A Behavioral Genetic Study of Age at First Birth of female UK Twins

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Abstract. There is a rising interest in latent family factors influencing fertility, particularly in the effects of genes and the family environment (Rodgers et al., 2001; Nisén, et at., 2013). This study investigates the genetic and environmental influence on the age at first birth (AFB) for women from the United Kingdom. Using data from TwinsUK that contain 2,274 monozygotic (945, 42 %) and dizygotic (1,329, 58 %) twin pairs born from 1919-1968, we apply behavioral genetics models in an innovative tobit framework to also include censored women who remained childless. The findings indicate that a substantial part (40 %) of the variation in AFB is caused by latent family characteristics. Genetic dispositions (26 %) play a more important role than the shared environment of siblings (14 %), with the non-shared environment having the strongest influence (60 %). A trend analysis across cohorts reveals large changes in estimations over time. While genetic influence is low for individuals who started childbearing during the Second World War and the economic crises of the 1970s, there is high heritability for the cohorts who started childbearing in times of changing mores and the sexual freedom (1950s and 1960s) and in most recent cohorts. This pattern supports the idea that environmental constraints suppress and individual freedom triggers the manifestation of genetic predispositions for fertility. A key contribution is that we demonstrate that the exclusion of censored information can severely bias results, offering a potential explanation for the mixed findings of previous studies.

Keywords: age at first birth, fertility, United Kingdom, heritability, genes, gene environment interaction
INTRODUCTION

There has been a massive delay in the age at first birth (AFB) across Europe since 1970, which is now between 28-29 years (Mills, Rindfuss, McDonald, &teVelde, 2011), often referred to as the ‘postponement’ transition (Kohler, Billari& Ortega, 2002). Primary reasons for postponement have been attributed to women’s increased move to obtain higher education and an employment career, but also cultural transformations surrounding the timing and role of children (Van de Kaa, 1987; Balbo, Billari& Mills, 2013). Explanations to explain differences and changes in AFB patterns within demography and the social sciences continues to almost exclusively rely on associations with socio-environmental factors (Goldstein, Sobotka, &Jasilioniene, 2009; Hobcraft, 2006). Yet a growing body of literature indicates interplay between individual genetic endowment and the environment, which in turns shapes subsequent life-course decisions and fertility behavior (Freese, 2008; Guo, 2005; Kohler, Rodgers, Miller, Skytthe, & Christensen, 2006; Murphy, 1999; Udry, 1996). Significant genetic influences related to fertility have been established for the age at first date or marriage (Mealey& Segal, 1993), age at first sexual intercourse (Dunne et al., 1997), number of sexual partners (Guo, Tong, &Cai, 2008) and likelihood of unprotected sexual intercourse (Daw&Guo 2011). One of the strongest genetic effects was found in a twin study examining the first attempt to have a child, where 40-50 % of the variance was explained by genetic differences (Kohler, Rodgers, & Christensen, 1999; Rodgers, Kohler, Kyvik, & Christensen, 2001).

A prominent technique to determine the extent to which genes or the environment explain AFB is the use of a twin study design. Twin data represent some of the best resources for evaluating the importance of genetic variation in observed traits by facilitating comparisons between monozygotic (MZ) and dizygotic (DZ) twins (Boomsma, Busjahn, &Peltonen, 2002). Existing research examining AFB that adopts this design, however, has produced very mixed findings. Whereas an Australian study found significant genetic
influences (Kirk et al., 2001), research in the U.S. (Neiss et al., 2002) and Denmark (Rodgers et al., 2008) report non-significant effects. The aim of this study is to extend existing knowledge on the influence of genetic and environmental factors on AFB in a significant way by not only replicating research on a new population of twins in the UK, but by introducing new analytical techniques that we demonstrate provide the potential to explain the mixed findings of previous results.

Our central research questions ask to what extent genes and environment explain differences in the UK, how this pattern has changed for cohorts born over the twentieth century and how results differ depending upon whether we apply the classic approach used until now or a new technique that we introduce which includes censored information.

The current study builds upon, but also extends the existing literature in three primary ways. The first key innovation is that we introduce more sophisticated statistical models and then demonstrate that they produce different results. The core difference is the handling of right-censored observations (i.e., individuals that did not have a first birth by the last time of observation). It appears that the studies that include childless women (Kirk et al., 2001) find significant genetic effects. Conversely, those that ignored censoring and thus only included women who had a first child, show weak or no genetic influences (e.g., Neiss et al. 2002; Rodgers et al. 2008). This study empirically examines whether the inclusion of censored information affects heritability estimates and if so, to what extent. This is achieved by introducing a tobit survival model into the behavioral genetic framework, which includes censored cases and then compare these to the estimates of the classic linear structural equation (SEM) models that exclude censored information.

A second contribution is the application of local linear regressions for each birth cohort in order to more explicitly describe underlying trends (Cleveland, 1979). This allows us to explore changes in the influence of latent factors contributing to AFB during the last century. Kohler et al. (1999s; 2002) demonstrated that the heritability of the number of children, for
instance, strongly depends on demographic changes during the past century. Our study uses the rich information from a large-scale twins register, TwinsUK, in the United Kingdom, which covers information on birth cohorts from 1919-1968. To align our results with previously genetically-informed research, we divide our samples to the cohorts investigated previously in Denmark (Kohler et al. 1999a; 2002). It is essential to examine cohort differences since the twentieth-century UK has been a setting of distinct social upheavals that strongly impacted AFB (Hobcraft, 1996, 2006; Murphy, 1993).

A third and related contribution is the study of this topic on a new population of UK women. Human traits and behaviour are the result of interplay between genetic and environmental factors (Freese, 2008). Genes provide predispositions for complex traits such as AFB, but environmental conditions determine whether such dispositions will be realized. To properly understand gene-environment interactions, we must therefore evaluate the estimated heritability of a trait across different environmental contexts (Guo, 2005). In substantive terms, the UK is also an interesting case since intergenerational correlations in fertility and AFB are well established and family background has been shown to be a key predictor of fertility over the twentieth century (Booth &Kee, 2009; Murphy, 2009). It is therefore compelling to understand in how far this association is influenced by genetic or environmental determinants.

The data used in this study come from TwinsUK, the largest adult twin registry in the UK, which consists of 12,000 twins and is primarily used to study the genetic and environmental aetiology of age related complex traits and diseases. We examine women only, with data available for 2,274 female twin pairs (and thus 4,548 individuals). In the following section we briefly introduce behavioral genetics concepts. We then review previous research on familial and genetic effects on fertility and introduce the TwinsUK dataset and our methods. This is followed by a presentation and discuss our findings.
BACKGROUND

INTERGENERATIONAL CORRELATIONS IN FERTILITY

The motivation to disentangle effects of shared genetic factors from shared family environment on fertility has early roots. Fisher (1930) conducted a study on the number of offspring among more than 2,000 British aristocrats at the end of the nineteenth-century and found an intergenerational correlation of .20. Murphy (1999) concluded that intergenerational correlations in fertility are very common and even increased ever since the end of the nineteenth-century century.

Whereas intergenerational correlations in fertility seem to follow a consistent pattern, their interpretations remain controversial. One explanation might be that parents each transmit 50% of their genetic variants and thus genetic predispositions to their children. Therefore, Fisher (1930) interpreted the intergenerational correlation of .20 entirely as a genetic effect, resulting in an explanation that 40% of the variance was explained by genetics. Conversely, others (Booth & Kee, 2009) argue that parents also transmit environmental characteristics such as socio-economic status to their children, which are important for fertility behavior. Intergenerational correlations from parents to children, therefore, lack valid interpretations in terms of their genetic or environmental origins and alternative designs such as twin studies are required to quantify the genetic and environmental contribution to fertility outcomes.

GENETIC RESEARCH ON HUMAN FERTILITY

The majority of fertility research adopting a twin design uses data from a historical Danish Twin registry, including virtually every twin pair born since 1870. Using this data, Kohler et al. (1999a; 2002) found evidence for a genetic influence on the number of children. They report that particularly after the strong fertility decline of the First Demographic Transition at the end of the nineteenth-century and Second Demographic Transitions in the second half of the twentieth-century, heritability as the proportion of genetic variance over the population
variance was at a moderate level of .40 for the number of children. In the interim phases, this proportion came close to zero.

Large changes in heritability after the Demographic Transitions support the hypothesis of gene-environment interaction. The authors associate these changes in genetic influence with the environmental changes during the Demographic Transitions. In particular, there were improvements in economic, medical and hygiene conditions during the First Demographic Transition, with the Second Demographic Transition characterized by the introduction of the pill and cultural transformations relaxing fertility norms (Van de Kaa, 1987). The underlying assumption is that genetic predispositions gain importance for fertility in environments that are less restrictive in their fertility choice. Genetically driven fertility preferences have a stronger impact on fertility outcomes in societies where individual choice predominates (Udry, 1996). Demographic Transitions provided such environments and in particular the introduction of the contraceptive pill during the Second Demographic Transition offered new freedom in the timing of the first child (Van de Kaa, 1987).

Research examining the level of early fertility (number of children at the age at which 25% of cohort members had a first child) in the Danish sample suggests the same pattern of gene-environment interaction as in the number of children (Kohler et al., 2002). The level of early fertility showed heritability of .52 for cohorts from 1961-1968 who were socialized after the Second Demographic Transition when fertility norms lost influence. For the cohorts born from 1945-1952, this effect is close to zero.

Investigations of the genetic and environmental influences on AFB, however, have shown mixed results. Neiss et al. (2002) conducted a study in the U.S. using constructed kinship data from the Longitudinal Survey of Youth. Based on information such as whether a person has lived with their biological mother and/or father, the relatedness of individuals in a household was identified (e.g., cousins, half siblings, full siblings and so forth). For cohorts born from 1958-1965, they found only low heritability of .06 for AFB, whereas the shared
environment explained up to 20% of the observed variance. Rodgers et al.’s (2008) study of twins from the Danish sample reports similar results. For birth cohorts from 1931-1952 (1,242 twins), the shared environment explained 26% of the variance in AFB and no genetic effect was found.

An Australian twin study, in contrast, reported a significant heritability of AFB. Kirk et al. (2001) investigated the common genetic factors of the age at menarche, age at first birth, age at menopause and fitness as a function of number of children (N=2,710 twins). Results showed that for cohorts born from 1900-1965, 21% of the observed variance was explained by genetic variance. The shared environment explained 18% of the observed variance and the non-shared environment accounted for the largest portion of 61%. At the same time, their multivariate biometric models provided evidence that genes influencing AFB were associated with overall reproductive success and therefore the survival of the genetic variants.

A recent investigation from Finland (Nisén et al., 2013) on cohorts born from 1950-1957, shows similar results. Genes explained 26% of the variance in AFB, shared environment 12% and 61% for the non-shared environment. At the same time, there is a genetic correlation between education and AFB indicating that genes provide an endogenous source for covariation for these traits.

Overall, the degree to which genes and the environment influence AFB appears to vary depending on the spatial and temporal environment. Additionally, the treatment of censored information might play a crucial role for the estimation. While the studies from the US, Denmark and Finland only included individuals who experienced AFB, the Australian models also included childless individuals by imputing the age at last observation, categorizing the ages and estimating an ordered SEM. For this reason, progress in this area of research should focus on the pursuit of better models that have the ability to capture censored cases.
In the following section we first describe the TwinsUK register which we use for historical comparison within the UK and then introduce the different model specifications we apply to gain insight in the consequences of excluding censored information. In contrast to previous historical research, our study focuses entirely on AFB. However, AFB is strongly related to the number of children and changes in number of children during the twentieth-century have been accompanied by postponement of childbearing (Balbo et al., 2012; Mills et al., 2011). Therefore, we expect similar patterns of genetic and environmental influences.

**METHOD**

**DATA**

The subjects of our sample are MZ and DZ twins, who voluntarily participated in surveys of the TwinsUK registry. The project involves more than 12,000 individuals and 60,000 observations since 1992 (Moayyeri et al., 2012) and represents the largest adult twin registry in the UK. It has primarily been conducted to answer questions of aetiology and epidemiology, but also contains demographic information.

Aligning our approach to the previously described research on Danish twins’ number of children and early fertility to make more direct comparisons, we focus on cohorts born before 1968. Information about the AFB is available in the main questionnaire and additionally in a behavioral questionnaire from 2005. In the instance where no AFB has been reported, we used information on the number of children to indicate whether the information about the fertility history is missing or the individual remained childless.

Using the main questionnaire we have a sample of 4,989 individuals. The behavioral questionnaire adds 1,663 individuals. The combined sample of 6,650 was cleaned by removing the following cases: 557 twin pairs are removed because of different gender or missing values...
in their demographic information. For 238 individuals, zygosity is missing and 223 twin pairs are males. 24 individuals reported a different race than Caucasian. Finally, 280 subjects had to be removed because no fertility information was available.

After applying these criteria, 4,548 individuals resulting in 2,274 complete twin pairs remain. Birth cohorts range from 1919-1968 and have been interviewed from 1992 and 2010. The average age of our sample is 57 (Table 1), thus the reproductive period is completed for most respondents.

Our univariate twin models consider AFB, age at censoring (C), zygosity and the siblings’ year of birth. Ages are computed by subtracting the year when a respondent is born from the reported year at first childbirth or the last year of observation in the case of right censoring, as in standard survival models (Mills, 2011). For right censored individuals, we use the age at last observation (ALO) as censoring age $C_i$ and replace it with age 45 if it is higher than that. This is the commonly assumed end of the reproductive life-span and the observation window of female fertility behavior (Leridon, 2008).

$$C_i = \begin{cases} ALO_i & \text{if } ALO_i \leq 45 \\ 45 & \text{if } ALO_i > 45 \end{cases}$$

(1)

In some cases both twins in a dyad did not participate in the same waves. We therefore use the life-history information of the most recent timepoint when information for both twins in a pair is available. Zygosity had been established using standardized questions about physical similarity and confirmed by multiplex DNA genotyping in cases of uncertainty (Ooki & Asaka, 2004).

To conduct the historical comparison, we follow two strategies. First, we divide the overall sample into four historical cohorts aligning to the previously described fertility research on Danish twins (Kohler et al. 2006). The oldest subsample contains the birth cohorts born from 1919-1944 (872 twin pairs, see Table 1). For this period, heritability of around .20
was reported for the number of children within the Danish sample (Kohler et al. 2002). The second subsample contains the birth cohorts born from 1945-1952 (663 twin pairs). In this cohort previous research found that the genetic influence on the number of children remained nearly at the same level (Kohler, Rodgers, & Christensen, 2002), while insignificant for AFB (Rodgers et al., 2008) and early fertility (Kohler et al., 2002). The third subsample consists of the cohort born from 1953-1960 (455 twin pairs), where heritability in the number of children and motivation for a first child increased to a moderate level of around .40. A study from Finland furthermore estimates heritability of .26 for AFB in cohorts born from 1950-1957 (Nisén et al. 2013). Finally, we analyze the cohorts born from 1961-1968 who were socialized during the Second Demographic Transition (284 twin pairs). In the Danish sample more than half of the variance in the level of early fertility was explained by genetic differences for these birth cohorts.

The second strategy more explicitly aims to describe the underlying trend of changes in the genetic and environmental influences on AFB. We therefore estimate local linear regressions for the latent factor estimations on the birth years separately (Cleveland, 1979). Results are presented in a smoothened scatterplot of a non-parametric regression of birth year on the results of the local regressions.

**BIO-SOCIAL MODELS AND THE TWIN DESIGN**

The aim of behavioral genetics or bio-social models (Udry, 1996) is to explain observed differences between individuals by differences in their genetic and environmental factors. Such models remain surprisingly rare in demographic research. This might partly be attributed to the predominant sociological and economic expertise of researchers in this area. Such models, furthermore, require data with genetic information, which is quite unusual in classic demographic study designs. However, an increasing number of studies emphasize the value of
multi- and interdisciplinary research in social science and demography (for an overview see Balbo et al., 2012; D'Onofrio&Lahey, 2010).

The most common way to disentangle the influence of the latent factors is to use the information in twin data. Identical (i.e., monozygotic twins (MZ)) are assumed to share common environmental influences such as their family members, the neighborhood they grew up in and other related aspects. More importantly, MZ are genetically identical (i.e., share all their genotypes). Fraternal, or dizygotic twins (DZ), in contrast, are akin to full siblings and assumed to share on average 50% of additive and 25% of their dominant genetic effects and assumed to share their family environment to the same extent as MZ twins. The degree to which MZ twins have a higher resemblance in their AFB than DZ twins, therefore, reflects the genetic influence (for details see Snieder, Wang, MacGregor, 2010; Boomsma et al., 2002; Snieder, Boomsma, van Doornen, & de Geus, 1997).

A naïve approach to estimate the proportion of explained variance by additive genetic effects is to compute the correlations of a trait separately for MZ and DZ twin pairs. Since they are assumed to share family environment to the same extent and MZ are twice as similar as DZ twins with regards to their genes, narrow sense heritability $h^2$ is two times the difference of the intra-group correlations of MZ and DZ. The effect of the shared environment of the twins is therefore the pairwise correlation of MZ minus $h^2$. Variance that is unexplained by these factors is due to non-shared environmental effects from outside or even within the family (Pike & Kretschmer, 2009) (including measurement errors: for details see Snieder et al., 2010). Based on this logic, genetic model fitting has become standard in twin research.

STRUCTURAL EQUATION MODELS
We fit SEMs to estimate the influence of genetic and environmental factors on AFB. The basic logic is in line with the comparison of intra-group correlations of twin pairs described above. Such correlations, however, have low power and large standard errors and do not make use of information available in variances and covariances (Snieder et al., 1997). SEM furthermore provides goodness of fit statistics thereby enabling us to test and compare alternative models (Snieder et al., 2010).

Figure 1 shows the ACE-path model. The capital letters in circles stand for the latent factors assumed to contribute to the observed variance in the sample. One-directional arrows refer to the directional non-standardized estimates of the respective variance components of the outcome. (a) represents additive genetic effects resulting from the sum of genetic effects of alleles\(^2\) from all contributing loci\(^3\). (c) are environmental effects resulting from environmental influences shared between twins of a pair and (e) are non-shared environmental effects resulting from the unique environment of an individual (including measurement error; for details see Snieder et al. 1997; 2010).

[FIGURE 1 HERE]

Bi-directional arrows indicate the assumed correlations between the latent variables for both groups of twins, which was introduced earlier. The unique environment (E) is assumed to be independent for both types of twins. The boxes contain the measured outcome variable \(Y_i\) of the member \(i \in \{1, 2\}\) of the twin pairs.

The basic structural equations in the model are:

\[
Y_1 = a \times (A_1) + c \times (C) + e \times (E_1) \tag{2}
\]

\[
Y_2 = a \times (A_2) + c \times (C) + e \times (E_2) \tag{3}
\]

whereas

\[
Cov (A_1, A_2) = \begin{cases} 
0.5 \text{ for DZ twin pairs} \\
1 \text{ for MZ twin pairs}
\end{cases} \tag{4}
\]

\(^2\) An allele is a variant of a gene for which different variants are possible.

\(^3\) A loci is a location of a gene on a chromosome.
and

\[ \text{Cov} (E_1, E_2) = 0 \]  

To gain empirically test how estimates differ between different kinds of censoring treatments, we estimate two models. First, we apply the classic SEM:

\[ Y_i = AFB_i \]  

where twin pairs with censored information do not contribute to the estimation, which has been the predominant way of modeling in the literature until now (Kohler et al., 1999b).

The exclusion of censored information, however, can have important implications such as biased inference of means and regression parameters. It furthermore reduces the sample size and in substantive terms it remains unclear whether the heritability patterns extend to twin pairs with censored information. The latter is in particular interesting, since the proportion of childless women in the UK increased during the twentieth century from about 10% in cohorts born around 1940 (at age 45) to about 20% in cohorts born around 1975 (at age 41). Childless women, therefore, become a group of growing demographic relevance (Rendall & Smallwood, 2003).

In a second step we introduce a tobit model using a recently developed lava R-package for the latent factor estimation (Holst & Budtz-Jørgensen, 2012). In this model, the \( AFB_i^* \) of an individual \( i \) is assumed to be a latent, normally distributed trait which cannot be observed over its entire range. If a woman has been interviewed before having a child, we do not know at what point in her future she would have one, whereas the end of the reproductive life-span at age 45 is the end of the observation window for all individuals. We therefore observe

\[ Y_i = \begin{cases} AFB_i^* & \text{if} \ AFB_i^* \leq C_i \\ C_i & \text{if} \ AFB_i^* > C_i \end{cases} \]  

The maximum likelihood estimation for censored cases is based on the probability to be censored given the observed values (see also Kohler et al. 1999b; Long, 1997).
standardize the estimates and report heritability and shared and non-shared environmental effects, we divide $a^2$, $c^2$, and $e^2$ with the overall variance of AFB—whereas the notations $h^2$ and $a^2$ are equivalent.

In the final stage of our analysis, we use the R package lowess to apply a locally weighted scatterplot smoother to linear local polynomial regressions of the latent factors (Cleveland, 1979). Therefore, the ACE-model is applied on twins from each birth year separately. For these time series of estimates, local regressions are fitted considering for 25% percent of the closest neighbor values to estimate a focal value. As a default of the nearest neighborhood method, lowess uses two thirds of the closest observations. We chose a lower value to reduce bias and increase the visibility of changes in latent factors. The regression estimates are plotted in a smoothened scatterplot of a non-parametric regression of year born on the regression estimates to draw the most complete picture of historical changes in genetic and environmental influence on AFB (for details see APPENDIX 1).

RESULTS

DESCRIPTIVE FINDINGS

Table 1 summarizes the variables for the overall sample as well as the cohorts, pooled and separately for MZ and DZ. MZ pairs are represented with a minimum of 35.4% for cohorts from 1953-1960 to a maximum of 45.8% in oldest cohorts (1919-1944). The mean AFB of the overall sample is 25.6 years with a standard deviation of 4.7. It is the highest (27.2) in the most recent cohorts (1961-1968), mirroring the postponement trend during the twentieth-century and is the lowest (24.9) in the baby boom cohorts after the Second World War (1945-1952). The mean AFB in our sample therefore follows the population trend in AFB of the UK during the past century on a slightly higher level (see Appendix 2).
The percentage of individuals who have not experienced AFB is 16.8 %, with 27.1 % of twin pairs containing censored information. These percentages increase during the century. They are lowest in the oldest birth cohorts (1919-1944) with 13.5 % individuals and 23.1 pairs and highest in the most recent cohorts (1961-1968), with 25.9 % of the individuals and 38.7 % of the pairs, where a higher share of women is still at risk to give for their first time.

Recall one of our primary research questions asks to what extent individual differences in the age at first birth can be explained by genetic factors. Table 1 reports a correlation in AFB of .24 for DZ twin pairs – only including uncensored pairs. DZ twins have the same degree of genetic relatedness as parents and children, and thus this value is in line with the review on family correlations in fertility (Murphy, 1999). The correlation in AFB of MZ twin pairs exceeds the correlation of DZ twin pairs in each sample of cohorts. This indicates a genetic component in AFB.

TWIN MODELS

Table 2 presents the results of the ACE model for the two model specifications: the tobit model (T), which adjusts for censoring, and the classic SEM (C) model that has been used until now. Both models report significant genetic effects on the AFB of females in the UK born from 1919-1968. These effects furthermore exceed the effects of the shared environment and therefore are the main cause of within family correlations in AFB.

The tobit model estimates heritability in AFB of .26 for the overall sample. Therefore, 26 % of the observed variance in AFB is explained by genetic variance. Variance in the shared environment of the twins – or between family variance – explains 14 %. The largest source of variance is attributed to the non-shared environment of the individuals, with 60 % of the observed variance in AFB.
Table 2 also reveals changes in the pattern of genetic and environmental influences across the defined cohorts. For cohorts born before the end of the Second World War (1919-1944) the tobit model estimates a moderate heritability of .36, and no significant effect of the shared environment can be reported. In the postwar generation (1945-1952), heritability decreases to .25 while the effect of the shared environment increases to .13 and becomes significant. This trend continues with cohorts born from 1953-1960 having insignificant genetic effects, while the shared environment peaksto 29% of the explained variance.

[TABLE 2 HERE]

In the most recent cohorts (1961-1968), we observed a new pattern with a moderate level of heritability (.32), and compared to the prior cohort, an almost constant level of the shared-environmental influence (.26). The non-shared environment explains the highest share of the variation in all models. However, it is the lowest in the most recent cohorts, where both the shared environment and the genetic influence have a moderate level of influence.

Table 1 showed differences between the standard deviations in AFB across cohorts. Since the estimates in Table 2 are ratios of the variance components over the total variance, results for the estimates can also vary depending on the overall variances. Therefore, Figure 2 depicts a stacked bar chart of the non-standardized variance components, underscoring the basic trends. For example, for the cohort 1953-1960, the overall variance is the largest, which might be a reason for the low level of heritability in this cohort (Table 2) – even if the genetically-related variance is constant. However, we can deduct from Figure 2 that the genetically-related variance on AFB also decreases strongly in absolute terms.

[FIGURE 2 HERE]

Figure 3 shows the scatterplot of the smoothened non-parametric regression curves for the fitted values of the local linear regressions. This explorative approach to the data reveals a strong peak in the genetic influence for cohorts born at the end of the 1930s. At the same time,
the influence of the non-shared environment of the twins experiences a strong drop. It furthermore depicts the continued growth of genetic influence for cohorts born in the mid-1950s and later. Whereas the influence of the shared environment is relatively stable, the non-shared environment appears to steadily decrease, while it consistently remains as the most important cause of variation.

[FIGURE 3 HERE]

Table 2 also illustrates the strong differences between the estimates of the two model specifications. In the overall sample, the results for the standard model overestimate heritability by 9 percentage points. Comparing the results across the cohorts, the difference increases from 4 percentage points in the oldest cohorts to 29 percentage points in cohorts born from 1953-1960. In the most recent cohorts the bias is also strong (27 percentage points), however, it goes in the opposite direction. As shown in Table 1, the number of censored individuals and twin pairs is the highest in the cohorts with the largest bias in the classic model.

Recall that the main difference between the two models is in their handling of censored information. In contrast to the tobit model, the classic model does not include twin pairs with censored information in the estimation of the covariances. Therefore, a potential explanation for the differences in the estimates lies in the constitution of censored cases. In general, there are two types of censored twin pairs, those in which both twins did not experience a first childbirth and those in which only one twin remains childless. Table 3 shows, for example, that in the youngest cohort the proportion of censored MZ twins who both did not experience first childbirth is about 15 percent points higher than in cohorts born from 1953-1960, while this value remains about the same for DZ. Therefore, the exclusion of censored information excludes more similar cases in MZ compared to DZ, which might explain the underestimation of heritability in the classic model for this cohort.

[TABLE 3 HERE]
In order to examine the effect of the exclusion of censored twin pairs, we furthermore estimated all models in a classic SEM imputing the \( C_i \) (see Appendix 3). Results are in line with the tobit model, with a slightly poorer inference, suggesting that the imputation of the last age of observation is quite a robust solution.

**SUMMARY AND DISCUSSION**

This article examined the relatively understudied topic of the influence of genes and the latent family environment on the AFB in UK twin cohorts born from 1919-1968. The findings indicate that a substantial part (40 %) of the variation in AFB is caused by latent family influences whereas genetic factors (26 %) play a more important role than the shared environment of siblings (14 %). The non-shared environment of siblings among siblings – which the characteristics of the partner probably play a crucial role (Kohler et al., 2002) – has the strongest influence (60 %). Our findings have substantial implications for demographic, sociological and biological research.

There are two main ways in which genetic dispositions may influence human fertility. First, there can be a direct effect on physiological characteristics such as fecundity. The age when a female enters menarche, for example, is strongly influenced by genetic variants (Anderson, Duffy, Martin, & Visscher, 2007). At the same time, this is the age when a woman is able to become pregnant. Therefore, depending on the extent to which physiological characteristics directly influence the AFB, social scientists actually underestimate the explanatory contributions of their predictors in reference to socially-related variance (Kohler & Rodgers, 2003).

Second, biological predispositions may affect the processes of decision-making and life-course planning both consciously and subconsciously (Kohler et al., 2006). In fact, biologists suspect that nowadays the traits most closely linked to fertility and fitness are
behavioral and psychological traits (Kirk et al., 2001). Empirical evidence such as the genetic influence on the first attempt to get pregnant (Kohler & Rodgers, 1999a; Rodgers et al., 2001) underscores the genetic component of the behavioral dimension.

Some biological and genetically-informed studies, therefore, challenge classic reasoning about the causal relationship between well-established associations of AFB and behavioral or socio-economic predictors. Rodgers et al. (2008) and Neiss et al. (2002), for example, show that the link between education and AFB becomes spurious within a behavioral genetics framework whereas the family background represents the endogenous source for the correlation of these traits. Nisén et al. (2013) recently demonstrated in a sample of Finish twins that genes can also represent a common factor for years in education and AFB.

In how far the family background influences subsequent life-course decisions and which individual characteristics mediate genetic effects on AFB remains an important topic for future investigation. Nevertheless, this study showed a moderate influence of genes contributing to the realized AFB. In the most recent cohorts (1961-1968), taking genes and the environment together, more than half of the overall variance in AFB is caused by endogenous family characteristics.

Biologists, furthermore, have highlighted the well-established association of AFB and the completed fertility of an individual in reference to their genetics. Kirk et al. (2001) found a robust genetic covariation between AFB and fitness for the Australian twins. Consequently, genes which lead to an early AFB also lead to a higher number of children and increase the chance of a woman to transmit her genes to the next generation. Such results suggest that modern societies are still evolving and subsequent generations carry a higher proportion of genetic dispositions for an early first birth. As Milot and colleagues (2011) conclude in a study of a population of natural fertility, the age at first birth decreased within the past 300 years as a response to natural selection. Multivariate genetic models have to be applied to determine the degree of genetic covariance in AFB and fitness. However, the moderate level of
heritability in our tobit model suggeststhat individuals with dispositions for an early onset of childbearing ensure the transmission of their genes to the next generation, while postponement of childbearing can lead to natural selection.

Whether genetic dispositions manifest themselves depends uponenvironmental conditions. Previous research highlights large changes in genetic and environmental influences on fertility, particularly an increase in the genetic influence as response to freedom in fertility choice after the Second Demographic Transition. Therefore, in a second step we examined trends in the latent factor estimates for birth cohorts born from 1919-1968 dividing them into subsamples aligned to previous research from Denmark (Kohler et al., 2002). However, the observed trends depart from previous findings, which reported an overall level of heritability in the number of children of around .20 with a strong increase for cohorts born in the 1960s who experienced the Second Demographic Transition in adulthood. In the UK, we find higher levels of genetic influence in particular for cohorts born from 1919-1944, and no significant heritability for cohorts born from 1953-1960.

Fertility behavior is often characterized as a response to uncertainty in the life course (Oppenheimer, Kalmijn & Lim, 1997; Mills & Blossfeld, 2005). Theoretical reasoning, therefore, suggests that environmental constraints such as economic uncertainty override and individual freedom triggers the manifestation of genetic predispositions for fertility. In particular, economic crises or exogenous shocks such as war represent environmental conditions that potentially override individual differences in genetically driven fertility motivation. Individual freedom in family planning, sexuality and birth control, in contrast, are supposed to trigger the genetic influence on individual fertility behavior in modern societies (Udry, 1996).

The UK has been a setting of distinct social upheavals in Europe during the past century with strong impact on fertility behavior (Hobcraft, 1996; Murphy, 1993). What we observe in the UK is a moderate level of heritability in AFB for cohorts born before the end of
the Second World War. The examination of the distinct birth cohorts, however, reveal strong environmental influence on the AFB for cohorts born in the 1920s and a peak in heritability for cohorts born in the mid and the end of the 1930s.

British women born in the environment of the 1920s grew up during the Second World War, in a context of major environmental constraints that impacted the timing of fertility onset. Food rationing began in January 1940 and the ‘Battle of Britain’ began in September the same year. In March 1941, 100,000 women were called to ‘come into the factories’, whereas married women who already had young children were exempt. The wartime rise in fertility from 1941 to 1944 was the strongest for women who were forced to postpone their (first) childbirth (Hobcraft, 1996), which explains the low level of heritability for these cohorts.

Individuals born in the 1930s, in contrast, benefited from changes after the war such as free National Health Service including maternity care, family allowances and free secondary education to “feed the aspirations of the middle classes”. The 1950s were furthermore a period of changing mores culminating in an area perceived as sexual revolution in the 1960s (Hobcraft, 1996). Sexual and familial self-control had become central paradigms in the lives of these individuals, leading to the manifestation of individual differences in AFB based on genetic predispositions.

A second strong shift in the influence of the latent factors can be seen in birth cohorts of the mid and the end of the 1950s. In contrast to previous fertility research, our results show a strong decline and even become insignificant in genetic influences for these births cohorts who grew up during the 1960th and started childbearing in the 1970s.

In the historical period when these cohorts started their childbearing, the UK had been the setting of severe economic disruption. At the end of 1972, the wage and rent freezes dominated the UK, and the rise in oil prices began 1973. House prices rose rapidly in 1974 and unemployment passed the one million thresholds in 1976 – rising to three million in the 1982
Additionally, the proportions of married women who had ever used the pill during the first five years of marriage increased from less than one-third in 1967 to around 80 per cent in 1976 (Murphy, 1993). Therefore, people were able to control childbearing and effectively respond to this period of economic insecurity in postponing childbirth. In fact, England and Wales reached the historical low point in the total fertility rate in 1977 (Hobcraft, 1996). The strong environmental impact on these cohorts is mirrored in our estimates, in which genetic differences had no significant influence at that time.

In cohorts born in the 1960s, we observed a new pattern with a growth in genetic influence and a still moderate level of family influence. This trend mirrors the previously investigated development in genetically-informed fertility research. These cohorts experience freedom in their life-course planning, in particular concerning relaxed family and career planning (Van de Kaa, 1987). The stable shared environmental influence after the Second Demographic Transition has been interpreted as a consistent influence of the family in particular on career planning and education, competing with fertility plans of women (Rodgers et al., 2008)

Overall, our results underscore the importance of the study of gene-environment interaction and fertility behavior. In particular, war and economic crises curbed the expression of genetic differences on the AFB in the UK, whereas changing mores triggered a peak of genetic influence. Since the introduction of the contraceptive pill, we also observed more rapid changes in the influences of the latent factors, since efficient birth control enables individuals to more directly respond to environmental changes and insecurity. Finally, in the youngest cohorts, latent family characteristics have a stronger influence than the individual environment, emphasizing the growing importance of genetic and family research in demography.

Previous research studying the genetics of AFB has produced mixed results. While Kirk et al. (2001) reported findings broadly in line with our estimates from the tobit model, research
from the US (Neiss et al., 2002:Rodgers et al., 2008) and Denmark found no significant genetic effect. We argue that this is attributed to the fact that these studies also engaged in different analytical strategies. Kirket al. (2001) applied an ordered probit model including censored individuals with the age at last observation and categorizing age groups to follow a normally distributed latent variable. Neiss et al. (2002) excluded childless observations (21%) and Rodgers et al. (2008) only included twin pairs, where both siblings had experienced AFB and applied a classic linear SEM.

To demonstrate how different analytical strategies impact the outcome, we applied two kinds of models: a recently developed tobit model and the classic SEM excluding censored cases for the covariance estimation. We found large difference between the two models, in particular for cohorts born since 1953 where the proportion of censored individuals and pairs is highest. We conclude that the choice of analytical strategies is critical. In particular, the exclusion of censored information appears to be crucial because, since we also applied classic models imputing censored cases with effectively the same results as in the tobit model. Some have suggested that more dissimilar twin pairs have higher chances to be excluded in the classic design due to the nature of censoring (Kohler et al. 1999b). However, for the biosocial model, the composition of similar and dissimilar pairs across zygosity is crucial and as shown in this study, a prediction of the direction of the bias cannot be made intuitively.

Although there are numerous strengths, the twin design applied in this study still has limitations. The strongest implicit assumption is that MZ and DZ share their family environment to the same extent – the equal environment assumption (EEA). From a sociological perspective this is a “heroic assumption” (Conley & Rauscher, 2011). Replication is required in designs which avoid or relax the EEA such as adoption studies or the recently developed tools of identity-by-descent estimates (Visscher et al., 2006).

Twin studies provide evidence for genetic and environmental influences on a trait. The actual genetic variants which influence the AFB, however, and the molecular and social
pathways remain to be investigated. In particular, in an environment such as the twentieth-century UK there were large changes in the determinants of fertility (Hobcraft, 1996). Therefore, it is essential to recognize that the mediating factors might also have changed across cohorts.

As Rodgers et al. (2001) stated, the answer to the question of whether genes affect fertility is no longer “no”, but rather “sometimes they do, sometimes they don’t”. Our study supports this statement, but also provides new insights in the mechanisms behind the changing influences and gene-environment interaction. While genetic influence is low in times of war and economic crises, genetic effects experience a peak in times of the sexual revolution and play an important role in the contemporary UK.

ACKNOWLEDGEMENT

This research is funded by a grant from the Dutch Science Foundation (NWO) (VIDI 452-10-012, granted to M. Mills). The Authors gratefully acknowledge Tomas Sobotka for information and advice on age at first birth data for the UK and the Department of Twin Research from the King’s College, University of London for providing the TwinsUK registry as well as their major research sponsors: Welcome Trust, the European Commission, the National Institute for Health Research and the Chronic Disease Research Foundation

REFERENCES


### Table 1. Summary statistics for female UK twins for the overall sample and the subsequent cohorts

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<tbody>
<tr>
<td>n of twin pairs</td>
<td>2,274</td>
<td>872</td>
<td>663</td>
<td>455</td>
<td>284</td>
</tr>
<tr>
<td>% MZ (n twin pairs)</td>
<td>41.6 (945)</td>
<td>45.8 (399)</td>
<td>39.4 (261)</td>
<td>35.4 (160)</td>
<td>43.7 (124)</td>
</tr>
<tr>
<td>% censored individuals</td>
<td>16.8</td>
<td>13.5</td>
<td>15.5</td>
<td>19.3</td>
<td>25.9</td>
</tr>
<tr>
<td>MZ</td>
<td>18.2</td>
<td>11.9</td>
<td>19.9</td>
<td>20.8</td>
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<td>DZ</td>
<td>15.8</td>
<td>14.9</td>
<td>12.6</td>
<td>18.5</td>
<td>21.6</td>
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<td>% twin pairs with censored information</td>
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<td>23.1</td>
<td>25.3</td>
<td>30.3</td>
<td>38.7</td>
</tr>
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<td>MZ</td>
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<td>20.1</td>
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<td>43.5</td>
</tr>
<tr>
<td>DZ</td>
<td>26.4</td>
<td>25.6</td>
<td>21.9</td>
<td>29.3</td>
<td>35.6</td>
</tr>
<tr>
<td>Mean (SD) age at censoring</td>
<td>57.0 (10.5)</td>
<td>67.3 (5.5)</td>
<td>56.3 (3.4)</td>
<td>48.4 (3.5)</td>
<td>40.1 (3.2)</td>
</tr>
<tr>
<td>MZ</td>
<td>58.0 (10.6)</td>
<td>67.9 (5.3)</td>
<td>56.8 (3.1)</td>
<td>49.0 (3.0)</td>
<td>40.3 (2.8)</td>
</tr>
<tr>
<td>DZ</td>
<td>56.2 (10.3)</td>
<td>66.9 (5.6)</td>
<td>56.0 (3.5)</td>
<td>48.1 (3.8)</td>
<td>40.0 (3.5)</td>
</tr>
<tr>
<td>Mean (SD) AFB</td>
<td>55.4 (11.1)</td>
<td>68.2 (5.5)</td>
<td>56.6 (2.6)</td>
<td>49.1 (3.0)</td>
<td>40.1 (2.7)</td>
</tr>
<tr>
<td>Mean (SD) AFB</td>
<td>25.6 (4.7)</td>
<td>25.5 (4.4)</td>
<td>24.9 (4.6)</td>
<td>25.9 (5.1)</td>
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<td>MZ</td>
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<td>25.1 (4.4)</td>
<td>26.2 (4.9)</td>
<td>27.7 (5.1)</td>
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<tr>
<td>DZ</td>
<td>25.4 (4.7)</td>
<td>25.2 (4.4)</td>
<td>24.8 (4.7)</td>
<td>25.7 (5.2)</td>
<td>26.8 (4.7)</td>
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<tr>
<td>Correlation in AFB</td>
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<td>.30</td>
<td>.23</td>
<td>.42</td>
</tr>
<tr>
<td>MZ</td>
<td>.40</td>
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<td>.32</td>
<td>.45</td>
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<tr>
<td>DZ</td>
<td>.24</td>
<td>.20</td>
<td>.26</td>
<td>.18</td>
<td>.39</td>
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</table>
MZ = Monozygotic; DZ = Dizygotic; AFB = Age at First Birth; SD = Standard Deviation; Mean AFB and Correlation AFB are computed for individuals who experienced AFB; Data: TwinsUK
Table 2. Standardized parameter-estimates of a ACE models in a classic (C) SEM and a tobit model (T) for the overall sample and the cohorts.

<table>
<thead>
<tr>
<th>Cohort</th>
<th>Model</th>
<th>$h^2$</th>
<th>95% CI</th>
<th>$c^2$</th>
<th>95% CI</th>
<th>$e^2$</th>
<th>95% CI</th>
<th>n</th>
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<td>(1 T)</td>
<td>.26</td>
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<td>.14</td>
<td>[.07-.25]</td>
<td>.60</td>
<td>[.55-.64]</td>
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<td>1919-1968</td>
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<td>.35</td>
<td>[.21-.52]</td>
<td>.07</td>
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<td>.58</td>
<td>[.52-.64]</td>
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<td>1919-1944</td>
<td>(2 S)</td>
<td>.36</td>
<td>[.19-.55]</td>
<td>.04</td>
<td>[.00-.50]</td>
<td>.60</td>
<td>[.54-.68]</td>
<td>872</td>
</tr>
<tr>
<td>1919-1944</td>
<td>(2 T)</td>
<td>.40</td>
<td>[.18-.66]</td>
<td>.03</td>
<td>[.00-.93]</td>
<td>.57</td>
<td>[.49-.68]</td>
<td></td>
</tr>
<tr>
<td>1945-1952</td>
<td>(3 T)</td>
<td>.25</td>
<td>[.08-.50]</td>
<td>.13</td>
<td>[.02-.39]</td>
<td>.62</td>
<td>[.55-.70]</td>
<td>663</td>
</tr>
<tr>
<td>1945-1952</td>
<td>(3 C)</td>
<td>.34</td>
<td>[.11-.66]</td>
<td>.07</td>
<td>[.00-.59]</td>
<td>.59</td>
<td>[.47-.70]</td>
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<tr>
<td>1953-1960</td>
<td>(4 T)</td>
<td>.06</td>
<td>[.00-.83]</td>
<td>.29</td>
<td>[.13-.52]</td>
<td>.65</td>
<td>[.54-.76]</td>
<td>455</td>
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<td>1953-1960</td>
<td>(4 C)</td>
<td>.34</td>
<td>[.07-.75]</td>
<td>.01</td>
<td>[.00-.100]</td>
<td>.65</td>
<td>[.49-.79]</td>
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<tr>
<td>1961-1968</td>
<td>(5 T)</td>
<td>.32</td>
<td>[.10-.64]</td>
<td>.26</td>
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<td>.42</td>
<td>[.32-.53]</td>
<td>284</td>
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<tr>
<td>1961-1968</td>
<td>(5 C)</td>
<td>.05</td>
<td>[.00-.75]</td>
<td>.39</td>
<td>[.12-.73]</td>
<td>.56</td>
<td>[.38-.73]</td>
<td></td>
</tr>
</tbody>
</table>

T = Tobit Model; C = Classic Model; $h^2$ = Heritability; $c^2$ = Shared Environmental Effects; $e^2$= Unique Environmental Effect; CVa = Coefficient of Genetic Variation; 95% CI = 95 % Confidence Interval; n = Number of Twin Pairs; all models control for the year the twins are born; Data: TwinsUK
Table 3. Percentage of twin pairs with single and pairwise censored information by zygosity; for cohorts born between 1953-1960 and 1961-1968

<table>
<thead>
<tr>
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<tbody>
<tr>
<td>% Both twins censored</td>
<td></td>
<td></td>
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<tr>
<td>MZ</td>
<td>28.9</td>
<td>44.4</td>
</tr>
<tr>
<td>DZ</td>
<td>26.7</td>
<td>23.2</td>
</tr>
<tr>
<td>% One twin censored</td>
<td></td>
<td></td>
</tr>
<tr>
<td>MZ</td>
<td>71.1</td>
<td>56.6</td>
</tr>
<tr>
<td>DZ</td>
<td>73.3</td>
<td>76.8</td>
</tr>
</tbody>
</table>

% of couples with censored information; MZ = Monozygotic; DZ = Dizygotic; Data: TwinsUK
Figure 1. ACE path model for the classic twin study (Monozygotic/Dizygotic Twins)

$A_1/A_2$ = Genetic Endowment; $C$ = Shared Environment; $E_1/E_2$ = Unique Environment; $a$ = Genetic Effects; $c$ = Shared-Environmental Effects; $e$ = Non-Shared Environmental Effects/Measurement Error; $Y_1/Y_2$ = Outcome of Twin 1 and Twin 2
Figure 2. Stacked Bar Chart of the unstandardized variance components of the ACE tobit models for the cohorts

Data: TwinsUK
Figure 3. Smoothened scatterplot of local polynomial regressions of the birth year on the latent factors estimates for birth cohorts 1923-1968

Trends of Latent Factors Across Cohorts

Cohorts Born Between 1923-1968: For birth cohorts; Data: TwinsUK
APPENDIX

APPENDIX 1. Local polynomial regression and non-parametric regression curve

In order to fully explore the underlying trends of the latent factors, we applied a local polynomial regression to the factor estimates of each birth cohort using the R package lowess. Therefore, we first estimated the ACE-model of twin pairs of each birth year separately producing time series of 46 estimates (birth cohorts born between 1923-1968) for the genetic influence, the shared environment and the non-shared environment. For cohorts born between 1919-1922, the small number of cases did not allow an estimation.

In a second step, we partitioned the data in local areas around each birth year and fit separately bivariate linear regressions of birth year on the latent factors within each section. In practice, the choice of the local window of neighborhood values, which are included in the local regression is made by the researcher. It is a trade-off between bias of the estimation and remaining variance between the local estimates. As default value of the nearest neighborhood method, lowess uses two third of the closest observations in the time series, in our cases two third of the next birth cohorts for each local estimation. We chose a lower value to reduce bias and increase the visibility of changes in latent factors. We provided the results for 25 % of the nearest neighbors, to show a good trade-off of reduced bias and acceptable variance in the factor trends. Note that the basic trends we discussed are not very sensitive and therefore the choice of this parameter was not critical for our substantial points.

The package loewess uses the tricube weight function to give higher weight to values which are closer to the focal value.

\[ z_i = (x_i - x_0)/h(A1) \]

\[ W_t = \begin{cases} (1 - |z|^3)^3 & \text{for } |z| < 1 \\ 0 & \text{for } |z| \geq 1 \end{cases} \] (A2)
whereas $z_i$ is the scaled distance between the predictor value and to the $i$th observation and $h$ is half the width of the observation window. Observations, which have a higher distance to the focal $x$ than $h$, receive the weight 0. The local estimation is carried out for the three factor estimations of each single birth cohort. Subsequently a local polynomial nonparametric regression curve over the plotted estimates is produced.

APPENDIX 2. Scatterplot of the estimated AFB for birth cohorts from the UK and the TwinsUK

**Age at First Birth in UK and TwinsUK**

AFB UK: The estimated average AFB in the UK. Because official data on birth order have been historically only collected within marriage, these values are based on estimates from the Office for National Statistics. Source: Office for National Statistics, Cohort fertility, Table 2.

AFB TwinsUK: The estimates are based on the average AFB per birth cohorts and smoothened in a linear local

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4Source: Office for National Statistics, Cohort fertility, Table 2.
Downloaded: 03.2013
regression (see APPENDIX 2) considering 20% of the closest neighborhood values.

Data: TwinsUK

APPENDIX 3. Standardized parameter-estimates of a ACE models in a classic SEM with imputed last year of observation in case of censoring (Cim) and a tobit model (T) for the overall sample and the cohorts.

<table>
<thead>
<tr>
<th>Cohort</th>
<th>Model</th>
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<th>95% CI</th>
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<td>2,274</td>
</tr>
<tr>
<td></td>
<td>(1 Cim)</td>
<td>.25</td>
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<td>.14</td>
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<td>.61</td>
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<tr>
<td>1919-1944</td>
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<td>1945-1952</td>
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<td>[.53-.73]</td>
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<td>1953-1960</td>
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<td>1961-1968</td>
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<td>.44</td>
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</tbody>
</table>

T = Tobit Model; Cim = Classic Model with imputed last year of observation in case of censoring; $h^2 =$ Heritability; $c^2 =$ Shared Environmental Effects; $e^2 =$ Unique Environmental Effect; 95% CI = 95% Confidence Interval; n = Number of Twin Pairs; all models control for the year the twins are born; Data: TwinsUK