

Boardman, J. D., & Fletcher, J. M. (2015). To cause or not to cause? That is the question, but identical twins might not have all of the answers. *Social Science & Medicine*, *127*, 198–200. doi:10.1016/j.socscimed.2014.10.013

Boker, S., Neale, M., Maes, H., Wilde, M., Spiegel, M., Brick, T., et al. (2011). OpenMx: an open source extended structural equation modeling framework. *Psychometrika*, *76*(2), 306–317.

Branigan, A. R., McCallum, K. J., & Freese, J. (2013). Variation in the heritability of educational attainment: An international meta-analysis. *Social forces*, *92*(1), 109–140.

Byars, S. G., Ewbank, D., Govindaraju, D. R., & Stearns, S. C. (2010). Natural selection in a contemporary human population. *Proceedings of the National Academy of Sciences*, *107*(suppl 1), 1787–1792.

Cohen, J. (2011). Childbearing impeded education more than education impeded childbearing among Norwegian women. *Proceedings of the National Academy of Sciences*.

D'Onofrio, B. M., Lahey, B. B., Turkheimer, E., & Lichtenstein, P. (2013). Critical Need for Family-Based, Quasi-Experimental Designs in Integrating Genetic and Social Science Research. *American Journal of Public Health*, *103*(S1), S46–S55. doi:10.2105/AJPH.2013.301252

de Kaa, D. J. Van. (1987). Europe's second demographic transition. *Population bulletin*, *42*(1), 1–59.

Freese, J. (2008). Genetics and the Social Science Explanation of Individual Outcomes1. *American Journal of Sociology*, *114*(S1), S1–S35.

Gustafsson, S., Kenjoh, E., & Wetzels, C. (2002). *The role of education on postponement of maternity in Britain, Germany, the Netherlands and Sweden*.

Happel, S. K., Hill, J. K., & Low, S. A. (1984). An economic analysis of the timing of childbirth. *Population Studies*, *38*(2), 299–311.

Hobcraft, J. (1996). Fertility in England and Wales: a fifty-year perspective. *Population studies*, *50*(3), 485–524.

Hoem, B. (2000). Entry into motherhood in Sweden: the influence of economic factors on the rise and fall in fertility, 1986-1997. *Demographic research*.

Hoffman, S., Foster, E., & Jr, F. F. (1993). Reevaluating the costs of teenage childbearing. *Demography*.

Jæger, M. M. (2012). The Extended Family and Children's Educational Success. *American Sociological Review*, *77*(6), 903–922.

Joshi, H. (2002). Production, reproduction, and education: Women, children, and work in a British perspective. *Population and Development Review*, *28*(3), 445–474.

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

Natural selection and quantitative genetics of life‐history traits in Western women: a twin study. *Evolution*, *55*(2), 423–435.

Kohler, H.-P., Behrman, J. R., & Schnittker, J. (2011). Social science methods for twins data: Integrating causality, endowments, and heritability. *Biodemography and social biology*, *57*(1), 88–141.

Kohler, H.-P., Billari, F. C., & Ortega, J. A. (2002). The emergence of lowest‐low fertility in Europe during the 1990s. *Population and development review*, *28*(4), 641–680.

Kohler, H.-P., & Rodgers, J. L. (2003). Education, fertility and heritability: Explaining a paradox. *Offspring: Human fertility behavior in biodemographic perspective*, 46–90.

Kohler, H.-P., Rodgers, J. L., & Christensen, K. (1999). Is fertility behavior in our genes? Findings from a Danish twin study. *Population and Development Review*, *25*(2), 253–288.

Kohler, H.-P., Rodgers, J. L., Miller, W. B., Skytthe, A., & Christensen, K. (2006). Bio‐social determinants of fertility. *International journal of andrology*, *29*(1), 46–53.

Kravdal, Ø. (2002). The impact of individual and aggregate unemployment on fertility in Norway. *Demographic Research*.

Lappegård, T., & Rønsen, M. (2005). The multifaceted impact of education on entry into motherhood. *European Journal of Population/Revue européenne de Démographie*, *21*(1), 31–49.

Lesthaeghe, R. (1995). The second demographic transition in Western countries: An interpretation. *Gender and family change in industrialized countries*, 17–62.

Liefbroer, A., & Corijn, M. (1999). Who, what, where, and when? Specifying the impact of educational attainment and labour force participation on family formation. *Journal of Population/Revue Européenne de Démographie*, *15*(1), 45–75.

Lutz, W., Butz, W., & Samir, K. (2014). *World population and human capital in the twenty-first century*.

Lyngstad, T. H., & Prskawetz, A. (2010). Do Siblings' Fertility Decisions Influence each Other? *Demography*, *47*(4), 923–934.

Marini, M. M. (1984). Women's educational attainment and the timing of entry into parenthood. *American Sociological Review*, 491–511.

Marini, M. M. (1985). Determinants of the timing of adult role entry. *Social science research*, *14*(4), 309–350.

Martin, S. P. (2000). Diverging fertility among U.S. women who delay childbearing past age 30. *Demography*, *37*(4), 523–533.

McCrary, J., & Royer, H. (2011). The effect of female education on fertility and infant health: Evidence from school entry policies using exact date of birth. *American Economic Review*, *101*(1), 158–195.

Miller, A. R. (2011). The effects of motherhood timing on career path. *Journal of Population Economics*, *24*(3), 1071–1100. doi:10.1007/s00148-009-0296-x

Miller, W. B., Bard, D. E., Pasta, D. J., & Rodgers, J. L. (2010). Biodemographic modeling of the links between fertility motivation and fertility outcomes in the NLSY79. *Demography*, *47*(2), 393–414.

Mills, M. C., Rindfuss, R. R., McDonald, P., & te Velde, E. (2011). Why do people postpone parenthood? Reasons and social policy incentives. *Human Reproduction Update*, *17*(6), 848–860. doi:10.1093/humupd/dmr026

Mills, M. C., & Tropf, F. C. (2015). The Biodemography of Fertility: A Review and Future Research Frontiers. *Kölner Zeitschrift für Soziologie und Sozialpsychologie*, *55*(Special Issues Demography), 397–424.

Moayyeri, A., Hammond, C., & Spector, T. D. (2013). Cohort profile: TwinsUK and healthy ageing twin study. *International journal of epidemiology*, *42*(1), 76–85.

Murphy, M. (1993). The contraceptive pill and women's employment as factors in fertility change in Britain 1963–1980: a challenge to the conventional view. *Population Studies*, *47*(2), 221–243.

Murphy, M. (1999). Is the relationship between fertility of parents and children really weak? *Biodemography and Social Biology*, *46*(1-2), 122–145.

Neale, M. C., & Cardon, L. R. (1992). *Methodology for genetic studies of twins and families*. Springer.

Neiss, M., Rowe, D. C., & Rodgers, J. L. (2002). Does education mediate the relationship between IQ and age of first birth? A behavioural genetic analysis. *Journal of Biosocial Science*, *34*(2), 259–276.

Nisén, J., Martikainen, P., Kaprio, J., & Silventoinen, K. (2013). Educational Differences in Completed Fertility: A Behavioral Genetic Study of Finnish Male and Female Twins. *Demography*, 1–22.

Nisén, J., & Myrskylä, M. (2014). Effect of family background on the educational gradient in lifetime fertility of Finnish women born 1940–50. *Population studies*, *68*(3), 321–337.

Office National Statistics. (2013). Cohort Fertility, Table 2. http://www.ons.gov.uk/ons/publications/re-reference-tables.html?edition=tcm:77-263133

Oppenheimer, V. K. (1994). Women's Rising Employment and the Future of the Family in Industrial Societies. *population and development review*, *20*(2), 293–342. doi:10.2307/2137521

Oreopoulos, P. (2006). Estimating average and local average treatment effects of education when compulsory schooling laws really matter. *The American Economic Review*.

Pike, A., & Kretschmer, T. (2009). Shared versus nonshared effects: parenting and children's adjustment. *International Journal of Developmental Science*, *3*(2), 115–130.

Rendall, M., Aracil, E., Bagavos, C., Couet, C., Derose, A., Digiulio, P., et al. (2010). Increasingly heterogeneous ages at first birth by education in Southern European and Anglo-American family-policy regimes: A seven-country comparison by birth cohort. *Population studies*, *64*(3), 209–227. doi:10.1080/00324728.2010.512392

Rendall, M., Couet, C., Lappegard, T., Robert-Bobée, I., Rønsen, M., & Smallwood, S. (2005). First births by age and education in Britain, France and Norway. *Population trends*, (121), 27–34.

Rietveld, C. A., Medland, S. E., Derringer, J., Yang, J., Esko, T., Martin, N. W., et al. (2013). GWAS of 126,559 Individuals Identifies Genetic Variants Associated with Educational Attainment. *Science, 340*(6139), 1467–1471.

Rijken, A. J., & Liefbroer, A. C. (2009). Influences of the family of origin on the timing and quantum of fertility in the Netherlands. *Population studies*, *63*(1), 71–85.

Rindfuss, R. R., Guilkey, D., Morgan, S. P., Kravdal, O., & Guzzo, K. B. (2007). Child care availability and first-birth timing in Norway. *Demography*, *44*(2), 345–372. doi:10.1353/dem.2007.0017

Rindfuss, R. R., John, C. St., & Bumpass, L. L. (1984). Education and the timing of motherhood: Disentangling causation. *Journal of Marriage and the Family*, 981–984.

Rindfuss, R. R., & St. John, C. (1983). Social Determinants of Age at First Birth. *Journal of Marriage and the Family*, *45*(3), 553–565.

Rodgers, J. L., Kohler, H. P., Kyvik, K. O., & Christensen, K. (2001). Behavior genetic modeling of human fertility: Findings from a contemporary Danish twin study. *Demography*, *38*(1), 29–42.

Rodgers, J. L., Kohler, H. P., McGue, M., Behrman, J. R., Petersen, I., Bingley, P., & Christensen, K. (2008). Education and cognitive ability as direct, mediating, or spurious influences on female age at first birth: behavior genetic models fit to Danish twin data. *AJS; American Journal of Sociology*, *114*(Suppl), S202.

Scott, J. (2004). Family, gender, and educational attainment in Britain: A longitudinal study.

Journal of Comparative Family Studies.

Skirbekk, V., & Prskawetz, A. (2006). *The marginal effect of school leaving age on demographic events. A contribution to the discussion on causality*.

Steenhof, L., & Liefbroer, A. C. (2008). Intergenerational transmission of age at first birth in the Netherlands for birth cohorts born between 1935 and 1984: Evidence from municipal registers. *Population Studies*, *62*(1), 69–84.

Thornton, A. (1980). The influence of first generation fertility and economic status on second generation fertility. *Population and Environment*.

Tropf, F. C., Barban, N., Mills, M. C., Snieder, H., & Mandemakers, J. J. (2015). Genetic influence on age at first birth of female twins born in the UK, 1919-68. *Population Studies*, 129–145.

van Doorn, M., Pop, I., & Wolbers, M. H. J. (2011). Intergenerational transmission of education across European countries and cohorts. *European Societies*, *13*(1), 93–117.

Tables and Figures

Tables

Table 1. Summary statistics for monozygotic (MZ) and dizygotic (DZ) twins separately and within-twin absolute differences for the two main variables of interest.

	DZ					MZ				
	Mean	SD	Min.	Max.	N	Mean	SD	Min.	Max.	N
Year born	1947.02	9.37	1919	1969	1398	1945.66	10.1	1924	1969	1354
Age at first birth	25.79	4.66	16	44	1398	25.87	4.44	15	44	1354
Education (age)	16.9	2.67	12	30	1398	16.88	2.49	13	30	1354
Within-twin absolute differences										
age at first birth	4.21	3.75	0	20	699[a]	3.41	3.27	0	22	677[a]
education	1.33	2.4	0	15	699[a]	0.88	1.8	0	15	677[a]

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

Table 2. Linear OLS and within-twin (FE) regression on age at first birth. Unstandardized estimates and standard errors in brackets.

	Full sample		MZ twins only		DZ twins only	
Model	1	2	3	4	5	6
	OLS	FE	OLS	FE	OLS	FE
Education	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	2752	2752	1354	1354	1398	1398
N pairs	1376	1376	677	677	699	699

* p<0.05, ** p<0.01, *** p<0.001, one-sided, OLS standard errors corrected for non-independence of twins, FE= within twin model with fixed effect on family level.
Source: TwinsUK, own calculations.

Table 3. Bivariate ACE and ACE-beta model on age at first birth and education.

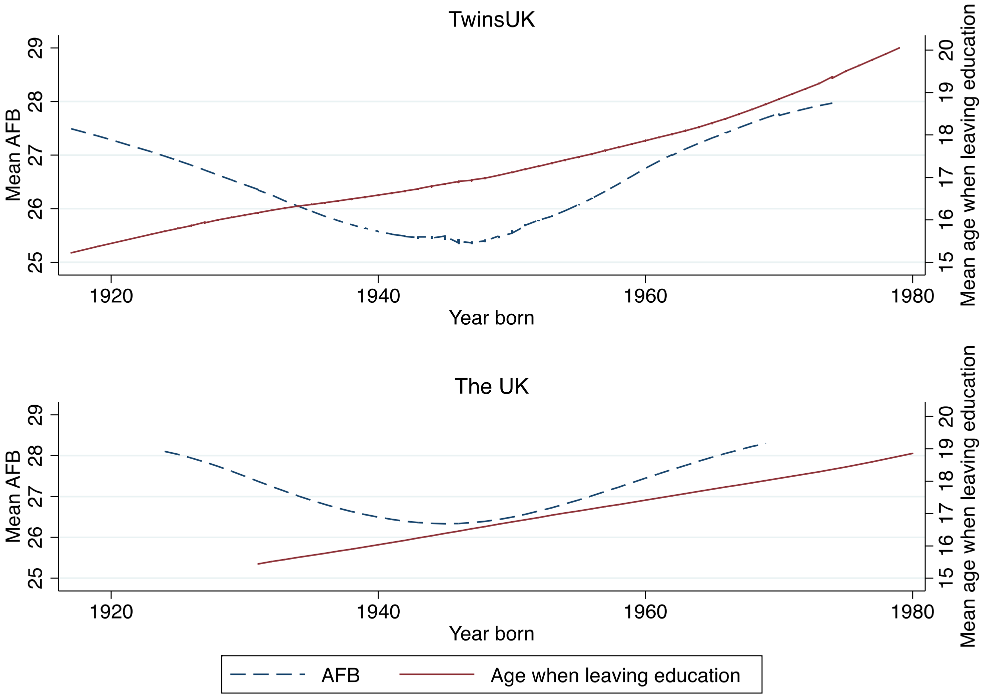
	Bivariate ACE	ACE-beta
Variance components		
Education		
a^2	0.46***	0.46***
	(0.06)	(0.06)
c^2	0.23***	0.23***
	(0.05)	(0.05)
e^2	0.31***	0.31***
	(0.02)	(0.02)
Age at first birth		
a^2	0.32***	0.32***
	(0.06)	(0.06)
c^2	0.12***	0.12***
	(0.05)	(0.05)
e^2	0.56***	0.56***
	(0.03)	(0.03)
Correlation of variance components		
r(a)	0.19*	0.14
	(0.10)	(0.15)
r(c)	1.0***	1.0***
	(0.00007)	(0.00007)
r(e)	0.03	-
	(0.04)	
B	-	0.07 (0.09)
Contribution to education-age at first birth link		
r(a) (additive genetic)	29	21
r(c) (shared environment)	66	62
r(e) (unique environment)	5	--
B (direct education effect)	--	17
N	2,752	2,752
N pairs	1,326	1,326

* p<0.05, ** p<0.01, *** p<0.005, two-sided

Source: TwinsUK, own calculations.

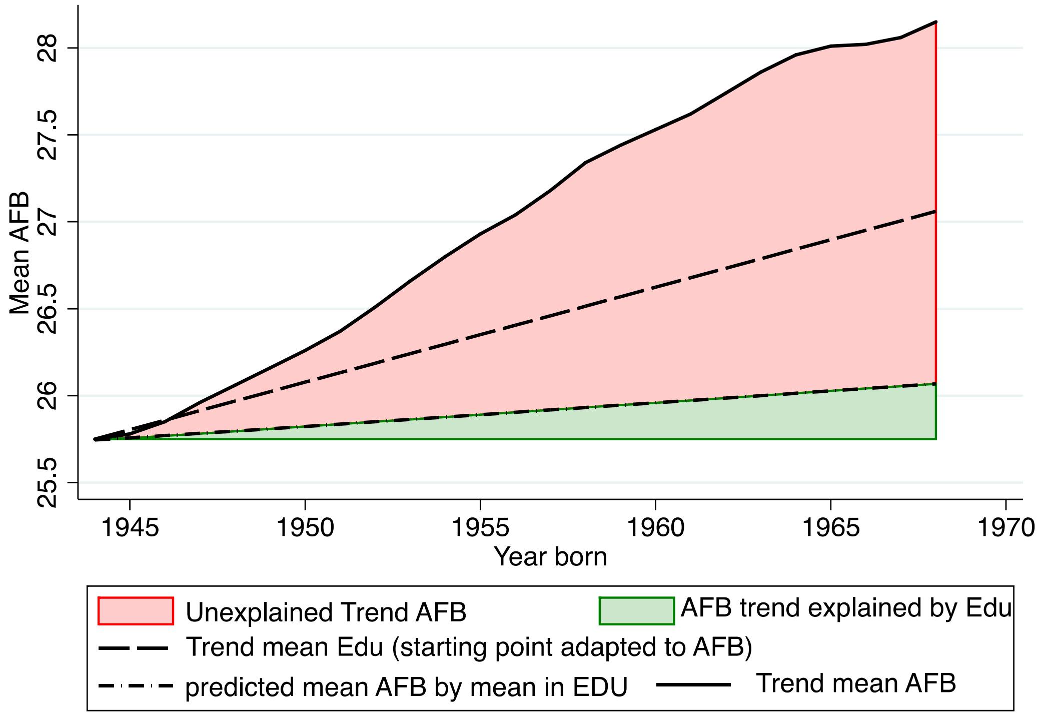
Figures

Figure 1. Trends in mean age at first birth and mean age when leaving full-time education in the TwinsUK sample and national representative data.



Source: TwinsUK and data from the National Office for Statistics (for details see methods section). AFB = age at first birth.

Figure 2. Can educational expansion explain postponement of childbearing since 1945? Explained and unexplained trends in age at first birth since 1945. Observed mean age at first birth (AFB), Education (EDU) and predicted mean AFB by EDU using the within MZ twin estimate of education.



Notes: Edu = age at leaving full-time education, the graph depicts the trend in the mean age at leaving education in year, whereas the starting point is adapted to the AFB, AFB =age at first birth, Predictions are based on estimates from Model 2, Table 2, and data from Table 1.

Source: Edu = British General Household Survey (2000-2006), AFB = Office for National Statistics, Cohort fertility, Table 2. (Office National Statistics 2013)