Between Nurture and Nature: The Shifting Determinants of Female Fertility in Danish Twin Cohorts

Hans-Peter Kohler[1] Joseph Lee Rodgers[2] Kaare Christensen[3]

February 6, 2003

[1]Associate Professor of Sociology, Population Studies Center, University of Pennsylvania, 3718 Locust Walk, Philadelphia, PA 19104-6299, USA, *email:* hpkohler@pop.upenn.edu, *www:* http://www.ssc.upenn.edu/∼hpkohler.

[2]Professor of Psychology, Department of Psychology, University of Oklahoma, Norman, Ok 73019. *Email:* jrodgers@ou.edu

[3]Research Professor, Institute of Public Health and Danish Center for Demographic Research, University of Southern Denmark-Odense, Sdr. Boulevard 23A, 5000 Odense C, Denmark, *Tel:* +45-6550 3049, *email:* kchristensen@health.sdu.dk

Abstract

Behaviors related to fertility constitute primary candidates for investigating the relevance of evolutionary influences and biological dispositions on contemporary human behaviors. Using female Danish twin cohorts born 1870–1968, we document important transformations in the relative contributions of 'nurture' and 'nature' to within-cohort variations in early and complete fertility, and we point towards a systematic relation between the socioeconomic context of cohorts and the relevance of genetic and shared environmental factors. This transformation is most striking for early fertility where genetic factors strengthen over time and are consistent with up to 50 per cent of the variation in early fertility in most recent cohorts. Understanding this emerging relevance of genetic factors is of central importance because early fertility constitutes an important determinant of complete fertility levels in low fertility societies, and because teenage motherhood and early childbearing are often associated with negative life-cycle consequences. Moreover, our results emphasize the need for socially and contextually informed analyses of nature *and* nurture that allow both factors to influence human reproductive behavior over time.

1 Introduction

Differential reproductive success constitutes one of the primary mechanisms through which evolutionary processes lead to natural selection within and between species. Many behaviors related to fertility are therefore strongly shaped by evolutionary influences (e.g., Dawkins, 1976), and these influences have led to important male-female differences in sexuality, fertility and related behaviors. These dissimilarities in male and female reproductive strategies begin with the asymmetrical efforts of males and females in producing egg and sperm cells, and they are present in different mating/marriage strategies, differences in attachment to children, and differential incentives to invest in offspring (e.g., Geary, 1998; Mealey, 2000; Miller and Rodgers, 2001; Trivers, 1972).

Evolutionary processes exerted their primary influence on reproductive and other behaviors while our ancestors lived in hunter and gatherer societies. Nevertheless, there is increasing evidence that even complex human behaviors continue to be affected by the evolution of our physiological characteristics and cognitive abilities (e.g., Carter, 1998; Kaplan, Hill, Lancaster, and Hurtado, 2000; LeDoux, 1996). An awareness about the relevance of such influences can substantially enhance the understanding and theoretical conceptualization of many human behaviors (Massey, 2002; Robson, 2001; Udry, 1988, 1995). In addition, any perception that acknowledging evolutionary/genetic influences on behavior reduces the scope for social theories to unfold is clearly unjustified: many evolutionary and genetic influences on behavior interact with the cultural, social and economic environment, and specific social or economic conditions may substantially alter the realization of evolved behavioral dispositions in contemporary behavior.

Behaviors related to fertility constitute primary candidates for investigating the relevance of evolutionary influences and biological dispositions on contemporary human behaviors for a number of reasons. First, fertility behaviors are an essential determinant of population growth and survival, and they directly contribute to the fitness criterion that is maximized by evolutionary processes. Second, the evolutionary theory of life-history traits is well developed (e.g., Low, 1998; Roff, 1992; Stearns, 1992) and yields testable implications regarding genetic influences on fertility outcomes (e.g., Hill and Kaplan, 1999; Low, Clarke, and Lockridge, 1992; Voland, 1998). A central example in this context is Fisher's Fundamental Theorem of Natural Selection (FTNS, Fisher, 1930) that has frequently been interpreted to mean that fitness traits, such as fertility and fertility precursors, have heritabilities near zero. Third, the cultural, social and economic context of human reproduction has been subject to important changes during the demographic transition, the diffusion of reliable contraception, and the emergence of below-replacement fertility. These well-documented variations in the context of fertility decisions allow for the possibility that genetic variation in fertility outcomes re-emerges in modern societies despite the tendency to eliminate such variation during the evolutionary process (Kohler and Rodgers, 2003; Rodgers, Hughes, Kohler, Christensen, Doughty, Rowe, and Miller, 2001a).

In order to address these questions, we analyze a unique dataset composed of Danish twin cohorts born over the course of almost one century from 1870 to 1968. The first part of the study focuses on early fertility behavior—that is, on fertility behavior up to ages 25—and considers the genetic and shared environmental factors that facilitate an early transition into parenthood. This part of our study is based on female twin pairs born between 1945 and 1968. We show that the determinants of early fertility in these female twin cohorts have been subject to a fundamental shift from 'nurture' towards 'nature': genetic influences are almost absent for the older and strongly relevant for the younger cohorts, as shared environmental

influences change in the opposite direction. In the second part of our study we embed this recent pattern in a study of completed fertility for twin cohorts born during 1870–1960. We show that the changes in the relative importance of genetic versus environmental influences, which we observe for early fertility, is not a unique phenomenon. To the contrary, there seem to be shifts in the relative importance of 'nature' and 'nurture' that are systematically related to the socioeconomic status of the cohorts. Fertility behavior is therefore embedded between 'nature' and 'nurture', and the relative contribution of these factors seems to vary with the specific context of a cohort's socioeconomic and demographic context.

The remainder of this paper is structured as follows. We begin in Section 2 with a discussion of the social and demographic context of fertility behavior in Denmark during the last century, and we provide a brief introduction into the twin-design that allows a decomposition of variation in behaviors into genetic, shared environmental and non-shared environmental components. We also elaborate on Fisher's Theorem of Natural Selection that constitutes an important theoretical framework for behavior genetic analyses of fertility and related life-history traits. In Section 3 we begin our empirical analyses with a focus on early fertility using twin cohorts born during 1945–68. In Section 4 we then shift the attention of our analyses to complete fertility. Most importantly, this allows us to investigate the fertility behavior of twins born during a period of almost one century from 1870 to 1960. In Section 5 we conclude our paper with a theoretical interpretation of the shifting contributions of "nurture" and "nature" to the variation in female fertility during the last century. An appendix provides a more detailed presentation of our methods.

2 Variations in Fertility within Populations: Biological versus Socioeconomic Determinants

2.1 The changing context of fertility decisions during the last century

The last century has witnessed fundamental demographic changes that have substantially transformed the demographic landscape in Europe as well as all other countries. The late 19th and the early 20th century were characterized by the Demographic Transition resulting in a decline in marital fertility across all countries (Coale and Watkins, 1986). The post-war period then led to an increase in period fertility, and to a much lesser extent in cohort fertility, which was unexpected to many contemporary observers (Bean, 1983; Kohler, 2000; Morgan, 1996; Pampel, 1993). Starting in the 1970s, period fertility declined to below-replacement levels, and this trend has been accompanied by an increase in the mean age at first birth, an increasing proportion of out-of-wedlock births, and the rise of cohabitation as the dominating form of partnership for young adults (Coleman, 1996; Lesthaeghe, 1983; van de Kaa, 1987).

The most substantial part of the Danish fertility transition took place in the 20th century and primarily affected cohorts born after 1880. The respective period fertility patterns during the last century, along with the complete fertility of cohorts born after 1880, are depicted in Figure 1. Cohort fertility reached a trough for individuals born around the turn of the century, and the decline in period fertility reversed in 1933. After WWII, Denmark experienced a marked increase in the total fertility during a post-war baby boom that was also associated with a slight rise in cohort fertility. In the late 1960s, period fertility dropped again substantially as part of a transformation of demographic behavior that was later labeled as the Second Demographic Transition (Lesthaeghe and van de Kaa, 1986;

van de Kaa, 1987). While the decline in the total fertility rate after 1965 is also reflected in a reduction of complete fertility for cohorts born after 1930, this reduction has been relatively modest. In contrast to many other European countries, Denmark also experienced a recovery of period fertility in the 1980s, and it currently belongs to the European countries with relatively high fertility. Moreover, cohort fertility is predicted to remain relatively constant at about 1.9 children for cohorts born during the 1960s (Council of Europe, 2000; see also Knudsen 1993). Denmark's relatively high level of fertility is shared by France, the Netherlands, the United States, and Nordic countries, but it differs from the situation in Southern and Eastern European countries that have been characterized as having lowest-low fertility in the 1990s (Kohler, Billari, and Ortega, 2002a). The Danish fertility trends thus reflect a typical demographic pattern in Western Europe and the U.S. for the 20th century, one that is characterized by a sequence consisting of decline of marital fertility during the demographic transition, a post-war baby boom that mainly led to increases in period fertility, and a subsequent baby bust leading to below-replacement fertility.

[Figure 1 about here.]

Explaining these substantial changes in the level of fertility and the patterns of fertility-related behaviors during the last century has been a hallmark of demographic and related research in recent decades. The proposed explanations have been widely discussed in numerous studies and include economic growth, changes in female labor force participation, family policies, etc. (e.g., see Bongaarts and Bulatao, 2000; Brewster and Rindfuss, 2000; Coale and Watkins, 1986; Hirschman, 1994). Moreover, in recent years there has also been an increasing emphasis on ideational aspects, cultural changes and process of social interactions as important factors contributing to fertility change both during the demographic transition as well as the baby boom and bust (Kohler, 2000; Kohler, Billari, and Ortega, 2002a; Lesthaeghe, 1983; Watkins, 1990).

It is important to emphasize that the above explanations of fertility change rarely collide with (or even mention) biological theories. Quite the contrary: recent evolutionary approaches to demographic change frequently incorporate transformations in the context of fertility decisions through socioeconomic change or technical innovations. In particular, socioeconomic changes and technological innovations lead to adjustments in the optimal fertility strategies because they imply altered incentives for allocating scarce resources, such as time and energy, to reproductive efforts, child-quality versus quantity, and somatic investments and several other competing uses (e.g., see Hill and Kaplan, 1999; Kaplan, Hill, Lancaster, and Hurtado, 2000). As a consequence, evolutionary and socioeconomic theories are in fact quite compatible in terms of explaining trends in the level of fertility (e.g., see Kaplan, Lancaster, Tucker, and Anderson, 2002). The challenge for incorporating biological dispositions with sociological theories, however, remains in the explanations of within-population variation in behavior. In particular, differential biological dispositions of individuals—resulting for instance from genetic variation or hormonal influences—can be important determinants of individual behavioral differences in addition to socioeconomic incentives or structural influences. On the one hand, some of these biological dispositions exert their effect on behavior through conscious decisions making, as for instance in decisions about human capital investments that depend in part on individuals' or their parents' knowledge about their own ability (e.g., see Behrman, Rosenzweig, and Taubman, 1994, 1996). On the other hand, individuals in many cases also may not be aware of 'background' influences caused by biological dispositions, which occur for instance when these dispositions

affect emotions, partner preferences, desires for children or nurturing, sexual activity, or cognitive abilities (e.g., Foster, 2000; Gangestad and Thornhill, 1998; Gangestad, Thornhill, and Garver, 2002; Halpern, Udry, Campbell, and Suchindran, 1999; Halpern, Udry, and Suchindran, 1997; Miller and Rodgers, 2001; Penton-Voak, Perrett, Castles, Kobayashi, Burt, Murray, and Minamisawa, 1999; Rodgers and Kohler, 2003).

Instead of focusing on specific biological mechanisms, our investigation tries to estimate the *net* contribution of a broad range of genetically-mediated biological factors to the variation in fertility behavior *within* cohorts. The advantage of this approach is that it provides an estimate of the overall relevance of genetically-mediated biological effects on variation of fertility behavior. This information about the overall relevance of genetically-mediated variation is interesting in itself. Moreover, this information also will guide future research investigating specific mechanisms and pathways of biological influences: if the net-overall variation attributed to genetic factors is high, the search for specific pathways (or even specific gene factors) is likely to be more promising as compared to a situation in which the overall influences is found to be low.

2.2 Identifying Determinants of Variation: The Twin Design

Our analyses are based on a large number of Danish twins whose fertility behavior is analyzed with behavioral genetic models. This 'twin design' allows a separation of two different effects that can lead to a similarity of fertility behaviors—as well as other behaviors and traits—among relatives. *Shared-environment effects* arise because individuals growing up in the same household are subject to similar processes of socialization, socioeconomic conditions of parents, and a similar family environment. *Genetic effects* originate through influences of inherited traits and predispositions on behavior. The distinction between these two mechanisms has been subject to a long and often heated debate on 'nature versus nurture'. Twin studies emerge as an important tool in this debate: they allow a separation between shared-environment and genetic effects on the variation of a trait using assumptions about the interaction of genetic alleles, the relevance of assortative mating and the treatment of siblings by parents (see Plomin, Defries, and Mclearn 1997 for an introduction to behavioral genetics and twin methods). For instance, this approach has been employed to investigate the role of genetic factors in schooling and educational attainment (Behrman and Taubman, 1989), cognitive abilities (Cherny, Cardon, Fulker, and DeFries, 1992), the propensity to divorce (McGue and Lykken, 1992), and mortality (Herskind, McGue, Holm, Sorensen, Harvald, and Vaupel, 1996; Yashin and Iachine, 1997).

Twin studies rely primarily on the comparison of correlations for sets of monozygotic (MZ) and dizygotic (DZ) twins. In particular, twin studies assume that members of MZ and DZ twin pairs are similarly affected by the parental household and other shared family environments. These 'shared environments' are common to reared-together twins and are thus a source of their similarity. If shared-environment effects are the only influences on a trait or behavior, we expect more similarity in this trait within a twin pair than among unrelated individuals picked at random from the population. However, we do not expect a systematic difference in the similarity of MZ and DZ twins on the basis of shared-environment effects. If MZ twins exhibit a greater similarity in a behavior than DZ twins, then this additional similarity is suggestive of a genetic influence on this specific trait, because MZ twins share all their genes while DZ twins share 50 per cent on average (like ordinary siblings).[1] The standard *additive* genetic model, which is used to interpret these differential correlations within MZ and DZ twin pairs in terms of genetic and shared environmental contributions,

assumes that many genes contribute additively to a phenotype. The model then provides a decomposition of the within-population variance of a trait into additive genetic variance (heritability, h^2) and shared environmental variance (c^2). The former measures the proportion of total phenotypic variance attributable to (additive) genetic variance, and the latter reflects the proportion of the total variance related to differences in shared-environmental conditions, such as parental background, socialization, etc. All of the remaining variance is typically absorbed in the residual term of the model that includes a combination of variance attributable to both nonshared environmental influences (those that make family members different from one another; e^2) and measurement error.

An important advantage of our study is that the analyses are relatively robust with respect to many criticisms of twin studies (e.g., see Guo, 1999; Lewontin, Rose, and Kamin, 1985). First our data are based on one of the world-wide largest twin registers. The analyses for cohorts born after 1945, for instance, include the complete population of identified female twin pairs in Denmark. These data are therefore nationally representative, in contrast to other twin samples that are selected on the basis of service in the military (e.g., the NAS-NRC Twin and Offspring sample), residency in a specific state (e.g., parts of the Minnesota Twin Registry) or participation in a twin convention (e.g., the Twinsberg, Ohio, Twins Convention Survey). Second, the focus in our study on *trends* of heritabilities and shared environmental influences across cohorts. This focus on trends increases the robustness of inferences because comparing patterns of heritability across cohorts 'differences out' potential over-estimates in the coefficient of heritability, h^2, that occur when the assumptions underlying the twin design are violated (e.g., see Lewontin, Rose, and Kamin, 1985).

2.3 Fertility and Fisher's Fundamental Theorem of Natural Selection

Obviously, one or a few genes cannot link to fertility desires and behaviors in a strong and causal sense. This situation would preclude many human practices that are typical among humans, including voluntarily childlessness, modern contraceptive practice, and postponement of childbearing to pursue education, among others (Rodgers, Kohler, Kyvik, and Christensen, 2001b). Indeed, the fertility transition, during which fertility declines rapidly and almost universally, is inconsistent with strongly deterministic models. However, the opposite extreme is equally untenable. To ignore the biological basis of fertility, or, more explicitly, to ignore the obvious conceptual link between the evolutionary process and the behavior driving that process— that is, fertility-related behavior—virtually precludes effective theorizing.

Fisher's Fundamental Theorem of Natural Selection (FTNS) suggested that natural selection causes genetic variation to disappear. Until recently, therefore, the literature has reflected skepticism of finding genetic influences on human fertility. For instance, Fisher's (1930) heritability estimate of $h^2 = .40$ for complete fertility was criticized by Williams and Williams (1974) as being an artifact of secular trends. Moreover, other studies have found low or zero heritability in fertility. Imaizumi, Nei, and Furusho (1970) found nonsignificant heritabilities in Japanese data from 1881–1930 using father-child ($h^2 = -.02$) and mother-child ($h^2 = .12$) comparisons. Mealey and Segal (1993) used data from U.S. twins raised apart, and found a monozygotic twin (MZ) correlation of .06 (implying $h^2 = .06$; $n = 32$ pairs) and a dizygotic twin (DZ) correlation of $r = .10$ (implying $h^2 = .20$; $n = 23$ pairs); both nonsignificant. Although these studies seem to confirm the implications of the FTNS, parts of his theorem has also been over-interpreted. In particular, Fisher recognized that various biological processes can re-introduce genetic variance, bringing the system back into

equilibrium with genetic variance above zero. Such biological processes include frequency-dependent selection, heterozygote advantage, sexual antagonism, and, especially mutation (e.g., Hughes and Burleson, 2000). These biological processes have been called "perturbing forces." But in modern society, social processes can also be perturbing forces, acting to re-introduce genetic variance into the system that has been cleansed of such variance by natural selection. One example of a perturbing force discussed by Fisher himself is contraceptive innovation (Fisher, 1930). Others include changes in marriage patterns, changes in family size norms, availability of abortion, and efficacious treatment of infertility. Obviously, it is easy to identify potential environmental factors that may act as "perturbing forces" in the modern world. When these are combined with the effects of mutation, what emerges is substantial motivation to measure and explain patterns of genetic variation in human reproduction and fertility.

The above arguments also emphasize the necessity of considering genetic influences on fertility in the context of changing socioeconomic conditions. Because perturbing forces are prerequisite for re-emergence of genetically mediated variation in fertility outcomes, these arguments imply an interaction between social change and genetic influences on within-population variation in human traits and behaviors. Instead of testing "nature *versus* nurture", our analysis therefore pursues a socially contextualized analysis of the shifting contributions of both nature *and* nurture to variation in human fertility behavior over time. This consideration therefore accommodates the recent argument proposed by sociologists and other social scientists that the extensive social theorizing about human fertility that has occurred over the past several decades by psychologists, sociologists, demographers, economists, and public health experts is flawed unless those social models are also informed by biological/genetic components as well (e.g., Rossi, 1994; Udry, 1995; Wood, 1994).

3 Early Fertility (Cohorts 1945–1968)

3.1 Genetic influences on variation in early fertility

Understanding the social and biological factors leading to an early onset of fertility are of central social relevance. First, it is well-known that a later onset of fertility tends to delay the progression to the second or third child and that it tends to reduce complete fertility (Kohler, Skytthe, and Christensen, 2001; Morgan and Rindfuss, 1999). Changes in the determinants of early fertility are therefore an important aspect in understanding the emergence of low and lowest-low fertility levels in many European and other developed countries (e.g., see Kohler, Billari, and Ortega, 2002a). Second, early fertility has substantial implications for an individual's life-course. For females, for instance, early fertility and teenage childbearing is often associated with lower educational attainment, a lower participation in the labor market and more frequent divorce, although earlier studies may have overestimated these effects (Geronimus and Korenman, 1992; Hotz, McElroy, and Sanders, 1999).

So far, most research on early fertility has focused on socioeconomic influences. For instance, fertility rates in teenage and young adult ages are subject to substantial period fluctuations due to varying socioeconomic conditions experienced by the respective cohorts in early adult years (Easterlin, 1980; Morgan, 1996; Rindfuss, Morgan, and Swicegood, 1988). Moreover, own education, parental social status, rural versus urban residence, parental separation, knowledge and availability of contraceptive methods have been associated with early fertility (Michael and Tuma, 1985; Morgan and Rindfuss, 1999; Thornberry, Smith, and Howard, 1997; Wu and Martinson, 1993). In addition, early fertility has also been

linked to contextual influences of neighborhoods and schools (Brewster, Billy, and Grady, 1993; South and Crowder, 1999), and 'epidemic' theories have been suggested to explain patterns of teenage childbearing over time (Crane, 1991; Rodgers, Rowe, and Buster, 1998).

Little attention has been paid to genetically-mediated individual differences in early fertility, despite the fact that genetic variation has been documented in the onset of sexual intercourse, marriage and divorce patterns (Dunne, Martin, Statham, Sltske, Dinwiddie, Bucholz, Madden, and Heath, 1997; McGue and Lykken, 1992; Mealey and Segal, 1993; Rodgers, Rowe, and Buster, 1999; Rowe, 2000; Treloar and Martin, 1990) and more recently also complete fertility (Kohler, Rodgers, and Christensen, 1999; Rodgers, Hughes, Kohler, Christensen, Doughty, Rowe, and Miller, 2001a; Rodgers, Kohler, Kyvik, and Christensen, 2001b) and reproductive fitness (Kirk, Blomberg, Duffy, Heath, Owens, and Martin, 2000).

This absence of studies about genetic influences on early fertility is even more surprising because this behavior is particularly interesting for investigating genetic contributions to variation in human fertility behavior. In particular, in populations with natural fertility levels and without conscious fertility control, the age at marriage is closely related with the age at first birth. Both ages constitute important determinants of complete fertility, reproductive success and fitness (e.g., Borgerhoff Mulder, 1990; Käär and Jokela, 1998; Käär, Jokela, Helle, and Kojola, 1996; Low, 1990). In Northern and Western Europe it is also well-documented that variations in complete fertility in response to changing socioeconomic or climatic conditions were often facilitated by variations in the age at marriage and the age of entering parenthood (Galloway, 1986; Lee, 1987, 1997), and similar variations have also been documented in anthropological populations, especially—but not only—when the marriage and first birth are closely tied to sexual and physical maturation that are subject to important variations caused by energy/food availability (Ellison, 1994; Menken, Trussel, and Watkins, 1981; Udry and Cliquet, 1982). If this pattern has prevailed for a sufficiently long time, evolutionary processes should have selected cognitive abilities and decision-making rules that lead to an optimal age at first birth and an optimal progression to the second child (Kaplan, Hill, Lancaster, and Hurtado, 2000; Kaplan and Lancaster, 2003). Moreover, this selection should have favored flexible strategies for the level and timing of early fertility in order to allow individuals to adjust their early fertility to new socioeconomic and environmental conditions.

These selection pressures would appear to be especially potent for females. Males are critically important to any broad model of human reproduction. However, the mechanisms by which biological dispositions are adjusted by environmental contingencies are represented most clearly in females. For example, the relation between nutrition or environmental conditions and fecundity is most clearly documented for females (Ellison, 1994; Menken, Trussel, and Watkins, 1981; Udry and Cliquet, 1982), which is related to the higher energy requirements of reproduction for females than for males. This asymmetry also persists in more modern contexts. For instance, a couples pregnancy would not necessarily influence a fathers educational and/or career activity as fundamentally as it would the mothers. Trade-offs between reproduction and career or education are more relevant for females than for males. For these reasons, we focus most of our analyses in this paper on female fertility where we expect stronger interactions between the relevance of genetic influences and the specific socioeconomic or demographic context. While we do not in any sense wish to ignore the male role in the process of reproduction, we will treat males–both in comparison to females, and also as being of interest in and of themselves—in future work.

The initial decline of fertility in European societies during the late 19th and early 20th century had a relatively modest effect on early fertility behavior and a substantial part of

the reduction in the number of children was caused by stopping behavior, i.e., the limitation of fertility within marriage after the desired number of children has been attained (Coale, 1986). The fertility decline during the demographic transition therefore implied more substantial transformations for 'late' than for 'early' fertility. In more recent decades, however, this pattern has changed and the patterns of early fertility behavior have been substantially transformed during the Second Demographic Transition. For instance, during the period from 1960 to 1995, the mean age at first birth in Denmark increased from 23.1 to 27.5 years, the proportion of out-of-wedlock births increased from 7.8 per cent to 46.5 per cent, and cohabitation prior to marriage has become commonplace (Eurostat, 1998; Knudsen, 1993). The cohorts born around 1945 experienced merely the beginning of this transformation in early adulthood, as the youngest cohorts in our study (born 1968) faced a social and demographic context of early fertility that differed substantially from that experienced by their predecessors born 23 years earlier.

Patterns of early fertility are therefore particularly interesting for investigating the interplay between nature and nurture for at least three reasons. First, early fertility behavior should have been subject to strong evolutionary selection because it is an important determinant of complete fertility and fitness. Second, the determinants of early fertility have been very flexible in order to provide a response mechanism to changes in climate and socioeconomic conditions. Third, the dramatic changes in the socioeconomic context of early fertility raise the questions of how these changes affected the relative contributions of genetic and environmental to fertility variations within recent cohorts.

3.2 Data and methods

Our analyses of early fertility are based on a unique data set consisting of 4776 female twin pairs from the Danish Twin Register (Kyvik, Christensen, Skytthe, Harvald, and Holm, 1996; Kyvik, Green, and Beck-Nielsen, 1995) who were born during 1945–68. These data include *all* female twin pairs born in Denmark between 1945 and 1968 whose twin status and zygosity could be identified.[2] The zygosity of same-sexed twins was determined by four questions about the similarity of the twins in a twin pair as either monozygotic (identical), dizygotic (fraternal) or of uncertain zygosity. The method has been shown valid to determine the zygosity correctly in approximately 95 per cent of twin pairs (Hauge, 1981).[3] The timing and level of early fertility in these cohorts has been established via a link with the Danish Civil Registration System (*CRS*) covering all births to these twin cohorts through 31 December 1998.[4,5] The fertility patterns of twins obtained from this linkage does not differ between monozygotic and dizygotic twin pairs, and they corresponds very closely with the fertility pattern of the Danish general population (Kohler, Knudsen, Skytthe, and Christensen, 2002b). Moreover, these data are not subject to potential recall errors or selective survey participation.

The availability of this population-based data on the timing and level of fertility of Danish twins allows us to study the genetic influences on fertility with a specific consideration of (*i*) differences between cohorts and (*ii*) interactions between the socioeconomic environment of fertility decisions and the relevance of genetic influences. However, any fertility measurement at a constant age is not necessarily a comparable indicator of early fertility across cohorts due to the fact that there has been a widespread and substantial postponement of fertility. We therefore standardize for the postponement of childbearing by choosing a definition of early fertility relative to the fertility behavior in each cohort. In particular, we consider in our analyses the following two measures of early fertility: (*a*) an

early onset of fertility, defined as having a child by the age (in complete years) when only 12.5 per cent of a cohort have experienced fertility, and (*b*) the *level of early fertility*, defined as the number of children at the age at which 25 per cent of cohort members have had a first child. These indicators are not affected by the delay in childbearing and they reflect the same notion of early fertility across cohorts: an early onset of fertility indicates that a woman belongs to the first in her birth cohort who have any children, and the level of early fertility indicates that a woman has relatively many children early in life as compared to her cohort mates. The primary difference between the above indicators is that the level of early fertility is measured at an age of 21–25 years, that is, on average about 2 years later than the measurement for an early onset of fertility. This difference is potentially important since fertility behavior at these slightly older ages reflects to a larger extent conscious fertility and marriage decisions, and it is less affected by a potentially unwanted early birth.

[Table 1 about here.]

The summary statistics in Table 1 show that the age at which 12.5 per cent of a cohort have experienced fertility increases from 19 years (for the cohort born in 1945) to 22 years (for the cohort born in 1968). The age at which 25 per cent of a cohort have experienced fertility increases from 21 years (for the cohort born in 1945) to 25 years (for the cohort born in 1968). Mean fertility at this age is .36 (SD = .63) with no systematic differences across cohorts. Moreover, less than one percent of individuals have three or more children by this age, so that the primary variation occurs between zero, one and two children. Polychoric correlations for this level of early fertility slightly decline for DZ twins and markedly increase for MZ twins (Table 1). In particular, the correlations for DZ twins in the cohorts born 1945–52, 1953–60, and 1961–68 are respectively .420, .379 and .341, and the corresponding correlations for MZ twins are .326, .545 and 0.578.[6]

Because the fertility measures in our data are either binary ('having an early onset of fertility birth') or concentrated on 0, 1, 2 children ('level of early fertility'), the standard methodology in twin studies for continuous outcomes (DeFries and Fulker, 1985; Kohler and Rodgers, 2000; Neale and Cardon, 1992) is not optimally suited for our purpose. For this reason we choose a different methodology developed for the analysis of binary and ordered outcomes (Kohler and Rodgers, 1999) that is based on bivariate (ordered) probit models. On the basis of this model we can estimate: (*a*) the degree of additive (or narrow) heritability h^2, reflecting the genetic influence on the variation of a phenotype, and (*b*) the extent of shared-environment effects c^2, which reflect the degree to which common environments lead to a similarity among twins. Moreover, this estimation accommodates the fact that it is more appropriate to think of the latent 'propensity' for a specific outcome, instead of the outcome variable itself, when a phenotype is binary or concentrated on a few realizations. (see Appendix A.1 for a more detailed discussion of this latent variable model for twin studies)

Conceptually the application of these bivariate (ordered) probit models is similar to the analyses with polychoric correlations, with the additional advantage that our approach provides a possibility to capture observed socioeconomic changes that affect the level and timing of early fertility. Unfortunately, our register-based data do not include individual-specific socioeconomic information about the twins. We therefore include separate birth-year dummies among the right-hand side variable of our bivariate ordered probit model. These dummy variables capture all cohort-specific influences that affect the timing and level of early fertility for all members in a cohort. The shared environmental effect, c^2,

that is revealed by our estimation then reflects environmental influences that are shared by members of a twin pair net of all environmental influences that are shared also by members of the same birth cohort. It therefore measures the relevance of shared environments for variation in early fertility *within* a birth cohort. All factors that lead to trends in the level and timing of early fertility across cohorts will be captured by the cohort dummies and do not enter the estimates of c^2.

3.3 Results

Table 2 reports the estimated additive genetic variances (heritabilities, h^2) and shared environmental variances (c^2) for our early fertility measures obtained from the above data.

[Table 2 about here.]

Early onset of fertility: Model 1 suggests that for females born during 1945–52 about 45 per cent of the variation in the factors leading to an early onset of fertility is due to shared environmental effects. The estimates also suggest a virtual absence of heritable influences for these cohorts. Shared family background and socialization thus constitute the most important determinants leading to an early onset of fertility for females born during 1945–52. These estimates for c^2 and h^2, however, reverse within merely 20 years. For the cohorts 1961–68, the estimate of c^2 in Model 1 has declined by two thirds as compared to the oldest cohorts. On other hand, the estimate for h^2 has risen to 47 per cent. Hence, almost half of the variation in the determinants leading to an early onset of fertility is due to genetic factors. This result is confirmed by our second specification (Model 2) which yields a positive coefficient for the interaction of h^2 with birth year and a negative coefficient for the interaction of c^2 with birth year. Genetic factors therefore gain, and shared environmental factors lose in importance for more recent birth cohorts (although the second interaction is not significant in this model, the interpretation is confirmed below when we consider the level of early fertility).

Level of early fertility: Model 3 in Table 2 reports the corresponding analysis for the level of early fertility. The estimated coefficients for c^2 and h^2 reveal a pattern across cohorts that is quite similar to the above analyses in Models 1–2: the influence of genetic factors on variation in early fertility increases, and the influences of shared environmental factors diminishes across cohorts born 1945–68. The same finding occurs also in Model 4 that estimates interaction terms between the birth cohort and the coefficients of heritability and shared environmental influence, c^2 and h^2. Because the estimated coefficient for h^2 for the cohorts 1945–52 in Model 3 is negative and statistically nonsignificant, which is consistent with the fact that the respective correlation in the level of early fertility for DZ twins slightly exceeds that of MZ twins (see summary statistics in Table 1), we re-estimate this model with no genetic component for these cohorts. The results of this best-fitting behavior-genetic model are depicted in Figure 2.[7]

[Figure 2 about here.]

The results reported in Models 1–4 and in Figure 2 reveal a striking transformation in the relative contributions of 'nurture' to 'nature' to the determinants of early fertility behavior: For cohorts born 1945–52, shared environmental factors constitute the most important influence that leads to within-cohort variation in the level of early fertility, and heritable factors are virtually absent. This pattern reverses for the cohorts 1961–68. Genetically-mediated

differences among individuals emerge as the most important determinant of within-cohort variation in the level of early fertility, and the influence of shared environmental factors vanishes almost completely.

In additional analyses (Models 5 and 6 in Table 2) we measure the level of early fertility at a constant age of 23 years instead of using a shifting age-limit as in Models 3–4 that adjusts for the postponement of childbearing across cohorts. The comparison between these two measurements of the level of early fertility provides a possibility to decompose the cohort trends in c^2 and h^2 in Table 2 into (a) factors that are due to the postponement of fertility, and (b) factors that change the relative importance of genetic and shared-environmental influences on early fertility at a constant age. Our results show that the decline in the relevance of shared environmental factors across cohorts is decreased if early fertility is measured at a constant age. In particular, the fertility behavior of the cohorts 1945–52 is subject to smaller shared environmental influences at age 23 as compared to Models 3 and 4. This change is expected because the most important source of shared environmental influences, the parental household, is more separated in terms of age from the measurement of fertility in Models 5 and 6, where fertility is measured at age 23, as compared to Model 3 and 4, where fertility is measured at ages ranging from 21 to 22 years. The most important result of Models 5 and 6 is that the increase in the relevance of genetic influences for early fertility behavior seems to be independent of the general postponement of fertility. This increase also occurs if early fertility is measured at a constant age of 23 instead of relative to the cohort fertility behavior.

In summary, the presence of strong genetically-mediated influences on early fertility in the most recent cohorts in Table 2 supports the evolutionary argument that early fertility behavior may still be under selection pressure even in contemporary societies (Kirk, Blomberg, Duffy, Heath, Owens, and Martin, 2000). In addition, this finding is also consistent with the existing behavioral-genetic evidence on fertility precursors. For instance, several studies have documented genetic influences on the onset of puberty, sexual behavior, dating and marriage (Dunne, Martin, Statham, Sltske, Dinwiddie, Bucholz, Madden, and Heath, 1997; Mealey and Segal, 1993; Rodgers, Rowe, and Buster, 1999; Rowe, 2000; Treloar and Martin, 1990), on the desire for children and the desired age at first birth (Kohler, Rodgers, and Christensen, 1999; Rodgers and Doughty, 2000; Rodgers, Kohler, Kyvik, and Christensen, 2001b), as well as some personality characteristics like anxiety or nurturance that are associated with an early fertility behavior (Miller, 1992; Miller, Pasta, MacMurray, Chiu, and Comings, 1999). Despite this apparently ample evidence of genetic influences on fertility precursors, there is little past evidence for heritability of early fertility. An exception is the study Rodgers and Doughty (2000) that finds evidence for heritability in fertility among U.S. adults in their early 20's, although the patterns across age were somewhat inconsistent (with significant heritabilities in the early and late 20's, but little heritability in the mid-20's).

An important reason for this limited evidence about genetic influences on early fertility may be the interaction of genetic influences with the societal context of early demographic behavior. The Danish twin cohorts born 1945–68, which underlie our investigation of early fertility, attained early adulthood during a period during which the societal context of early demographic behavior was profoundly transformed as part of the Second Demographic Transition. In the presence of strong social and normative influences of fertility and marriage behavior, as well as in the presence of tight economic conditions that restrict individuals' choices in early demographic behavior, genetic influences on fertility precursors may not translate to genetic influences on fertility outcomes. In these situations the socioeconomic

and cultural context of early fertility is likely to dominate in demographic outcomes. This environmental pressure leaves little room for genetically-mediated differences to express themselves in early fertility behavior. As a consequence, heritability h^2 is low, while shared environmental influences c^2 are of considerable relevance. This "constraint" on genetic influences exerted by the environment is likely to lessen during the second demographic transition and the trend towards low fertility. For instance, Udry (1996, p. 335) predicted this interaction between the importance of biological factors and the societal context argued that low-fertility societies are better suited for studying biological factors: "Low-fertility societies provide wide behavior choice. Where behavior choice is broad and opportunities are egalitarian, biological variables, reflecting natural differences in behavioral dispositions, explain increasing variations in behavior. Applications of this principle to demographic research suggests that, increasingly, gendered behavior, fertility, contraception, abortion, nuptiality, occupational choice and other behaviors of interest to demographers will be influenced by biological choice."[8]

Comparable analyses of the level of early fertility in male cohorts, which are not reported here, yield a statistically significant estimate of .30 for heritability. The results thus indicate that genetic influences on an early onset of fertility seem to be present also for males. At the same time, we cannot conclusively support a time trend towards an increasing relevance of genetic factors. Neither the main effect for shared environmental factors is statistically significant in the different birth cohorts if we conduct the analyses in Table 2 for males, nor are the differences between the heritability estimates h^2 in the three cohorts. The results for males therefore suggest the presence of genetic influences on an early onset of fertility, but they do not suggest that these genetic influences are subject to a clear trend across cohorts. We expand on these results for males—and a comparison between males and females—in future research.

4 Compete Fertility (Cohorts 1870–1960)

4.1 Genetic influences on variation in complete fertility

In the subsequent analyses we shift our focus from early fertility to complete fertility using cohorts born 1870–1960. The period spanned by these life-experiences of these cohorts includes the fertility decline during the demographic transition, the baby boom and bust, and the second demographic transition (see Section 2.1). Most importantly, this long-term time series allows us to investigate whether the demographic changes during the last century also affected the relative importance of socioeconomic and biological influences on fertility behavior. In particular, these long-term analyses allow us to address the question of whether this shifting relevance between 'nurture' and 'nature', which we have documented in the previous section for early fertility, is a historically unique phenomenon in recent decades or a phenomenon that has also occurred during earlier periods of rapid demographic change.

4.2 Data and methods

Our long-term analysis of heritabilities across cohorts combines different segments of the Danish Twin Register. The first set of twins in our analyses was born during 1870–1910, and these twins were interviewed at several times in the 1950–60s primarily for health related issues (Hauge, 1981; Hauge, Harvald, Fischer, Gotlieb-Jensen, Juel-Nielsen, Raebild, Shapiro, and Videbech, 1968). This part of our twin data has already been used in our earlier

analyses (Kohler, Rodgers, and Christensen, 1999) and is reanalyzed here. The remaining parts of our twin fertility data are analyzed for the first time. In particular, the second set of twins is born during 1900–23 and these twins were interviewed as part of the *Longitudinal Study of Aging in Danish Twins* (LSAD) in several surveys on health related aspects during 1995–97 (McGue and Christensen, 1997). These surveys asked about the number of children ever born as part of the general socioeconomic background questions.[9] The third part of our twin data consists of twins born during 1931–44 who were interviewed in 1997 on a wide range of health related issues and their socioeconomic determinants (Gaist, Bathum, Skytthe, Jensen, McGue, Vaupel, and Christensen, 2000).[10] The final part of the data consists of cohorts 1945–60 for which the timing and level of fertility has been obtained from a link with the birth registration covers all children born until the end of 1998 (see Section 3.2).[11]

The dependent variable for our analysis is the number of children born to each twin. For cohorts born before 1945, this fertility measure pertains to complete fertility. For cohorts born after 1945, fertility is measured up to December 1998 and it is therefore 'incomplete' fertility for cohorts that had not finished childbearing as of 1998. Since our analysis is restricted to cohorts born up to 1960, that is, cohorts who were 38 years or older in 1998, the truncation of late fertility is not very relevant.

[Table 3 about here.]

Summary statistics about the sample composition and the fertility level of the twins are reported in Table 3. The number of female twin pairs available for the analysis is clearly largest for the early cohorts 1870–1910 and again for the post-war cohorts born after 1945. For the interim years between 1910 and 1945 the data are somewhat sparse since the surveys on health, which provide the fertility information used in our study, include only a sample of all twin pairs in these cohorts. Across all cohorts included in our study, the fertility levels of the twins in Table 3 correspond relatively well with the overall trends in fertility (see Figure 1) and other known patterns about Danish fertility, such as the trend in childlessness (Anderson, 1977; Knudsen, 1993; Matthiessen, 1970).

We estimate the heritability (h^2) and common environmental variance (c^2) via a 'local polynomial regression approach' that builds on DeFries and Fulker (DF) analyses. The latter have been proposed by DeFries and Fulker (1985) as a method of estimating h^2 and (c^2) with twin data by a simple linear regression of a co-twin's trait on the twin's trait and the degree of genetic relatedness. In particular, the 'augmented DF-analysis' estimates the regression

$$w_{1j} = \beta_0 + \beta_1 w_{2j} + \beta_2 R_j + \beta_3 R_j w_{2j}, \tag{1}$$

where w_{ij} is the trait value of twin $i = 1, 2$ in pair j, and R_j is the degree of genetic relatedness of the twin pair. In this regression the coefficient β_0 provides an unbiased estimate for the shared environmental effect (c^2) and the coefficient β_3 provides an estimator for the genetic influences (h^2) on a trait.[12] The ambiguity as to which twin's trait should be used as the dependent, and which as the independent variable, is frequently resolved by using double-entry. Each twin pair is entered twice in the data, and each member of a twin pair provides once the dependent and once the explanatory variable. In Kohler and Rodgers (2000) we have established the correct asymptotic covariance matrix of the coefficients obtained via this DF analysis with double-entry data.

The local polynomial regression approach is an extension of this method that allows for the fact that the coefficients $\beta_0, \ldots, \beta_3$ may not be constant over time or birth cohorts. As

compared to other varying coefficient models (Hastie and Tibshirani, 1993), the local polynomial regression approach is easily implemented and has very good asymptotic properties (Fan and Gijbels, 1996). In particular, we denote as $\beta(\tau) = (\beta_0(\tau), \beta_1(\tau), \beta_2(\tau), \beta_3(\tau))'$ the coefficient vector that pertains to a particular birth cohort τ. Intuitively, the estimate of this varying coefficient vector is obtained from the standard DF regression in Eq. (1) with the addition that each term on the right-hand-side is interacted with the difference between a twin pair's birth year and the time point for which the estimate $\hat{\beta}(\tau)$ is obtained. This interaction term therefore allows for the possibility that each coefficient in the DF regression Eq. (1) exhibits a cohort-trend. In order to allow for a flexible shape of this cohort trend, the regression is locally weighted so that twin pairs that are born relatively close to the estimation-year τ receive a larger weight than twin pairs that are born in years that are relatively distant to τ. As a result, the locally weighted DF regression estimates a smooth curve for each parameter of the DF regression (1). Our particular interest, of course, is focused on the cohort trends of the estimates of heritability h^2 and shared environmental influences c^2. (See Appendix A.2 for a more detailed discussion of local polynomial regression applied to DF analyses.)

Two specifications of the genetic model can be distinguished and implemented with the above DF-regression. The standard additive model assumes that many genes contribute additively to a phenotype. In this case the coefficient of genetic relatedness R_j in Eq. (1) equals 0.5 for DZ and 1 for MZ twins since fraternal twins share on average half and identical twins all their genes. Dominance effects due to the interaction of genes at one locus of the chromosome can be implemented in the estimation of the DF model by specifying a coefficient of genetic relatedness that equals 0.25 for DZ and 1 for MZ twins. The coefficient of heritability in these dominance models is denoted d^2 and is interpreted similarly to h^2. Specific tests for the presence of dominance versus additive effects are only possible when shared environmental influences can be dropped from the analysis (Waller, 1994), which is not possible across all cohorts in our analysis.[13] At the same time, an indication for the presence of dominance effects is obtained when the analysis of the standard additive model yields estimates for shared environmental effects that are negative. In this case, the resemblance between MZ twins as compared to DZ twins is "too strong" in order to be consistent with additive genetic influences, and the analysis should be re-estimated using the dominance genetic influences.[14]

4.2.1 Results

Figure 3 displays the results obtained from the additive and dominance genetic model for female twin cohorts born 1870–1960. The dashed-dotted line shows the fertility level for the cohorts. With the exception of the first years, the cohorts follow the same fertility trend as the general population in Figure 1: cohort fertility declines substantially during the demographic transition and then increases slightly due to the post-war baby boom. For cohorts born after 1940 the fertility level exhibits again a downward trend, and for the youngest cohorts this is partially due to incomplete fertility. The full line in Figure 3 depicts the estimated heritability (h^2) across birth cohorts, and the broken line displays the corresponding estimates for the shared environmental effect (c^2). The characters 'c' and 'h' at the bottom of the graph indicate when the estimated c^2 and h^2 is statistically different from zero at the 10 per cent level. Because the estimates for c^2 become negative in the 1880s and after 1955, this suggests that a dominance genetic model may be more appropriate for the analysis (the significance of this dominance effect is indicated by 'd' at

the bottom of Figure

3b). This choice between the additive and dominance genetic model only affects the level of the estimated coefficient for heritability and shared environmental influences, but not the respective time trends. We therefore present the results of both the additive genetic model (Figure 3a) and of the corresponding dominance genetic model (Figure 3b). In the Appendix Table A.1 we also report standard DF regressions that estimate c^2 and h^2 separately for different groups of cohorts, and provide significance tests for differences between these cohorts.

[Figure 3 about here.]

Similar to our earlier study (Kohler, Rodgers, and Christensen, 1999), we find that the 'pre-transitional' cohorts born during the 1870s are characterized by a low genetic influence and a high shared environmental influence on fertility behavior.[15] The relevance of these shared environmental factors, however, fades over time and genetic factors gain in importance as we encounter the cohorts with rapidly declining fertility who achieved the early stages of the fertility decline during the demographic transition.

After a peak of these genetic influences around 1885, this pattern reverses. Shared environmental factors regain in importance and genetic factors loose in relevance. While our earlier study was only based on cohorts until 1910, the present analysis allows us to observe this trend further. Female cohorts born until 1915 continue to be characterized by low genetic and moderately strong shared environmental effects. In the 1920s this pattern alters again. Genetic factors regain a dominant influence while shared environmental factors fade. After 1945 we observe a renewed change towards an increasing relevance of genetic and a diminishing relevance of shared environmental factors. This pattern also mirrors the increase in the relevance of genetic variation in early fertility that we have found in Section 3.

It is noteworthy that the sum of genetic and shared environmental factors has remained substantially constant in their influence on fertility across cohorts, as the two contributing factors 'nature' and 'nurture' each differed individually. Across all cohorts born 1870–1960, there has been relatively little change in the overall pattern that approximately 25–35 per cent of the variation in complete fertility is attributable to the joint influence of genetic and shared environmental effects. About 65–75 per cent of the variation in fertility therefore is attributable to measurement error and non-shared environments of the twins, among which marriage/cohabitation and the characteristics of the partner probably constitute important elements. Although the combined influence of shared environmental and genetic factors on within-cohort variation in complete fertility has therefore remained approximately constant, the relative importance of these two factors was subject to important shifts. Our analyses reveal that the birth years 1870–1960 contain cohorts for which genetic factors exerted a strong effect on within-cohort variation in fertility, while in other cohorts shared environmental factors are primarily responsible for this variation. This shifting relevance between 'nature' and 'nurture' indicates important interactions between a cohort's socioeconomic and demographic context during the prime ages of childbearing and the relevance of genetic factors in variation of fertility between individuals.

5 Between Nurture and Nature: The Remarkable Shifts in the Determinants of Female Fertility over Time

The first part of our study shows that early female fertility behavior—that is, fertility behavior up to ages 25—has been subject to an important transformation in merely a quarter of a century: shared family background and socialization are consistent with about 50 per cent of the variation in early fertility for females born 1945–52, while heritable influences contribute about 50 per cent of the variation in female cohorts born 62–68. Genetic influences are almost absent for the former cohorts, and shared environmental factors are of only small relevance for the latter cohorts. The second part of our study provides a long-term perspective on these changing contributions of genetic and shared environmental factors, and focuses on the complete fertility of twins born during a period that ranges almost one century from 1870 to 1960. The analyses suggest that the *joint* influence of shared environmental and genetic factors on within-cohort variation in complete fertility has remained remarkably stable across cohorts 1870–1960 and contributes to about 25–35 per cent of the total variance fertility. However, the relative contributions of 'nature' and 'nurture' shift across cohorts. In particular, we find that shared environmental effects are relevant for the fertility of female cohorts born in the 1870s, while genetic factors do not seem to be an important determinant. This pattern changes quite drastically as cohorts participate in the demographic transition. Genetic factors attain their highest influence for cohorts born around 1885, while shared environmental factors loose their importance. As we encounter cohorts born at the beginning of the 20th century, the relative roles of 'nature' and 'nurture' reverse again and shared environmental factors constitute the most important element. For birth cohorts born in the 1920s, there is a renewed reversal and genetic factors temporarily extend their influence. Starting with cohorts born in the 1950s, we observe a renewed and marked increase in the relevance of genetic factors for variation in complete fertility, following a similar pattern as is found for early fertility, and a slow decline in the importance of shared environmental factors.

Although the evidence is necessarily indirect and demands further investigation, the above shifts between 'nurture' and 'nature' suggest a systematic interdependence between demographic trends, the socioeconomic context of cohorts, and the relevance of genetic influences during the last century. The absence of genetic, and the relevance of shared environmental influences in the oldest cohorts is consistent with theoretical predictions. In particular, evolutionary theories predict a small relevance of genetic variation in fertility in pre-transitional societies (Fisher, 1930), compared to many demographic theories that suggest social and normative influences on fertility that should be relatively strong (e.g., Lesthaeghe, 1980). As the demographic transition progresses, these social restrictions relax, and the socioeconomic conditions in general facilitate a wider choice of demographic behavior that includes the conscious control of marital fertility. This changing context of fertility decisions apparently leads to a fading of shared environmental influences, and an emergence of strong genetic influences on fertility behavior. For female cohorts that are born after 1890, shared environmental factors regain influences. We think that this is related to the fact that the early adult life experiences of these cohorts were severely affected by the various political and economic difficulties in the first half of the 20th century. In these difficult periods the relevance of shared environment may have reemerged in a different form: economic and political crises lead to an overall lower fertility level, and also to an greater restriction of demographic choices by socioeconomic conditions. Hence, the number

of children may have become more dependent on the immediate social environment, such as the economic conditions of the parental household, which in turn is reflected in an increased relevance of shared environmental factors in our study.

Consistent with this interpretation is the fact that genetic influences are strengthened for cohorts born around 1920, which are the cohorts that gave birth to a large extent during the post-war baby boom and caused the rapid increase in the total fertility rate in the late 1940s. In our opinion, the resolution in the earlier social and economic crises reflects itself not only in an increased fertility rate in general. The increased behavioral choices also favored the re-emergence of genetic influences on fertility decisions. This effect, however, has been short-lived—similar to the peak of the baby boom itself—and cohort born after 1945 are characterized by 'only' moderate genetic influences and moderately strong shared environmental effects.

The most recent shift between 'nurture' and 'nature' occurs for female cohorts born after 1945. In particular, our study documents an increase in the relevance of genetic factors for variation in both early fertility behavior and complete fertility. The cohorts who are affected by this shift have experienced and facilitated the Second Demographic Transition with a substantial postponement and reduction of fertility, a rise of out-of-wedlock childbearing, and the emergence of cohabitation as the most common partnership form for young adults. This process increased not only the range of demographic choices and other life-course alternatives that are available to individuals in younger cohorts, but it also implied an increasing individualization of demographic behavior (Lesthaeghe, 1983; van de Kaa, 1987). It seems that this context of individualistic decisions among a broad range of demographic alternatives in the most recent cohorts facilitated the emergence of genetic influences on early female fertility: the increased behavioral 'freedom' in demographic behavior is again associated with the re-emergence of strong genetically-mediated within-cohort variation in fertility.

A reviewer of this paper suggested a related but slightly different interpretation, which also deserves consideration. The reviewer referred to the shifts in c^2 and h^2 as an "oscillator," and suggested that this oscillator is "an important human adaptive strategy for maximizing reproductive success during environmental perturbation." To extend the idea, the reviewer suggested that when the environment changes in ways that preclude reliance on learning and cultural transmission of behaviors, then innate responses must be necessarily be broad and variable. The reviewer concludes, "Not everyone will act correctly, but high individual variability will maximize the possibility of some subset of individuals giving the adaptive response." In this perspective, it makes sense that genetic variance would be latent and unobservable in settings with low social choice, but would emerge and become measurable—especially for beginning childbearing in the first place—at times when there are a broad range of social choices.

In summary, the present study documents on the basis of unique twin data obtained from almost one century of birth cohorts that there seems to be a systematic relation between the relevance of genetic influences on fertility and the socioeconomic context of cohorts. In periods when fertility decisions are most deliberate, and when social norms and economic conditions allow a broad range of life-course alternatives, the heritability of female fertility is high and shared environmental effects fade in relevance. This pattern strongly supports the recent arguments, also within the sociological literature (e.g., Morgan and King, 2001; Udry, 1996), that the understanding of genetic influences—and more generally also evolutionary and other biological influences such as hormones—should become an increasingly relevant aspect in the analysis of contemporary fertility behavior. Moreover, our study

also demonstrates that only the integration of sociological approaches, which focus on environmental influences on behavior, and biological approaches, which focus on biologically mediated variation in a trait, is likely to disentangle the complex web of how social and biological factors interact in order to shape contemporary behavior.

Acknowledgments: We gratefully acknowledge the support we have received from the Max Planck Institute for Demographic Research, the National Institutes of Health, the National Institute on Aging, and the Danish National Research Foundation. We are indebted to Axel Skytthe for research assistance in providing the data used in this research.

A Appendix

A.1 Bivariate (ordered) probit model for estimating heritability and shared environmental influences in early fertility

The bivariate (ordered) probit model used in Section 3 for the estimation of heritability is specified as follows (see also Kohler and Rodgers, 1999): n_{ij}^* is a latent scalar measuring the propensity to have children with $n_{ij}^* = x_{ij}\beta + \varepsilon_{ij}$, where $i = 1, \ldots, N$ denotes twin pairs, $j = 1, 2$ denotes twins within twin pairs, x_{ij} is a $1 \times K_1$ vector of covariates, and ε_{ij} is a normally distributed random term which is independent across twin pairs but correlated within pairs. The observed variable n_{ij} equals 0, 1, …, ω if the latent variable n_{ij}^* falls into the intervals $(-\infty, c_1], \ldots, (c_\omega, \infty)$, where $c_1, \ldots, c_\omega$ are estimated cut points and $\omega + 1$ is the number of categories. The bivariate probit model is a special case with only two categories and $\omega = 1$. The correlation of the random terms ε_{i1} and ε_{i2} within twin pairs is specified as $\rho_i = \delta_1 + \delta_2 R_i$, where R_i is the genetic relatedness of twin pair i. The model is estimated via the maximum likelihood estimation given in Kohler and Rodgers (1999). The coefficients δ_1 and δ_2 yield estimates for the shared environment effect c^2 and the genetic effect h^2 respectively, while the coefficient β estimates the influence of the covariates x_{ij} on the mean realization of a trait.

A.2 Local polynomial regression for estimating heritability and shared environmental influences in complete fertility

Formally, the local polynomial regression that is used in Section 4 for estimating heritability and shared environmental influences in complete fertility is specified as follows. We first define as $\beta(\tau) = (\beta_0(\tau), \beta_1(\tau), \beta_2(\tau), \beta_3(\tau))'$ the coefficient vector in the DF model (see Eq. 1) that pertains to a particular birth cohort τ. In addition, we define as $\delta(\tau) = \nabla_\tau \beta(\tau)$ the first derivative of the coefficient vector $\beta(\tau)$ with respect to the time τ. An estimate for the 'local' value of the parameter $\beta(\tau)$ along with an estimate for the first derivative $\delta(\tau)$ can be obtained by a weighted regression that solves

$$(\hat{\beta}(\tau), \hat{\delta}(\tau)) = \arg\min \sum\nolimits_j \sum\nolimits_{i=1}^{2} [(y_{ij} - \tilde{x}_{ij}(\tau)\beta(\tau))^2 K((t_j - \tau)/s)], \tag{2}$$

where y_{ij} is the trait (fertility, in our case) of twin i within pair j, and $K(.)$ is a kernel function that determines the weight a twin pair j receives in this regression based on the smoothing parameter s, the birth year t_j, and the year τ for which the parameter $\hat{\beta}(\tau)$ is estimated. The vector $\tilde{x}_{ij}(\tau)$ contains the co-twin's trait interacted with both the genetic relatedness of a twin pair and the difference between twin j's birth year and the cohort τ for which the parameter $\beta(\tau)$ is to be estimated. This difference is given by $(t_j - \tau)$.

In particular, the vector $\tilde{x}_{ij}(\tau)$ for twin 1 in a pair is given by $\tilde{x}_{1j}(\tau) = (1,\ w_{2j},\ R_j,\ R_j \cdot w_{2j},\ t_j - \tau,\ w_{2j} \cdot (t_j - \tau),\ R_j \cdot (t_j - \tau),\ R_j \cdot w_{2j} \cdot (t_j - \tau))$. This vector is identical to the right-hand-side variables included in the DF-regression in Eq. (1), with the exception that every term is also interacted with $(t_j - \tau)$ that measures the difference between a twin's birth year and the year τ for which the parameter $\beta(\tau)$ is estimated. The corresponding vector $\tilde{x}_{2j}(\tau)$ for the second twin within a pair is defined identically with only the indices '1' and '2' reversed. The statistical properties of these estimators are given in the statistical literature (e.g., Fan and Gijbels, 1996). In our application to twin data, we additionally adjust the variance estimator for the fact that our analysis were based on double entry twin data (Kohler and Rodgers, 2000). The estimates can be implemented with any statistical package that included weighted OLS regressions.

The statistical literature on these estimators also contains a relatively complicated discussion on how the weighting function $K((t_j - \tau)/s)$, and in particular the parameter s that determines the extent of smoothing, are optimally chosen. Since the proposed methods are not easily implemented with our data, and since these data-driven methods are often only an addition to a more intuitive specification of s, we estimated the above regression using several choices for s and then select a value which provided a good trade-off between reducing the bias in the local estimation of $\hat{\beta}(\tau)$ and the need to obtain estimators with an acceptable variance. The results which we discuss in the next section, however, are not very sensitive to the choice of s, and the specification of this smoothing parameter is not a critical choice in our analyses. The analyses reported below are based on a normal kernel $K(.)$ and a smoothing parameter $s = 8$.

[Table 4 about here.]

Notes

[1]The reader should understand that the word 'influence' in the behavior genetic literature is intended to convey the notion of overlapping variance. While other disciplines might be inclined to interpret this language as implying a strong causal connection, that is not our intention. To say that there is 'genetic influence' or 'environmental influence' means that the model identified variance from these domains overlapping with phenotypic variance, including variance from both direct causal and also correlational sources.

[2]This implies that the twins had to be alive in April 1968 when the Central Person Register was introduced in Denmark. Since mortality in infant and teenage years after WWII was already very low in Denmark, almost all twin pairs are represented in the twin register.

[3]Misclassification of either type will bias heritability coefficients in a negative direction. Misclassifying MZ twins as DZ will inflate the DZ correlation, and misclassifying DZ twins as MZ will deflate the MZ correlations. In both cases, the result is a convergence of the MZ–DZ correlations, which results in an (artificially) lower heritability. Alternatively, mis-classification of either type will also act to bias c^2 in a positive direction, with misclassification of DZ twins as MZ having a greater positive bias on c^2 than misclassification of MZ as DZ.

[4]A birth is included if the maternal reference in the Danish CRS is to the Twin Register. Still-born children are not included in the data set, because no Personal Number is assigned to them. The links in the *CRS* between children and mothers represent the legal parenthood, and the register contains no information about the biological parents of adopted children. However only about 1.2 per cent of the children born in the study period are adopted according to the official statistics, and this proportion is likely to be much lower for early fertility. A further description of the data linkage and the Danish twin register is contained in Kohler, Knudsen, Skytthe, and Christensen (2002b).

[5]It is also worth noting that there seem to be no differences in the fecundity between female MZ and DZ twins and neither between twins and singletons (Christensen, Basso, Kyvik, Juul, Boldsen, Vaupel, and Olsen, 1998)

[6]Because the DZ twin correlation for the first set of cohorts exceeds that of MZ twins, we will argue below that a behavior genetic model with no shared environmental effects for these cohorts is most appropriate.

[7]The analyses of early fertility in Table 2 assumes an additive genetic model. Theoretical arguments suggest that the additive genetic variance in traits like fertility, which are highly relevant for fitness, should be reduced by natural selection over time. Remaining genetic effects are then mainly due to dominance effects in which genes at one locus of the chromosome interact, rather than add up, to affect behavior (for a discussion of Fisher's FTNS see Section 2.3 and Rodgers, Hughes, Kohler, Christensen, Doughty, Rowe, and Miller 2001a). In the presence of such dominance effects, the resemblance between parent and children or between ordinary siblings is relatively weak despite the presence of strong genetic influences on a behavior. Consistent with this hypothesis, studies of fecundity (measured by the

waiting time to pregnancy) and the age at menarche or have found the presence of non-additive genetic influences (Christensen, Kohler, Basso, Olsen, Vaupel, and Rodgers, 2003; Treloar and Martin, 1990). Similar reasoning would suggest the presence of nonadditive factors also in early fertility. Tests for such non-additive genetic factors with twins who are raised together is possible only if the shared environmental factors can be dropped from the model. In this case, the behavior genetic model can be re-estimated by constraining the c^2 coefficient to zero and introducing an additional coefficient that captures dominance effects. In particular, dominance effects imply a genetic relatedness of 1 between MZ twins and of .25 for DZ twins (instead of .5 for DZ twins in the presence of additive genetic effects). Because the coefficient c^2 is not statistically significant in Model 5–6 for the cohorts 1961–68, we have re-estimated these models for the cohorts 1961–68 allowing for additive and dominant genetic effects (but no shared environmental effects). The results, however, do not suggest the presence of dominance influences and we cannot empirically support the argument that nonadditive genetic factors constitute important differences in individuals' level of early fertility.

[8]Similar arguments suggesting interactions between the social context and the importance of genetic (or biological) influences on behavior have also been proposed in the evolutionary anthropology literature (see for instance Borgerhoff Mulder, 1992 or Voland, 1998) as well as in the psychological literature (see for instance Rutter, Dunn, Plomin, Simonoff, Pickles, Maughan, Ormel, Meyer, and Eaves, 1997; Rutter, 1997).

[9]Most twins in this *LSAD* sample were born after 1910, but this survey includes some additional twins that overlap with the birth-cohorts of the first data.

[10]Actually, this survey included twins born until 1952. For twins born after 1945, however, more complete fertility information is obtained from the linkage with the birth register (see Section 3.2)

[11]The zygosity of all twins included in our analyses and in the Danish Twin Register has been ascertained using questions about similarity, which has been shown to determine the zygosity correctly in approximately 95 per cent of the twin pairs (Hauge, 1981).

[12]Although the initial development of the DF method focused on selected twin data in which one member of each pair is a proband with a particularly high or low value of a trait, the method has subsequently been generalized to include random samples of twins (Cherny, Cardon, Fulker, and DeFries, 1992; LaBuda, DeFries, and Fulker, 1986; Rodgers and McGue, 1994).

[13]For a related discussion of dominance genetic effects, see also footnote 7.

[14]It can also be that part of these dominance effects are due to effects of assortative mating, which may appear in the analyses just like nonadditive genetic effects (Plomin, Defries, and Mclearn, 1997, p. 144).

[15]The estimates for c^2 become statistically significant if the low estimates for genetic influences, h^2 or d^2, are constrained to zero.

References

Anderson, O. 1977. The Population of Denmark. United Nations / Cicred, New York and Paris.

Bean, F. D. 1983. The Baby Boom and Its Explanations. The Sociological Quarterly, **24** (3):353–365.

Behrman, J. R., M. R. Rosenzweig, and P. Taubman. 1994. Endowments and the Allocation of Schooling in the Family and in the Marriage Market: The Twins Experiment. Journal of Political Economy, **102** (6):1131–1173.

——— 1996. College Choice and Wages: Estimates Using Data on Female Twins. Review of Economics and Statistics, **73** (4):672–685.

Behrman, J. R., and P. Taubman. 1989. Is Schooling "Mostly in the Genes"? Nature-Nurture Decomposition Using Data on Relatives. Journal of Political Economy, **97** (6):1425–1446.

Bongaarts, J., and R. A. Bulatao. (eds.) 2000. Beyond Six Billion: Forecasting the World's PopulationWashington, DC. National Academy Press.

Borgerhoff Mulder, M. 1990. Early maturing Kipsigis women have higher reproductive success than late maturing women and cost more to marry. Behavioral Ecology and Sociobiology, **24**:145–153.

——— 1992. Reproductive Decisions. *In* E. A. Smith, and B. Winterhalder (eds.), Evolutionary Ecology and Human Behavior, pp. 339–374. Aldine de Gruyter, Hawthorne, NY.

Brewster, K. L., J. O. G. Billy, and W. R. Grady. 1993. Social Context and Adolescent Behavior: The Impact of Community on the Transition to Sexual Activity. Social Forces, **71** (3):713–740.

Brewster, K. L., and R. R. Rindfuss. 2000. Fertility and women's employment in industrialized nations. Annual Review of Sociology, **26**:271–296.

Carter, R. 1998. Mapping the Mind. University of California Press, Berkeley.

Cherny, S. S., L. Cardon, D. Fulker, and J. DeFries. 1992. Differential heriability across levels of cognitive ability. Behavior Genetics, **22** (2):153–162.

Christensen, K., O. Basso, K. O. Kyvik, S. Juul, J. Boldsen, J. W. Vaupel, and J. Olsen. 1998. Fecundability of Female Twins. Epidemiology, **9** (2):189–192.

Christensen, K., H.-P. Kohler, O. Basso, J. Olsen, J. W. Vaupel, and J. L. Rodgers. 2003. The Correlation of Fecundability Among Twins: Evidence of a Genetic Effect on Fertility? Epidemiology, **14** (1):60–64.

Coale, A. J. 1986. The Decline of Fertility in Europe since the Eighteenth Century as a Chapter in Demographic History. *In* A. J. Coale, and S. C. Watkins (eds.), The Decline of Fertility in Europe, pp. 1–30. Princeton University Press, Princeton.

Coale, A. J., and S. C. Watkins (eds.). 1986. The Decline of Fertility in EuropePrinceton. Princeton University Press.

Coleman, D. (ed.) 1996. Europe's Population in the 1990sOxford. Oxford University Press.

Council of Europe 2000. Recent Demographic Developments in Europe. Council of Europe Publishing, Strasbourg.

Crane, J. 1991. The epidemic theory of ghettos and neighborhood effects on dropping out and teenage childbearing. American Sociological Review, **96** (5):1226–1259.

Dawkins, R. 1976. The Selfish Gene. Oxford University Press, Oxford.

DeFries, J. C., and D. W. Fulker. 1985. Multiple regression analysis of twin data. Behavior Genetics, **15** (5):467–73.

Dunne, M. P., N. G. Martin, D. J. Statham, W. S. Sltske, S. H. Dinwiddie, K. K. Bucholz, P. A. Madden, and A. C. Heath. 1997. Genetic and environmental contributions to variance in age at first sexual intercourse. Psychological Science, **8**:211–216.

Easterlin, R. A. 1980. Birth and Fortune: The Impact of Numbers on Personal Welfare. University of Chicago Press, Chicago.

Ellison, P. T. 1994. Advances in human reproductive ecology. Annual Review of Anthropology, **23**:255–275.

Eurostat 1998. New Cronos Database: Population and Social Conditions. Eurostat, Brussels.

Fan, J., and I. Gijbels. 1996. Local Polynomial Modelling and Its Applications. Chapman & Hall, London.

Fisher, R. A. 1930. The Genetical Theory of Natural Selection. Clarendon Press, Oxford [1999].

Foster, C. 2000. The Limits to Low Fertility: A Biosocial Approach. Population and Development Review, **26** (2):209–234.

Gaist, D., L. Bathum, A. Skytthe, T. K. Jensen, M. McGue, J. W. Vaupel, and K. Christensen. 2000. Strength and anthropometric measures in identical and fraternal twins: No evidenceof masculinization of femals with male co-twins. Epidemiology, **11** (3):340–343.

Galloway, P. 1986. Long-Term Fluctuations in Population and Climate in the Preindustrial Era. Population and Development Review, **12** (1):1–24.

Gangestad, S. W., and R. Thornhill. 1998. Menstrual cycle variation in women's preferences for the scent of symmetrical men. Proceedings of the Royal Society of London, Series B: Biological Sciences, **265** (1399):927–933.

Gangestad, S. W., R. Thornhill, and C. Garver. 2002. Changes in women's sexual interests and their partner's mate retention tactics across the menstrual cycle: Evidence for shifting conflict of interests. Proceedings of the Royal Society of London, Series B: Biological Sciences, p. forthcoming.

Geary, D. C. 1998. Male, Females: The Evolution of Human Sex Differences. American Psychological Association, Washington, D.C.

Geronimus, A. T., and S. Korenman. 1992. The socioeconomic consequences of teen childbearing reconsidered. Quarterly Journal of Economics, **107**:1187–1214.

Guo, S. W. 1999. The behaviors of some heritability estimators in the complete absence of genetic factors. Human Heredity, **49** (4):215–228.

Halpern, C. T., J. R. Udry, B. Campbell, and C. Suchindran. 1999. Effects of body fat on weight concerns, dating, and sexual activity: A longitudinal analysis of black and white adolescent girls. Developmental Psychology, **35** (3):721–736.

Halpern, C. T., J. R. Udry, and C. Suchindran. 1997. Testosterone predicts initiation of coitus in adolescent females. Psychosomatic Medicine, **59** (2):161–171.

Hastie, T., and R. Tibshirani. 1993. Varying-Coefficient Models. Journal of the Royal Statistical Society, Series B - Methodological, **55** (4):757–796.

Hauge, M. 1981. The Danish Twin Register. *In* S. A. Mednich, A. E. Baert, and B. P. Bauchmann (eds.), Prospective Longitudinal Research: An Empirical Basis for the Primary Prevention of Pshychological Disorders, pp. 217–221. Oxford University Press, Oxford.

Hauge, M., B. Harvald, M. Fischer, K. Gotlieb-Jensen, N. Juel-Nielsen, I. Raebild, R. Shapiro, and T. Videbech. 1968. The Danish twin registers. Acta Geneticae Medicae et Gemellologiae, **2**:315–331.

Herskind, A. M., M. McGue, N. V. Holm, T. I. A. Sorensen, B. Harvald, and J. W. Vaupel. 1996. The heritability of human longevity: A population based study of 2872 Danish twin pairs born 1870–1900. Human Genetics, **97** (3):319–323.

Hill, K., and H. Kaplan. 1999. Life history traits in humans: Theory and empirical studies. Annual Review of Anthropology, **28**:397–430.

Hirschman, C. 1994. Why Fertility Changes. Annual Review of Sociology, **20**:203–233.

Hotz, J. V., S. W. McElroy, and S. G. Sanders. 1999. Teenage childbearing and its life cycle consequences: exploiting a natural experiment. NBER Working Paper No. 7397.

Hughes, K. A., and M. H. Burleson. 2000. Evolutionary causes and consequences of variation in fertility and other fitness components. *In* J. L. Rodgers, D. C. Rowe, and W. B. Miller (eds.), Genetic Influences on Fertility and Sexuality, pp. 7–34. Kluwer Academic Publishers.

Imaizumi, Y., M. Nei, and T. Furusho. 1970. Variability and heritability of human fertility. Annals of Human Genetics, **33** (3):251–259.

Kääär, P., and J. Jokela. 1998. Natural selection on age-specific fertilities in human females: comparison of individual level fitness measures. Proceedings of the Royal Society of London Series B – Biological Sciences, **265** (1413):2415–2420.

Kääär, P., J. Jokela, T. Helle, and I. Kojola. 1996. Direct and correlative phenotypic selection on life-history traits in three pre-industrial human populations. Proceedings of the Royal Society of London Series B – Biological Sciences, **263** (1376):1475–1480.

Kaplan, H., K. Hill, J. Lancaster, and A. M. Hurtado. 2000. A theory of human life history evolution: Diet, intelligence, and longevity. Evolutionary Anthropology, **9** (4):156–185.

Kaplan, H., and J. Lancaster. 2003. An evolutionary and ecological analysis of human fertility, mating patterns, and parental investment. *In* K. W. Wachter, and R. A. Bulatao (eds.), Fertility Behavior in Biodemographic Perspective. National Research Council, Committee on Population, Division on Behavioral and Social Sciences and Education. Washington, D.C.: The National Academies Press.

Kaplan, H., J. B. Lancaster, W. T. Tucker, and K. G. Anderson. 2002. An Evolutionary Approach to Below Replacement Fertiltiy. American Journal of Human Biology, **14**:233–256.

Kirk, K. M., S. P. Blomberg, D. L. Duffy, A. C. Heath, I. P. F. Owens, and N. G. Martin. 2000. Natural Selection and the Quantitative Genetics of Life History Traits in Western Women: A Twin Study. Evolution, **55** (2):423–435.

Knudsen, L. B. 1993. Fertility Trends in Denmark in the 1980s: A Register Based Sociodemographic Analysis of Fertility Trends. Statistics Denmark, Copenhagen.

Kohler, H.-P. 2000. Social Interaction and Fluctuations in Birth Rates. Population Studies, **54** (2):223–238.

Kohler, H.-P., F. C. Billari, and J. A. Ortega. 2002a. The Emergence of Lowest-Low Fertility in Europe During the 1990s. Population and Development Review, **28** (4):641–681.

Kohler, H.-P., L. Knudsen, A. Skytthe, and K. Christensen. 2002b. The Fertility Pattern of Twins and the General Population Compared: Evidence from Danish Cohorts 1945–64. Demographic Research *[online available at http://www.demographic-research.org]*, **6** (14):383–408.

Kohler, H.-P., and J. L. Rodgers. 1999. DF-like Analyses of Binary, Ordered and Censored Variables using Probit and Tobit Approaches. Behavior Genetics, **29** (4):221–232.

——— 2000. DF-Analyses of heritability with double-entry twin data: Asymptotic standard errors and efficient estimation. Behavior Genetics, **31** (2):179–191.

——— 2003. Education, Fertility and Heritability: Explaining a Paradox. *In* K. W. Wachter, and R. A. Bulatao (eds.), Fertility Behavior in Biodemographic Perspective. National Research Council, Committee on Population, Division on Behavioral and Social Sciences and Education. Washington, D.C.: The National Academies Press.

Kohler, H.-P., J. L. Rodgers, and K. Christensen. 1999. Is Fertility Behavior in our Genes: Findings from a Danish Twin Study. Population and Development Review, **25** (2):253–288.

Kohler, H.-P., A. Skytthe, and K. Christensen. 2001. The age at first birth and completed fertility reconsidered: Findings from a sample of identical twins. Max Planck Institute for Demographic Research, Rostock, Germany, Working Paper #2001-006 (available at http://www.demogr.mpg.de).

Kyvik, K. O., K. Christensen, A. Skytthe, B. Harvald, and N. V. Holm. 1996. The Danish Twin Register. Danish Medical Bulletin, **43** (5):465–470.

Kyvik, K. O., A. Green, and H. Beck-Nielsen. 1995. The new Danish twin register: Establishment and analysis of twinning rates. International Journal of Epidemiology, **24**:589–596.

LaBuda, M. C., J. C. DeFries, and D. W. Fulker. 1986. Multiple Regression Analysis of Twin Data Obtained From Selected Samples. Genetic Epidemiology, **3** (6):425–433.

LeDoux, J. 1996. The Emotional Brain: The Mysterious Underpinnings of Emotional Life. Simon & Schuster, New York.

Lee, R. D. 1987. Population Dynamics of Humans and Other Animals. Demography, **24** (4):443–466.

——— 1997. Population Dynamics: Equilibrium, Disequilibrium, and Consequences of Fluctuations. *In* M. R. Rosenzweig, and O. Stark (eds.), Handbook of Population and Family Economics, pp. 1063–1117. North Holland, Amsterdam.

Lesthaeghe, R. 1980. On the Social Control of Human Reproduction. Population and Development Review, **6** (4):527–548.

——— 1983. A Century of Demographic and Cultural Change in Western Europe: An Exploration of Underlying Dimensions. Population and Development Review, **9** (3):411–435.

Lesthaeghe, R., and D. van de Kaa. 1986. Twee demografische transities? *In* R. Lesthaeghe, and D. van de Kaa (eds.), Bevolking: Groei en Krimp, pp. 9–24. Van Loghum Slaterus, Deventer.

Lewontin, R. C., S. Rose, and L. Kamin. 1985. Not in our Genes: Biology, Ideology, and Human Nature. Pantheon Books.

Low, B. S. 1990. Reproductive life in nineteenth century Sweden: An evolutionary perspective on demographic phenomena. Ethology and Sociobiology, **12**:411–448.

——— 1998. The evolution of human life histories. *In* C. Crawford (ed.), Handbook of Evolutionary Psychology: Ideas, Issues and Applications, pp. 131–161. Aldine de Gruyter, Hawthorne, NY.

Low, B. S., A. L. Clarke, and K. A. Lockridge. 1992. Towards an Ecological Demography. Population and Development Review, **18** (1):1–31.

Massey, D. S. 2002. A Brief History of Human Society: The Origin and Role of Emotion in Social Life. American Sociological Review, **67** (1):1–29.

Matthiessen, P. C. 1970. Some Aspects of the Demographic Transition in Denmark. G. E. C. Gad, Kopenhagen.

McGue, M., and K. Christensen. 1997. Genetic and environmental contributions to depression symptomatology: Evidence from Danish twins 75 years of age and older. Journal of Abnormal Psychology, **106** (3):439–448.

McGue, M., and D. T. Lykken. 1992. Genetic Influence on Risk of Divorce. Psychological Science, **3** (6):368–373.

Mealey, L. 2000. Sex Differences: Development and Evolutionary Strategies. Academic Press, London.

Mealey, L., and N. L. Segal. 1993. Heritable and Environmental Variables Affect Reproduction-Related Behaviors, but not Ultimate Reproductive Success. Personality and Individual Differences, **14** (6):783–794.

Menken, J., J. Trussel, and S. Watkins. 1981. The Nutrition Fertility Link: An Evaluation of the Evidence. Journal of Interdisciplinary History, **11** (3):425–441.

Michael, R. T., and N. B. Tuma. 1985. Entry into marriage and parenthood by young men and women: The influence of family background. Demography, **22** (4):515–544.

Miller, W. B. 1992. Personality traits and developmental experiences as antecedents of childbearing motivation. Demography, **29** (2):265–285.

Miller, W. B., D. Pasta, J. MacMurray, W. Chiu, and D. Comings. 1999. Genetic influences on childbearing motivation and parental satisfaction: A theoretical framework and some empirical evidence. *In* L. Severy, and W. B. Miller (eds.), Advances in Population: Psychological Perspectives, Vol. III. Jessica Kingsley.

Miller, W. B., and J. L. Rodgers. 2001. The Ontogeny of Human Bonding Systems: Evolutionary Origins, Neural bases, and Psychological Manifestations. Kluwer Academic Publishers, Boston.

Morgan, S. P. 1996. Characteristic Features of Modern American Fertility. Population and Development Review, **22** (Suppl.):19–63.

Morgan, S. P., and R. B. King. 2001. Why have children in the 21st century? Biological predispositions, social coercion, rational choice. European Journal of Population, **17** (1):3–20.

Morgan, S. P., and R. R. Rindfuss. 1999. Reexamining the Link of Early Childbearing to Marriage and to Subsequent Fertility. Demography, **36** (1):59–75.

Neale, M. C., and L. R. Cardon. 1992. Methodology for Genetic Studies of Twins and Families. Kluwer Academic Publishers, London.

Pampel, F. C. 1993. Relative Cohort Size and Fertility: The Socio-political context of the Easterlin Effect. Americal Sociological Review, **58** (4):496–514.

Penton-Voak, I. S., D. I. Perrett, D. L. Castles, T. Kobayashi, D. M. Burt, L. K. Murray, and R. Minamisawa. 1999. Menstrual cycle alters face preference. Nature, **399** (6738):741–742.

Plomin, R., J. C. Defries, and G. E. Mclearn. 1997. Behavioral Genetics. Freemann & Co.

Rindfuss, R. R., S. P. Morgan, and G. Swicegood. 1988. First Births in America: Changes in the Timing of Parenthood. University of California Press, Berkeley, CA.

Robson, A. J. 2001. The Biological Basis of Economic Behavior. Journal of Economic Literature, **39** (1):11–33.

Rodgers, J. L., and D. Doughty. 2000. Genetic and Environmental Influences on Fertility Expectations and Outcomes using NLSY Kinship Data. *In* J. L. Rodgers, D. C. Rowe, and W. B. Miller (eds.), Genetic Influences on Human Fertility and Sexuality, pp. 85–106. Kluwer Academic Publishers, Boston.

Rodgers, J. L., K. Hughes, H.-P. Kohler, K. Christensen, D. Doughty, D. C. Rowe, and W. B. Miller. 2001a. Genetic Influence Helps Explain Variation in Human Fertility Outcomes: Evidence from Recent Behavioral and Molecular Genetic Studies. Current Directions in Psychological Science, **10** (5):184–188.

Rodgers, J. L., and H.-P. Kohler (eds.). 2003. Biodemography of Human Reproduction and FertilityBoston. Kluwer Academic Publishers.

Rodgers, J. L., H.-P. Kohler, K. Kyvik, and K. Christensen. 2001b. Behavior Genetic Modeling of Human Fertility: Findings from a Contemporary Danish Twin Study. Demography, **38** (1):29–42.

Rodgers, J. L., and M. McGue. 1994. A simple algebraic demonstration of the validity of DeFries - Fulker analysis in unselected samples with multiple kinship levels. Behavior Genetics, **24** (3):259–62.

Rodgers, J. L., D. C. Rowe, and M. Buster. 1998. Social Contagion, Adolescent Sexual Behavior and Pregnancy - A Nonlinear Dynamic EMOSA Model. Developmental Psychology, **34** (5):1096–1113.

Rodgers, J. L., D. C. Rowe, and M. Buster. 1999. Nature, nurture and first sexual intercourse in the USA: Fitting behavioural genetic models to NLSY kinship data. Journal of Biosocial Science, **31** (1):29–41.

Roff, D. 1992. The Evolution of Life Histories: Theory and Analysis. Chapman & Hall, London.

Rossi, A. S. 1994. Eros and Caritas: A Biosocial Approach to Human Sexuality and Reproduction. *In* A. S. Rossi (ed.), Sexuality Across the Life Course, pp. 3–38. University of Chicago Press, Chicago.

Rowe, D. C. 2000. Environmental and genetic influences on pubertal development: Evolutionary life history traits? *In* J. L. Rodgers, D. C. Rowe, and W. B. Miller (eds.), Genetic Influences on Human Fertility and Sexuality, pp. 147–169. Kluwer Academic Publishers.

Rutter, M., J. Dunn, R. Plomin, E. Simonoff, A. Pickles, B. Maughan, J. Ormel, J. Meyer, and L. Eaves. 1997. Integrating nature and nurture: Implications of person-environment correlations and interactions for developmental psychopathology. Development and Psychopathology, **9** (2):335–364.

Rutter, M. L. 1997. Nature-nurture integration: The example of antisocial behavior. American Psychologist, **52** (4):390–398.

South, S. J., and K. D. Crowder. 1999. Neighborhood effects on family formation: Concentrated poverty and beyond. American Sociological Review, **64** (1):113–132.

Stearns, S. C. 1992. The Evolution of Life Histories. Oxford University Press, Oxford.

Thornberry, T. P., C. A. Smith, and G. J. Howard. 1997. Risk factors for teenage fatherhood. Journal of Marriage and the Family, **59** (3):505–522.

Treloar, S. A., and N. G. Martin. 1990. Age at menarche as a fitness trait: Nonadditive genetic variance detected in a large twin sample. American Journal of Human Genetics, **47**:137–148.

Trivers, R. L. 1972. Parental investment and sexual selection. *In* B. Campbell (ed.), Sexual Selection and the Descent of Man, pp. 136–179. Aldine, Chicago.

Udry, J. R. 1988. Biological predispositions and social-control in adolescent sexual behavior. American Sociological Review, **53** (5):709–722.

——— 1995. Sociology and Biology: What Biology Do Sociologists Need to Know. Social Forces, **73** (4):1267–1278.

——— 1996. Biosocial models of low-fertility societies. Population and Development Review, **22** (Supplement):325–336.

Udry, J. R., and R. L. Cliquet. 1982. A Cross-Cultural Examination of the Relationship Between Ages at Menarche, Marriage, and First Birth. Demography, **19** (1):53–63.

van de Kaa, D. J. 1987. Europe's Second Demographic Transition. Population Bulletin, **42** (1):1–59.

Voland, E. 1998. Evolutionary ecology of human reproduction. Annual Review of Anthropology, **27**:347–74.

Waller, N. G. 1994. A DeFries and Fulker regression model for genetic nonadditivity. Behavior Genetics, **24** (2):149–153.

Watkins, S. C. 1990. From Local to National Communities: The Transformation of Demographic Regimes in Western Europe, 1870–1960. Population and Development Review, **16** (2):241–272.

Williams, L. A., and B. J. Williams. 1974. A Re-examination of the Heritability of Fertility in the British Peerage. Social Biology, **21**:225–231.

Wood, J. W. 1994. Dynamics of Human Reproduction: Biology, Biometry, Demography. Aldine de Gruyter, Hawthorne, NY.

Wu, L. L., and B. C. Martinson. 1993. Family-structure and the risk of premaritial birth. American Sociological Review, **58** (2):210–232.

Yashin, A. I., and I. A. Iachine. 1997. How frailty models can be used for evaluating longevity limits: Taking advantage of an interdisciplinary approach. Demography, **34** (1):31–48.

List of Figures

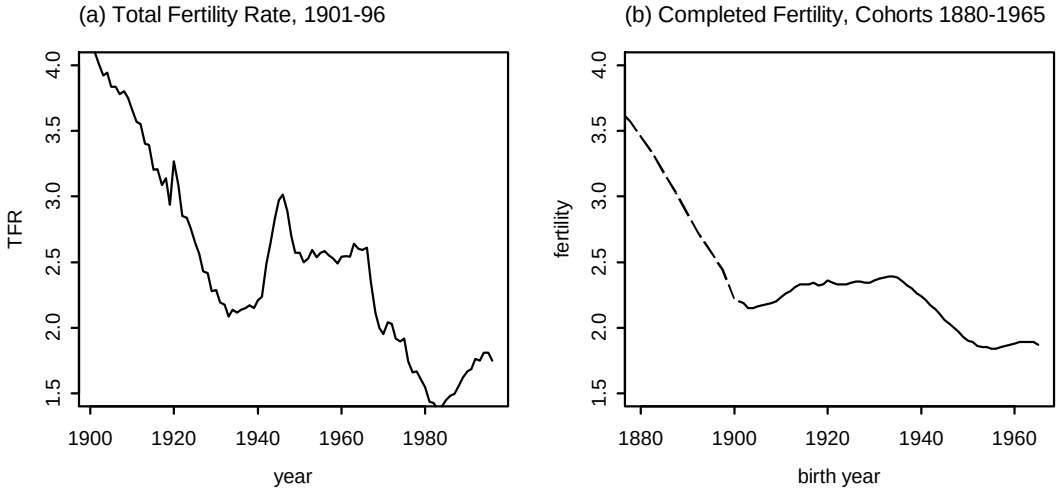

Figure 1: Period and cohort fertility in Denmark (*Sources:* prior to 1900: estimates obtained by Matthiessen 1970; period 1901-1950: Eurostat 1998; after 1950: estimates of complete cohort fertility from Eurostat 1998)

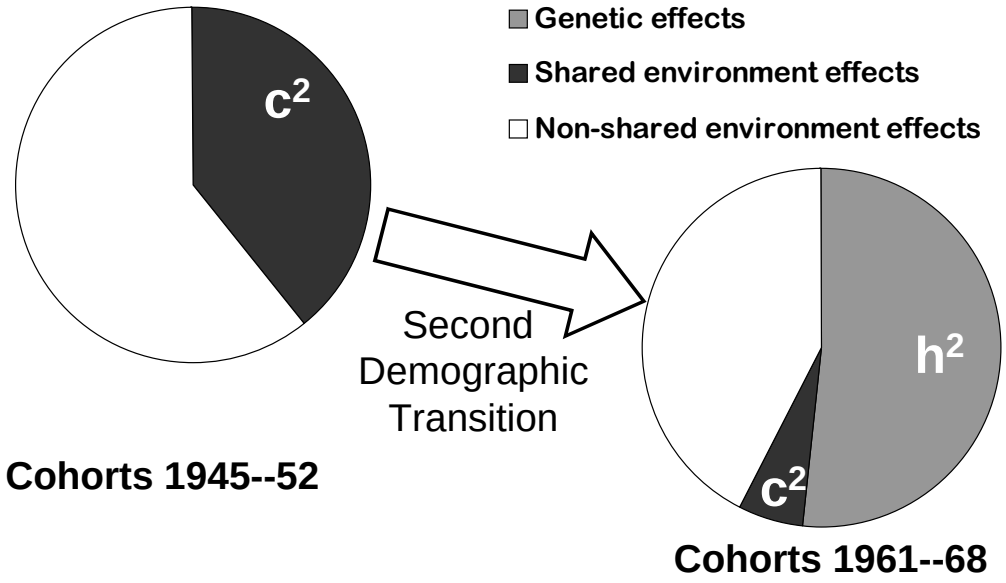

Figure 2: Genetic and shared environmental influences on the level of early fertility in Danish twin pairs based on best-fitting model

Note: For methods see notes to Table 2. Estimates for cohorts born 1945–52: $c^2 = .393$ ($SE = .039$, $p < .001$) and h^2 is constrained to zero because no genetic effects are present; estimates for cohorts born 1961–68: $c^2 = .058$ ($SE = .095$, $p = .54$) and $h^2 = .517$ ($SE = .117$, $p < .001$)

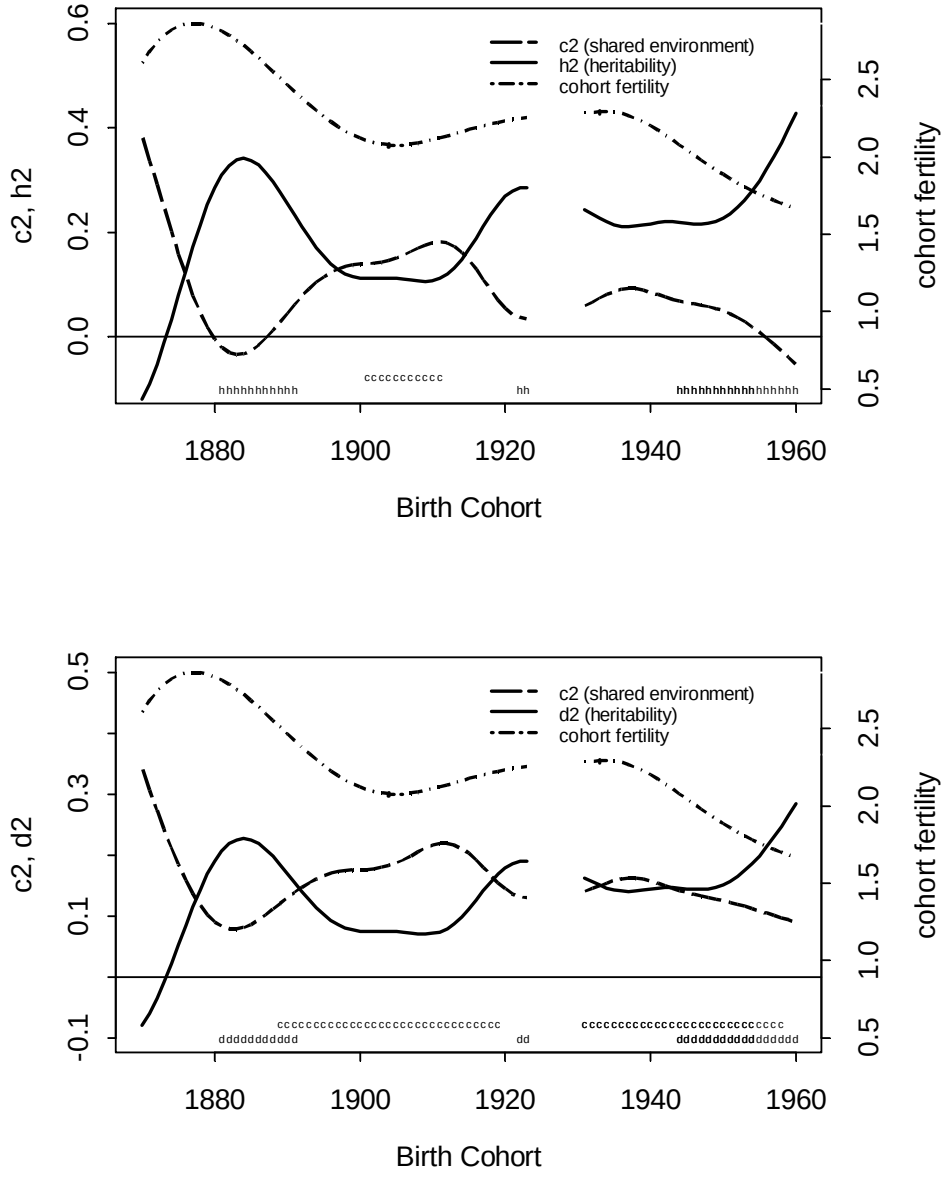

Figure 3: Genetic and shared-environmental contribution to variation in complete fertility, female twin cohorts born 1870–1960

List of Tables

Table 1: Summary statistics for early fertility in the female twin cohorts and within twin pair correlations in fertility outcomes

Cohorts	**1945–52**	**1953–60**	**1961–68**
N (number of twin pairs)	1321	1165	1790
Fertility measure	**Early onset of fertility**		
Age limit 1 (age at which at least 12.5% of a cohort had a first child)[a]	19–20	20–21	21–22
Prop. with at least one child at age limit 1	0.146	0.151	0.151
Within-pair correlation[b]			
DZ twins	0.435	0.437	0.391
	(.30–.55)	(.30–.56)	(.27–50)
MZ twins	0.416	0.542	0.611
	(.22–.59)	(.37–.69)	(.49–.71)
Fertility measure	**Level of early fertility**		
Age limit 2 (age at which at least 25% [b,c] of a cohort had a first child)[a]	21–22	22–23	23–25
Prop. with at least one child at age limit 2	0.295	0.275	0.286
Fertility level at age limit 2[c]	0.372	0.359	0.368
	(.623)	(.604)	(.644)
Within-pair corr. of fert. level[b]			
DZ twins	0.420	0.379	0.341
	(.33–.50)	(.27–.48)	(.25–.43)
MZ twins	0.326	0.545	0.578
	(.18–.46)	(.42–.65)	(.49–.65)

Notes: (*a*) The calculation of the age limits 1 and 2 is specific for each birth year, and it is based on twins born within ±2 years of each birth cohort during 1945–68. The calculation of these age limits is in full years of age; (*b*) Polychoric correlation and 95% confidence intervals estimated with categories 0 and 1 (early onset of fertility) and 0, 1, 2 or more children (level of early fertility); (*c*) Standard deviations in parentheses.

Table 2: Estimated heritability (h^2) and shared environmental effects (c^2) for early fertility behavior

Fertility measure	**Early onset of fertility**	**Level of early fertility**	
Dependent variable	First child before ($\leq$) age limit 1[a]	Number of children at age limit 2[b]	Number of children at age 23
Method	Bivariate Probit[c]	Bivariate Ordered Probit[c,d]	Bivariate Ordered Probit[c,d]
	Model 1	*Model 3*	*Model 5*
c^2 cohort 1945–52	0.445	0.489	0.276
	(0.159)**	(0.116)**	(0.099)**
c^2 cohort 1953–60	0.315	0.190	0.225
	(0.161)*	(0.119)	(0.117)$^+$
c^2 cohort 1961–68	0.143	0.058	0.003
	(0.132)	(0.095)	(0.106)
h^2 cohort 1945–52	-0.038	-0.148	0.116
	(0.231)	(0.171)	(0.139)
h^2 cohort 1953–60	0.219	0.353	0.306
	(0.216)	(0.155)*	(0.153)*
h^2 cohort 1961–68	0.471	0.517	0.588
	(0.165)**	(0.117)**	(0.130)**
Tests for c^2 coefs	–	g, h	f
Tests for h^2 coefs	f, i	g, h, i, j	g, h, j
	Model 2	*Model 4*	*Model 6*
c^2 (cohort 1956-57)	0.297	0.245	0.164
	(0.087)**	(0.063)**	(0.062)**
$c^2 \times$ (birth-year$-1956.5)^e$	-0.016	-0.021	-0.010
	(0.012)	(0.009)*	(0.008)
h^2 (cohort 1956-57)	0.225	0.242	0.338
	(0.118)$^+$	(0.086)**	(0.081)**
$h^2 \times$ (birth-year$-1956.5)^e$	0.027	0.031	0.020
	(0.016)$^+$	(0.011)**	(0.011)$^+$

Notes: Standard errors in parentheses. *p-values:* $^+$ $p \leq 0.10$; * $p \leq 0.05$; ** $p \leq 0.01$. (a) Age limit 1 is the age (in complete years) at which at least 12.5 per cent of a cohort have had a first child; see Table 1 for summary statistics. (b) Age limit 2 is the age (in complete years) at which at least 25 per cent of a cohort have had a first child; see Table 1 for summary statistics. (c) The model includes dummies for each birth-year in order to account for possible cohort effects. (d) The categories are 0, 1, 2 or more children. (e) 1956.5 is subtracted from birth year so that the main effects for c^2 and h^2 pertain to the "middle cohort" cohort that is born during 1956–57. (f) The coefficients for the cohorts 1945–52 and the cohorts 1961–68 are statistically different at a 7.5 per cent or higher significance level. (g) The coefficients for the cohorts 1945–52 and the cohorts 1961–68 are statistically different at a 1.5 per cent or higher significance level. (h) Equality of the coefficients for the three sets of cohorts is rejected at a 5 per cent or higher significance level. (i) The estimates for c^2 and h^2 are statistically different at a 2.5 per cent or higher significance level for the cohorts 1945–52. (j) The estimates for c^2 and h^2 are statistically different at a 2.5 per cent or higher significance level for the cohorts 1961–68.

Table 3: Summary statistics for complete fertility in twin cohorts 1870–1960 (female twin pairs only)

Cohorts	**1870–1910**	**1911–23**	**1931–44**	**1945–60**
N (No. of twin pairs)	2009	392	382	2486
Proportion monozygotic	0.36	0.39	0.53	0.35
Proportion with at least one child	0.68	0.81	0.91	0.79
number of children[a]	2.30[b]	2.25	2.31	1.69
	(2.56)	(1.80)	(1.26)	(1.19)
Within- twin pair correlation[c] of fertility				
DZ twins	0.19	0.22	0.24	0.17
MZ twins	0.29	0.33	0.31	0.29

Notes: (*a*) Standard deviation in parentheses. (*b*) The number of children averaged across all twin pairs in the cohort 1870–1910 is relatively low because of the asymmetric distribution of twin pairs across cohorts: there are many more pairs in cohorts with already low fertility. An estimate of the trends in cohort fertility during 1870–1910 is included in Figure 3; (*c*) Pearson correlation coefficient.

Table A 1: DF-analysis for the number of children in twin pairs 1870–1923 (additive genetic model). Column 2 reports the estimated c^2 and h^2 coefficients, and columns 3–5 report the p-values of tests for equality of the estimated c^2 and h^2 coefficients between different cohorts.

	Estimated coefficient based on DF analysis	p-value of test for equality with cohort		
'old' cohorts		**1881–95**	**1896–1915**	**1916–23**
c^2 (cohorts 1870–80)	0.242	0.17	0.87	0.23
	(0.205)			
c^2 (cohorts 1881–95)	-0.099	–	0.07	0.96
	(0.139)			
c^2 (cohorts 1896–1915)	0.206	–	–	0.16
	(0.094)*			
c^2 (cohorts 1916–23)	-0.089	–	–	–
	(0.190)			
h^2 (cohorts 1870–80)	-0.041	0.13	0.76	0.20
	(0.268)			
h^2 (cohorts 1881–95)	0.455	–	0.07	0.93
	(0.184)*			
h^2 (cohorts 1896–1915)	0.050	–	–	0.17
	(0.125)			
h^2 (cohorts 1916–23)	0.428	–	–	–
	(0.244)$^+$			
'young' cohorts		**1945–54**	**1955–60**	
c^2 (cohorts 1931–44)	0.167	0.73	0.35	–
	(0.207)			
c^2 (cohorts 1945–54)	0.090	–	0.28	–
	(0.081)			
c^2 (cohorts 1955–60)	-0.047	–	–	–
	(0.098)			
h^2 (cohorts 1931–44)	0.136	0.93	0.36	–
	(0.237)			
h^2 (cohorts 1945–54)	0.160	–	0.21	–
	(0.116)			
h^2 (cohorts 1955–60)	0.388	–	–	–
	(0.138)**			

Notes: Standard errors in parentheses. *p-values:* $^+$ $p \leq 0.10$; * $p \leq 0.05$; ** $p \leq 0.01$.