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Multivariate Cholesky Models of Human Female Fertility Patterns in the NLSY

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Abstract

Substantial evidence now exists that variables measuring or correlated with human fertility outcomes have a heritable component. In this study, we define a series of age-sequenced fertility variables, and fit multivariate models to account for underlying shared genetic and environmental sources of variance. We make predictions based on a theory developed by Udry [(1996) Biosocial models of low-fertility societies. In: Casterline, JB, Lee RD, Foote KA (eds) *Fertility in the United States: new patterns, new theories*. The Population Council, New York] suggesting that biological/genetic motivations can be more easily realized and measured in settings in which fertility choices are available. Udry's theory, along with principles from molecular genetics and certain tenets of life history theory, allow us to make specific predictions about biometrical patterns across age. Consistent with predictions, our results suggest that there are different sources of genetic influence on fertility variance at early compared to later ages, but that there is only one source of shared environmental influence that occurs at early ages. These patterns are suggestive of the types of gene–gene and gene–environment interactions for which we must account to better understand individual differences in fertility outcomes.

Keywords

Fertility; Fisher's theorem; FTNS; Heritability; Shared Environment; Multivariate models; Phenotypic plasticity; Hox genes; Life history theory

Fisher's (1930) Fundamental Theorem of Natural Selection (the FTNS) has been misinterpreted by many researchers for many years. The traditional interpretation is that fitness traits and behaviors strongly affected by natural selection will "lose" their genetic variance in the long run. The implication is that fertility and fertility precursors should have little or no genetic variance, and thus zero heritability. This inference is correct if natural selection is the only process at work. But it is not. The FTNS has been interpreted by many to mean that, by definition, for fertility (and other traits related to fitness), $h^2 = 0$. But it does not.

Hughes and Burleson (2000) suggested a number of different processes that re-introduce genetic variance into fitness traits, even while natural selection is washing it out. These include

mutation (the most important), frequency-dependent selection, heterozygote advantage (overdominance), sexual antagonism, and environmental perturbations. Fisher (1930) clearly did not intend or participate in this misinterpretation of the FTNS. In the same source in which he presented the FTNS, he also discussed the role of contraception (an environmental perturbation) in re-introducing genetic variance into human fertility (see also Houle 1992, for additional and more modern discussion of “perturbing forces” from the environment). Further, Fisher presented empirical data suggesting significant heritability in family size among the British aristocracy, $h^2 = 0.40$. Even though this figure was subsequently criticized as being at least partly artifactual (Williams and Williams 1974), its publication demonstrates that Fisher did not expect empirical heritabilities of fitness traits necessarily to be zero. And they are not; at least not always.

Until recently, few researchers looked at the heritability of fertility or its correlates, and presumably the misinterpretation of the FTNS played a role in this neglect—after all, why bother expending research time and energy in estimating heritabilities in traits and processes that Fisher’s theorem appeared to guarantee were zero in the population? The first few glances in this direction were at least ambiguous. Fisher (1930) found significant heritability, but Williams and Williams (1974) dismissed this finding as an artifact of family size changes over time within Fisher’s population of interest. Mealey and Segal (1993) also found low heritabilities of fitness traits in a study of twins raised apart. Houle (1992) reviewed these and several other animal studies.

Since the late 1990’s, a number of studies have been published showing empirical support for significant genetic variance underlying both human fertility outcomes and fertility precursors (e.g., age at first intercourse, age at marriage, age at first pregnancy attempt, etc.) These were reviewed in Rodgers et al. (2001b), and the patterns will be briefly summarized here. In studies of fertility outcomes (typically measured as completed family size), Kohler and Christensen (2000) and Kohler et al. (1999; 2002) found significant heritabilities using population-based data, the Danish twin registry. Kohler et al. (1999) used a time series approach, and found two different periods of significant and moderately high Danish fertility heritabilities, following the two “demographic transitions” that occurred in Denmark (van de Kaa 1987) It is notable that these periods were ones associated with high levels of personal choice in fertility decision-making in Denmark. It is also notable that during long periods within the time series, heritabilities were small or zero. Rodgers and Doughty (2000) used national data from the U.S. (the National Longitudinal Survey of Youth; the NLSY) to estimate significant heritabilities for young adult fertility outcomes (through age 28).

In addition, other studies documented significant genetic variance in fertility precursors. Different phenotypes that have been studied include age at puberty (Doughty and Rodgers 2000; Rowe 2000), age at first intercourse (Dunne et al. 1997; Miller et al. 1999; Rodgers et al. 1999), age at first pregnancy attempt (Christensen et al. 2003), age at marriage (Trumbetta and Gottesman 2000), and fertility motivations/expectations (Miller et al. 2000; Pasta and Miller 2000; Rodgers and Doughty 2000). The findings in these studies were based on univariate biometrical models, in which heritabilities (and also direct measures of genetic variance, coefficients of genetic variation, CV_g ’s) were estimated from empirical data (except for Miller et al. 1999 and 2000, which were based on molecular genetic analyses).

Two studies used multivariate biometrical models to estimate common genetic variance underlying fertility precursors and outcomes. Kirk et al. (2000) fit multivariate models to data from the Australian twin registry, linking age at first reproduction, age at menopause, and a general measure of fitness. They found significant overlapping genetic variance between the two other measures and the fitness measure. Rodgers et al. (2001c) fit a bivariate Cholesky Model to Danish twin registry data that linked age-at-first-attempt-to-get-pregnant (a measure

of fertility motivation) and number of children (a fertility outcome measure) in a sample that ranged in age from 35 to 41. They found a small but statistically significant amount of overlapping genetic variance between these two measures.

Within these studies, the definition of fertility is related to both evolutionary and demographic perspectives. Our focal definition of fertility emerges from the concept of fitness within the evolutionary literature. Though fitness depends on variables other than childbearing (e.g., longevity, age-at-menopause, reproductive success of grandchildren), the number of children that an individual has plays a critical role in overall evolutionary fitness. We operationalize fertility as “number of children born” (for females, “number of live births”) which is the typical way that fertility is measured within the demographic literature, as well as a typical measure within evolutionary accounts.

To summarize these recent empirical results, it no longer appears relevant to ask the question, “Is there (or can there be) genetic variance in fertility precursors or outcomes?” The answer clearly is, “yes, often—but not always.” Obviously, we need more nuanced ways to address the problem that started with the statement of the FTNS, and becomes more focused and interesting in light of the pattern of findings described above.

Udry’s theory, canalization, phenotypic plasticity, and life history theory

Udry (1996), on conceptual grounds (and without supporting data at that time), suggested that biologically based (genetic) variance can only emerge in cultural settings in which there is substantial choice over fertility outcomes. Thus, for example, Udry’s ideas suggested that significant heritabilities should be observed in fertility in modern contracepting societies, but not in natural-fertility regimes. In the latter, any individual differences in fertility motivation—which might be partially realized in contracepting societies—would be phenotypically hidden, because of the reproductive biology of unprotected sex passing through societal and biological norms to have children as often as possible. Kohler et al.’s (1999) empirical results supported Udry’s suggestion, in that they showed rising heritabilities during fertility transition (when falling family sizes reflected increased individual-level choice over reproduction).

An implication of Udry’s theory is that in complex, modern societies like the U.S., where individualism and free choice are emphasized, the magnitude of fertility-related heritabilities and the types of genes underlying them will vary during the life course. This is because the kinds of decisions that individuals face change substantially with increasing age. Thus it seems reasonable to conclude that those genes contributing to fertility heritabilities during the teenage years could be different from those contributing to heritabilities during peak reproductive years of the 20’s and early 30’s, which in turn could be different from those making a contribution to later life fertility from the mid-30’s to the mid-40’s. Similarly, environmental influences would be expected to change over the lifetime as well. For example, the influence of parents on young adults would be expected to mitigate with age. We note that these age patterns can emerge without positing separate selection pressures on the different ages emerging in direct response to modern reproductive patterns (which would probably not have had time to develop in modern societies). Rather, the theory hypothesizes individual differences in underlying (and often latent) fertility motivation, arising through many generations of selection pressures—and realized phenotypically (for example) through different gene–environment interactions at different ages. It is important that the dynamic features of this theoretical orientation are environmental; as fertility choice changes (e.g., across ages), genetic variance either emerges or is suppressed.

The specific micro-level mechanisms underlying developmental changes in genetic expression is not a focus of the current paper, although our thinking has been informed by the

groundbreaking developments that have recently occurred in the domain called Evo-Devo, Evolutionary Developmental Biology. These new models provide explanations for how gene expression can turn off and on developmentally, and also provide genome-level explanations of gene–environment interactions through, for example, Hox genes (which produce RNA that produces repressor protein that binds back to DNA sequences to support inhibition or expression of genes in relation to features of the environment). Carroll (2005) provided a readable account of these new developments (see, e.g., Carroll’s pages 58–59, in which he illustrates this process in relation to bacteria’s ability to repress genetic expression when lactose is not present through the production of the lac repressor protein). His treatment cannot be studied without recognizing the implications of these new models for gene–environment interactions, including in particular the relevance of this work for motivating individual differences in human reproduction. We will further discuss the important role of gene–environment (and also gene–gene) interactions in our Discussion section. In this paper, we use implications from Udry’s theory to predict differences in heritabilities of fertility across ages in the NLSY.

The literature on biological and evolutionary genetics contains mechanisms—genetic canalization and phenotypic plasticity—that are relevant to this discussion. The concept of genetic canalization explains how certain phenotypes can remain constant, even in the presence of genotypic variability. As Flatt (2005) explained, “Canalization implies that a genotype’s phenotype remains relatively invariant when individuals of a particular genotype are exposed to different environments.... genetic canalization can be viewed as a particular kind of epistasis, and environmental canalization and phenotypic plasticity are two aspects of the same phenomenon” (p. 87). Genetic canalization produces fixed phenotypes across different environments. Phenotypic plasticity is the reverse, “the ability of a phenotype associated with a single genotype to produce more than one continuously or discontinuously variable alternative form of morphology, physiology, and/or behavior in different environmental circumstances” (West-Eberhard, 2003, p. 33). Phenotypic plasticity produces multiple phenotypes across different environments. Certain features of human traits or behaviors are apparently adaptive if they are fixed; having a single nose is an example. Other traits/behaviors are adaptive to the extent that they vary in valuable ways; humans’ different facial structure creating different appearances is an example, allowing humans to distinguish family, friends, and potential enemies quickly.

Within Udry’s (1996) theory both canalization and phenotypic plasticity can be identified. In cultural settings in which continual and high-levels of childbearing is expected (often referred to as “natural fertility societies”), there is little social choice; rather, limits to women’s childbearing are only biological, and family sizes are relatively large. Even in the presence of underlying genetic variation in fertility desires, genetic canalization of fertility outcomes means that women have lots of children, limited only by mate availability, infertility, or life-course features (e.g., age at puberty and menopause). This type of setting—a pre-fertility-transition regime—is characteristic of humans who do not exert fertility control, as well as many other species. A form of conscious phenotypic plasticity occurs following fertility transition. Humans choose to limit their childbearing, for rational, volitional reasons (to pursue education, because infant mortality is not problematic, because additional children drain human and financial capital, etc.; see Johnson and Rodgers 2006). In this fertility regime, the trade-off between child quality and child quantity becomes one of choice, and not biological or societal mandate. In this fertility regime, genetic variation in fertility outcomes—which was latent, canalized, and not realized in pre-transition cultures—is expressed, and realized phenotypically. Several scholars have cast these issues within the framework of life history theory (e.g., Geary 2002), which focuses on the sequence within the lifecourse of traits and behaviors that are adaptive. Life history theory accounts for adaptive tradeoffs across different traits and behaviors, including in particular the tradeoff of child quality and child quantity. Rowe (2000) studied

pubertal maturation within a life history perspective. Vigil et al. (2005) used life history theory to frame the relation between child sexual abuse and several fertility-related measures, including menarche, age-at-first-intercourse, and fertility desires. Geronimus (1991) studied teenaged childbearing within a life history perspective. Figueredo et al. (in press) cast a broad array of phenotypes related to personality, medical, and family outcomes within a life history perspective. Within Udry's (1996) theory, human reproductive behavior in post-demographic-transition societies allows for explicit and volitional decision-making in defining these life history tradeoffs; Udry's theory suggests that the more choice, the more decision-making is involved in fertility outcomes, and the more genetic variance will be expressed and measurable.

Method

The methodological approach on which the current study is based is a multivariate biometrical (Cholesky) model fit to national kinship data from the U.S. The variables of interest are fertility outcome measures, defined at different ages, and measured longitudinally. These models allow us to test for common genetic and environmental variance underlying fertility outcomes at different ages. Previous research has identified significant heritabilities at different ages and time periods; in the current research we will evaluate whether different ages share common genetic/environmental variance.

The data

The National Longitudinal Survey of Youth (NLSY) originated as a household probability sample of adolescents first interviewed in spring 1979; the 12,686 respondents were 14–21-years-old on the sampling date of December 31, 1978. The basic sample consisted of all respondents in the age range in the original sample of 3000 households ($N = 6111$), plus additional individuals included to oversample minorities and economically disadvantaged whites ($N = 5295$), and a military sample ($N = 1280$; this latter sample will not have relevance to the current research, because there were no siblings recruited into the military sample).

The NLSY respondents have been followed longitudinally since the original interview, yearly until 1986, and then every 2 years thereafter. Because of funding constraints, follow-up interviews with the military sample and the oversampled disadvantaged white respondents were eliminated in 1984 and 1990, respectively. Except for these systematic deletions, relatively small attrition occurred over this time period; over 80% of both the household sample and the still-eligible oversample were in the interview pool over 20 years later in 2000. Summary information about the NLSY is available from Center for Human Resource Research (2003).

To fit biometrical models requires kinship information indicative of genetic relatedness for different categories of kinship level. Although no questions within the NLSY survey distinguished different kinship levels (e.g., full- from half-siblings), there are variables within the NLSY that allow valid and reliable classification. The most important variables indicated, for every respondent, whether they lived in the same household with their biological mother for every age from 0 to 18, and whether they lived in the same household with their biological father for every age from 0 to 18. By matching the time period for NLSY respondents who lived in the same household in 1979, we developed a kinship-linking algorithm that defined kinship relatedness for a large majority of the eligible kinship pairs in the NLSY files.

The validity of these kinship indicators has been evaluated in previous research. First, Rodgers (1996) reported a validity analysis showing that height and weight measures—which are known to be highly heritable in the population—had very similar biometrical structure using these kinship links to those estimated from meta-analyses. Further, a number of published studies have used these links successfully, and some of these demonstrate the discriminant and

convergent validity of these links when compared to other published studies using the same or similar phenotypes. Examples include Rodgers et al.'s (1999) study of age at first intercourse (compare to Miller et al. 1999); Rodgers et al.'s (2001a) study of delinquency (compare to Lyons et al. 1995; Miles and Carey 1997); Neiss et al.'s (2002) multivariate analysis of education, IQ, and age-at-first-birth (compare to Retherford and Sewell, 1989; Tambs et al. 1989); and van den Oord and Rowe's (2000) research on race differences in birth weight, birth length, and gestational length (compare to Kallen 1993; Sappenfield et al. 1987).

Using these kinship links, we focused on female–female kinship pairs. Separate models of fertility must be developed across gender, and our focus in the current study is on female fertility. Further, our analysis required longitudinal structure that allowed us to construct measures of fertility up to age 20, from age 20 to 25, from age 25 to age 30, and from age 30 to age 35. After imposing these data restrictions, we had a data base with 1198 individuals defining 599 female–female kinship pairs; these broke down into the following categories: 15 MZ/DZ twins of unknown zygosity (we could identify that these siblings were twins, but did not have information to define their zygosity), 474 full-sibling sister pairs, 10 half-sibling sister pairs, 22 female-cousin pairs, and 78 female pairs who are definitely at least half-siblings, but who are indistinguishable as either full or half siblings. Although these categories are disproportionate, they are approximately representative of the distribution of these same-sex female categories within the population of U.S. households with adolescents in 1979—up to attrition and non-response. In fact, this is a strength of using the NLSY for biometrical research. Few representative datasets with biometrically informative information exist. Most are either based on national twin registries (and therefore do not account for other types of kinship relatedness) or on purposive samples (e.g., twin samples or adoptive samples) with low external validity.

The measures

For each female in our sample, we used their longitudinal responses between 1979 and 2000 to construct four primary measures of fertility: Number of biological children born by age 20, number of biological children born after their 20th birthday and up until their 25th birthday, number of biological children born after their 25th birthday and up until their 30th birthday, and number of biological children born after their 30th birthday and up until their 35th birthday. We will refer to these measures as F20, F25, F30, and F35, respectively, and verbally refer to these age periods as “early fertility,” “early-mid fertility,” “middle fertility,” and “later-mid fertility,” respectively. Because all respondents in the sample were at least age 35, there was no right truncation on this variable (a common problem with previous NLSY analyses). A large majority of childbearing was accounted for by age 35; however, future research (when this sample has aged a bit more) can account for childbearing patterns at older ages (past age 35) as well.

We note that these particular cut points were defined at least in part because of the convenience of defining and interpreting age categories in five-year increments. There is both policy and theoretical relevance to the definition of the first age category, because of general interest in teenaged childbearing. In addition, however, these five-year increments are the age categories used in standard demographic analysis and record-keeping. Further, we investigated other cut points empirically (e.g., we reran the primary analyses in this paper using only three age categories—before age 20, age 20–30, and after age 30—and also using other cut point specifications as well). Our basic interpretations did not change across these different specifications of age-ranges, and so we used the standard demographic categories reflecting 5-year age categories in our analysis.

These measures were consistently skewed positively, as expected. In each category, the modal response was zero children, with the upper limit at four children. Table 1 presents demographic

distributions of race, fertility at ages 20, 25, 30, and 35 years, and the dispersion among the five classifications of the 599 kinship pairs assessed. The mean number of children born to the NLSY respondents within each of the 5-year age categories were $M_{F20} = 0.20$, $M_{F25} = 0.48$, $M_{F30} = 0.59$, and $M_{F35} = 0.48$. The skewness is obvious in these measures in Table 1, and the skewness coefficients support the visual appearance.

Analyses

SAS version 8.1 was used to create text files of the NLSY fertility information, converting the data into Mx readable rectangular raw data files. Analyses were performed within Mx using the raw data option, full information maximum likelihood (FIML) estimation, and ordinal thresholds estimated in relation to underlying normal distributions (which assume ordinal cut-points that demarcate phenotypic normally distributed childbearing patterns; variances of these distributions were arbitrarily constrained to values proportional to observed variances in order to identify the models). As discussed, the distributions of the four fertility phenotypes exhibited moderate to high positive skew, due to modal responses of zero at each age and left-truncation at zero. Details of the ordinal estimation procedure can be found in Neale et al. (2003) and applications of its use in Mx include Kuhnert and Do (2003) and Mehta et al. (2004). Advantages of the technique include unbiased estimation of parameters given missing data that conform to the Missing at Random (MAR) or Missing Completely at Random (MCAR) mechanisms (see Little and Rubin 2002) and the capability to include quantitatively measured moderator variables.

Mx is also capable of handling multi-group indicators that potentially convey moderating influences. This aspect of Mx allows for the estimation of group differences in covariance structure and, therefore, provides an effective tool for analyzing genetically informative data. In our case, group covariance structures were inspected for differences across our five kinship pair classifications. Quantitative genetic theory (e.g. Falconer, 1979) provided the necessary theoretical average kinship correlations for each group (0.125 among cousins, 0.25 among half-siblings, 0.375 among ambiguous half- or full-siblings, 0.50 among full-siblings, and 0.75 among twins of ambiguous zygosity; note that two of these are the average across the two unknown kinship categories). Global heritabilities of fertility outcomes across time were estimated by constraining latent additive genetic influences on each kinship's phenotype to correlate at these averages. Similarly, given the known household sampling structure of the NLSY (i.e., all related kin in these analyses lived in the same household environments for at least part of the time they were growing up, including the cousin pairs), common environmental influences on phenotypic outcomes were estimated by constraining correlations among latent shared environment sources of variability to be one. Finally, these genetically informative models estimated two non-shared latent influences (in one combined category) that account for both measurement error in the phenotype reports and non-shared environmental sources of variability. For greater detail on basic fundamentals and formulas underlying genetic variance-component estimation using structural equation modeling see Neale and Cardon (1992).

As a preliminary analysis, we fit standard univariate biometrical models to each of the four fertility measures, F20, F25, F30, and F35. In Fig. 1, we present a graphical portrayal of a standard ACE biometrical model, in which A represents the additive genetic source of variance, C represents the common environmental source of variance, and E represents a combination of the unique environmental variance and variance due to measurement error. The covariance term between the two kin's C term is fixed at unity, assuming a common environment producing perfectly correlated shared environments. (The cousin pairs in our analysis may not be as likely to meet this assumption as the other pairs; however, this is a relatively small sample size, there was no empirical difference between results with and without cousins, and it is important to remember that these particular cousins lived together in the same household in

1979, and in most cases probably much longer than that. We will empirically evaluate the equal environments assumption in our empirical analyses.) The covariance between the two kin's A term reflected the expected genetic correlation between the two members of the kinship pair.

Because the F20, F25, F30, and F35 measures were time-ordered, we fit multivariate Cholesky Models to this time series. This model is a standard ACE biometrical model, with multivariate time-ordered links defined across the age-related fertility measures. In Fig. 2a, we show the genetic part of this Cholesky Model, for one member of the kinship pair only; in Fig. 2b, we show the common environmental part. We note, however, that we describe analyses and present results separately for genetic and environmental components for interpretational convenience; the estimation routine fit the multiple kinship levels and both members of each kinship pair simultaneously. Syntax for fitting generic Cholesky Models in Mx can be found in Neale et al. (2003). Syntax for models presented below is available from the authors.

There are major advantages and minor disadvantages of fitting a multivariate ordinal model, and using the individual-level data option and FIML estimation in Mx. The first, and most obvious, advantage is that the multivariate structure provides a richer and potentially more complex account of the genetic and environmental processes within the model than the separate univariate models. Second, and related, whereas the univariate model accounts for relationships (correlations, covariances) within kinship pairs, the multivariate model accounts for these as well as the relationships between the variables of interest. Third, more of the information in the data is accounted for; applying this multivariate approach and the individual data option allows all of the available data to be used in the analysis, and the ordinal option accounts for the measurement level. The major disadvantage of this modeling method is that it is highly computer-intensive (e.g., estimating these models often took several days of continuous runs even on high-speed desktop computers). Further, there are methodological and statistical issues that have not been completely worked out in using these models. These will be discussed in more detail shortly.

The full-information feature of our estimation method—the cause of the computational intensity—is a strong advantage. The fit of the model is optimized to the individual data values, rather than to covariances or correlations, which allows more of the data to be used. As a result, however, there do not exist the usual kinship correlations that form the basis of many other simpler biometrical fitting routines. We present in the Appendix a set of kinship correlations that are estimated from the individual data; these are the equivalent of polychoric correlations, estimated from the FIML fitting routine. However, we emphasize that the model results we present did not fit these particular correlations (nor their equivalent covariances), but rather were fit to the individual level data points using FIML methodology.

Another disadvantage of using the FIML/ordinal fitting routine described above is that the specification of saturated models and the use of appropriate fit statistics are two issues that are still in development. At the time of this writing, we are unaware of any discussion of distributional features of fit statistics in FIML estimation, their relationship to well-known sampling distributions (e.g., chi-square distributions), or a general strategy for defining fit statistics when analyzing multi-group, variance decomposition models. Previous FIML use in situations similar to ours has apparently raised the same concerns in terms of proper fit reporting. Posthuma et al. (2000), for example, conducted multivariate behavior genetic Cholesky decompositions in a similar fashion to those we propose. They reported changes in the $-2 \times \text{Log Likelihood}$ and degrees of freedom from the saturated decomposition model and a saturated multivariate normality model (where all covariances and variances are estimated freely), but were obviously careful not to call this a chi-square distribution or report a *P*-value. We agree with Posthuma et al. (2000) that the information obtained from such a Log-Likelihood-difference statistic provides valuable information about the fit of a model, but we

also share their concern with reporting a parametrically derived *P*-value for our observed differences. We use their approach to reporting fit statistics to help guide our own.

Predictions

Using Udry's (1996) theory as a prediction tool, and the tenets of life history theory discussed above, we make several predictions for both the univariate and multivariate models. Udry's theory considers the amount of individual autonomy—individual choice—that occurs in fertility decision-making. If there is considerable fertility choice, he theorized that biologically/genetically based differences in fertility and fertility motivation could emerge. Otherwise, environmental influences acting as social norms could effectively suppress these motivational differences. (For example, a societal norm against teenage sexuality and childbearing strong enough to influence behavior would limit the expression of genetically based differences among teenagers in their sexual and fertility motivations.) We note that these environmental influences are not necessarily ones that would be detected as shared or non-shared environmental variance, because social norms can suppress variance that would otherwise emerge from within the family or non-family environment that would make individuals different from one another.

The current study's design rests on the hypothesis that fertility choices—and the social norms that constrain them—can change across age. We posit that the greatest amount of choice over fertility may occur in the early-mid fertility and middle fertility reproductive categories (i.e., during the age periods defining the 20's). In contrast, both the early (before age 20) and the mid-late/late categories (after age 35) are more strongly affected by cultural and societal norms. For early teenage childbearing—and perhaps even into the early 20's—(adult) norms would discourage childbearing in favor of continuing education, finding a suitable spouse, etc.; for late (say post-age-35, or perhaps even post-age-30) childbearing, norms generally would encourage completing childbearing as quickly as possible.

However, a different type of “fertility choice” is available to the young age group. As they become aware of their potential to reproduce, for the first time they realize an option to make an individual-level choice about sexuality and reproduction. For some adolescents, this choice reflects their knowledge of and interest in achieving adult status (see Rodgers 1998 for discussion of different reasons adolescents give for engaging in sexual relationships). Further, whereas the adult norms may mitigate against teenaged sexuality and childbearing, there is often some peer support for beginning early childbearing (e.g., Geronimus and Korenman 1992). Thus, the prediction from Udry's theory is more difficult for the F20 variable reflecting teenaged fertility patterns—because we can make a plausible argument for either increased choice, or for strong constraints and limitations on choices. As a result, we will let this question be addressed empirically. At the multivariate level, we expect—because of these various dynamics that are distinctive to adolescent sexuality and childbearing—that the genetic influences may be separate for this group compared to childbearing patterns after age 20.

As far as predictions related to the shared environment, we note that for the younger fertility categories, there is still likely to be some influence from the shared family environment—family/parental norms, for example. For the older age categories those shared environmental influences may be substantially reduced, especially as they pass through the opinions of a spouse or other adult friends. Thus, we expect separate and stronger shared-environmental variance for F20—and perhaps also for F25—than for the two older categories.

Finally, life history theory implies tradeoffs between fertility events at different ages. Within a society that effectively limits its fertility—and especially if family sizes are relatively small—early fertility implies the relative absence of later fertility. Lack of early fertility, in favor of education or career advancement, implies a strong mandate for later fertility. Thus, a negative

relation between the genetic and environmental processes underlying early and later fertility is implied.

Thus, in relation to the Cholesky Models, we expect different sources of genetic variance to operate early and later, for the reasons suggested above. Further, we expect whatever shared environmental influences exist to dissipate across time, and to be separate for the early and later categories. Also, we expect a negative relationship between early and later fertility.

Methodological notes

There are two methodological notes that give perspective to the analyses that we present in this paper. First, some time after these analyses were completed, a paper was published in *Behavior Genetics* (Carey 2005) raising doubts about the validity of the chi-square statistic typically used in Cholesky Models. The basic concern is that the sampling distribution of the chi-square test statistic is not in fact distributed as a theoretical chi-square under the null hypothesis. This position is being evaluated and reviewed; in fact, papers in the theoretical statistics literature have raised this concern in the past (e.g., Self and Laing 1987), although this is probably the first time that it has been brought forward within the behavior genetics literature. Thus, we are cautious in treating the difference between our $-2 \times \text{Log Likelihood}$ fit statistic as a theoretical chi-square within this paper. When we do, we do so with the understanding that this method may need to be adjusted in future research. In fact, the legitimacy of using a chi-square as a reference distribution has already been raised in this type of analysis on other grounds (see the discussion in the previous section and Posthuma et al. 2000).

The second methodological comment also has to do with testing models. Although our sample size appears to be moderately large (total $N = 1198$, which implies 599 kinship pairs), in fact even this moderately large sample may result in a somewhat underpowered analysis in the context of the complex multivariate analysis that is the core of this study. Further, because our kinship categories result in an unbalanced design, due to disproportionate sample sizes, this also costs statistical power (note, however, that this cost is against a benefit of increased external validity, since the disproportionate sample sizes approximately reflect the population). For example, if the design were balanced, but with only $N = 30$ in each kinship category, the univariate power to test the coefficient that estimates either c^2 or h^2 in a standard regression model, assuming a population c^2 or h^2 of 0.10 (falling between Cohen's 1998 definition of a small and medium effect size), would be approximately 0.90, providing high power. However, power is substantially different in multivariate ordinal FIML models and in the presence of missing data. We employed a procedure from Neale and Cardon (1992) which uses Mx to estimate multivariate power. There are a number of assumptions that are difficult to resolve without previous research (e.g., the multivariate equivalent of the univariate effect size estimate is an estimated variance–covariance matrix). By referring to estimates obtained in Rodgers et al. (2001c) using Danish twin fertility data to make guesses about those variance–covariance matrices, we ran several such analyses to estimate power for one degree of freedom tests of genetic and environmental coefficients within a Cholesky Model. The highest power estimate we obtained was 0.70, and most were lower. As a result, we do not expect large power to test individual coefficients.

However, Udry's theory and the predictions derived from it do not predict outcomes associated with those one degree-of-freedom tests. Rather, the predictions suggest patterns at the level of the whole model. For example, in Fig. 1 the predictions above suggest that meaningful coefficients will be associated with a subset of the links whereas the other links are expected to have approximately zero coefficients. Because our predictions inform the model, we have substantially more statistical power to evaluate the overall model than to test individual

coefficients themselves. We will discuss the power issue further in the Discussion section in relation to our empirical analyses.

Results

Univariate ACE models

We first fit univariate ACE Models (see Fig. 1) to each of the four age-related fertility variables, F20, F25, F30, and F35. The biometrical parameter estimates for heritability (h^2), common environmental variance (c^2), and unique environment/error variance (e^2) are shown in Table 2. These patterns are suggestive of strong genetic influence for early fertility (F20) and moderate genetic influence for middle fertility (F30). Shared environmental variance was estimated to be moderate for early-mid fertility (F25), and small for early (F20) and late-mid (F35) fertility. The size of the unique environmental/measurement error variance term increased systematically across age.

These results will be given interpretation in relation to the predictions of Udry's theory in the Discussion Section. The reproductive cycle implications of that theory force the automatic question, how do these common sources of variance at different ages overlap across these ages? This is exactly the question that the Cholesky Model addresses.

Multivariate Cholesky ACE models

We fit the overall four-variable Cholesky Model (see Fig. 2a, b). The estimation routine was optimized to the individual data using an ordinal FIML analysis, as described previously. Thus, there are no kinship covariances or correlations that precede the model estimate process (as there usually are in biometrical analyses). To give perspective to this fitting process, though, we present two different sets of correlations. As previously discussed, in the appendix we present the complete estimated set of correlations that are derived from the model-fitting procedure applied to the raw data. We emphasize that these are derived from the model, rather than being input to the model. They do represent, however, FIML-estimates of polychoric correlations (see Mehta et al. 2004), which are commonly used with ordinal or nominal data (like ours). It is very difficult to study these matrices and extract interpretive value from them, both because of their multivariate complexity and also because the kinship categories differ substantially in sample size. The twin correlation matrix, for example, reflects only 15 twin pairs, whereas the full sibling correlation matrix reflects 474 sibling pairs. Thus, the sibling correlations are estimated with much greater precision, are more stable, and contribute much more substantially to the results than do the twin correlations.

To provide some interpretive value, we used the raw data to compute polychoric correlations (using PROC CORR in SAS); these are presented in Table 3. These were estimated by combining the cousins, half siblings, and ambiguous half siblings into one category (called the "low-related group"; total sample size = 220 individuals, 110 kinship pairs), and the full siblings and twins into a second category (called the "high-related group"; total sample size = 978 individuals, 489 kinship pairs). These can be inspected with some value. For example, the low-related group and the high-related group both have moderately large correlations between F20 and F25, but these do not differ very much across kinship levels. This pattern should be reflected in an estimate of a meaningful c^2 factor that relates these two variables (and indeed such a factor will be one of the core components of the model estimation results), but low h^2 . Also notice that the negative correlations between F20 and F35 increase substantially negatively from the lower-related group to the high-related group, which should eventually be a feature of the genetic part of the estimated model (and will in fact show up in this way in the model). Other interpretations can be extracted from Table 3, although we caution that the more-complex correlations in the Appendix are the ones most strongly reflective of the covariance

structure that is being modeled; note, for example, that Table 3 only portrays cross-pair correlations; to observe cross trait-within individual correlations, the Appendix should be consulted.

We fit the overall Cholesky Model, with parameters estimated by Mx to optimize $-2 \times \text{Log Likelihood}$. We tested and produced confidence intervals for each of the estimated coefficients; results are shown in Table 4. Most individual coefficients within this table are not significant, although the overall model fit is good. The overall fit is evaluated by constructing a saturated multivariate normality model (with all covariances and variances in each group estimated freely), which had $-2 \times \text{LL} = 6996.4$ with 4216 degrees of freedom; the full Cholesky Model had a $-2 \times \text{LL} = 7155.0$ with 4408 df's. The difference is 158.2 with 192 df's; though there may be concerns over using a theoretical chi-square as a reference, this comparison strongly supports the full model as a more parsimonious improvement over the saturated model, because the difference in the fit statistics is much smaller than the df difference.

To find a more parsimonious improvement on the full Cholesky Model, we chose to retain only those coefficients that accounted for over 3% of the variance as defined by the partial regression coefficients (measured by squaring the standardized coefficients corresponding to the unstandardized coefficients presented in Table 4). When the resulting model was refit using the FIML routine, there were two additional paths which accounted for less than 3% of the variance. We then re-fit the model with these paths dropped; in the resulting model, all of the standardized coefficients were greater than 0.03; we refer to the resulting sub-model as the "preferred" or "reduced" model.

We compared the fit of this reduced model to the overall Cholesky Model in Table 4 (which itself was a statistical improvement on the saturated model); the reduced model had a $-2 \times \text{LL} = 7162.8$ with 4422 df's, a difference compared to the full model of 7.8 against a df difference of 14; this comparison supports the reduced model as an improved model compared to the full model. It has a very large *P*-value in relation to a chi-square reference distribution (note that the mean of a theoretical Chi-square equals its degrees of freedom, suggesting in this case a *P*-value larger than 0.89). The unstandardized coefficients and 95% confidence intervals from the resulting model are presented in Table 5, and the standardized coefficients are presented in Fig. 3a and b. Each of the coefficients within the model is either significant ($P < 0.05$) or marginally significant ($P < 0.10$).

We evaluated many other reduced models, and tested the fit of individual coefficients in these models. Each such evaluation was a substantial model fitting effort, requiring several days of computer time to estimate each model and perform statistical evaluations. Across all of these model-fitting exercises, the reduced model in Fig. 3 was consistently the preferred model.

Although the model was fit to each of the ACE components simultaneously, we will discuss each of the sources of variance separately. First, the genetic part of the preferred reduced model is shown in Fig. 3a. This model contains two genetic factors, one that accounts for common genetic variance shared between early fertility (F20) and middle fertility (F30)/late-mid fertility (F35); and another that accounts for (separate) shared variance between our two middle fertility periods (F25 and F30). Three of the five coefficients were positive; those that were not were for the first A factor's link to F30 and F35. We will offer interpretations of these factors and coefficients in the Discussion Section. The common environmental part of the model is shown in Fig. 3b. It suggests a single shared common environmental factor, accounting for shared variance in F20, F25, and F35. The coefficients for the first two were positive, and for the last was negative. Again, we will interpret this pattern in the Discussion Section.

There are other ways to define the boundaries between our age-related fertility measures than the one we used above. To investigate whether our findings were partly an artifact of our age

cut-points, we present several comments and an additional analysis. First, we fit the models above using the ordinal option in Mx, which provided empirical boundary cut-points (assuming an underlying normal distribution) that were estimated from the model. These cut points are of some substantive interest themselves, and are presented in Table 6. For each fertility variable, they are interpreted as the standard normal deviates corresponding to the proportion of the normal distribution underlying childbearing associated with having one, two, three children, and four or more children. (The smallest standard normal deviates are still larger than zero, the mean and median in the standard normal, because there are so many females with zero children in a given time period.) To further address this concern empirically, we also re-defined our fertility variables, and re-ran the modeling exercise as described above. First, we defined three (instead of four) fertility variables, the same F20, a variable that combined F25 and F30 from the earlier analysis, and a third that was identical to F35. Results were very similar using these re-defined fertility variables, and led to the same interpretations. Other similar revisions did as well.

In previous work using these kinship links, the cousin and/or half-sibling categories have occasionally exerted undue influence on the results, because their structure was not consistent with either genetic or environmental models. As another test of the assumptions of our model, we deleted individual kinship categories corresponding to half siblings and to cousins, and re-ran the analyses. Results and interpretations were stable across these deletions. For example, in the reduced model from Fig. 3a and b, the coefficient linking the first genetic factor to F20 in Fig. 3a changed from 0.21 to 0.13 when half-siblings were deleted, and from 0.21 to 0.13 when both full siblings and cousins were deleted. The coefficient linking the first shared environmental factor to F20 and F25 in Fig. 3b changed from 0.34 and 0.34, respectively, to 0.39 and 0.30 when half siblings were deleted, and from 0.34 and 0.34, respectively, to 0.40 and 0.30 when half siblings and cousins were deleted. Similar slight shifts occurred in the full model with these deletions as well. The conclusion from this sensitivity analysis is that it does not appear that either of these two lowest-related kinship categories are having an undue influence on the results.

Discussion

Our best-fitting Cholesky sub-model (Table 5 and Fig. 3a, b) identified two genetic factors and one shared environmental factor. These conceptually link the individual differences in fertility across ages among our sample of U.S. females.

Before we discuss our results in relation to the predictions that have emerged from consideration of both Udry's theory and life history theory, we interpret the negative coefficients in Fig. 3a and b. These may emerge from a fundamental feature of modern society. When family size can be (approximately) chosen by using effective contraception, those who realize their family size desires early can choose not to have more children; and many who do not realize their family size desires early (e.g., though conscious decision-making in relation to education, careers, etc.), can have children at later ages. Further, as previously described, we expect genetic processes to function differently at the different ages, and we expect the shared environmental processes to wane as the individual passes through these age periods. Taken together, these considerations lead us to expect this type of negative relationship between processes causing early childbearing and those contributing to later childbearing; these processes should either be separate, reflected in separate factors, or should be negatively correlated, reflecting a potential form of inverse causation; our models identified both.

The different genetic factors may emerge from a slightly different interpretation. Early sexuality and childbearing may have a risk-taking component that is not a part of the same process at later ages. For example, Rodgers and Rowe (1991) found that adolescent sexual

behavior shared variance with other adolescent risk-taking behaviors, but had some separate variance as well: “sexuality and mild deviance domains overlap, but they also can be distinguished empirically” (p. 291). Moffitt (1993) proposed a theory distinguishing the typical “adolescent limited” adolescent risk-taker from the more unusual “life-course persistent” deviant, which can help explain why different genetic and environmental sources of variance appear to underlie early and later childbearing. Combined, these findings suggest that risk-taking—of which sexual risk-taking is one component—is a different process for teenagers and adults. Thus, we interpret the two different genetic factors to arise from potentially different bases of sexual and reproductive behavior during adolescence and in adulthood.

The positive links in Fig. 3a and b are, presumably, those directly related to fertility decision-making. First, we note a general pattern of agreement between our predictions—derived from Udry’s theory and life history theory—and the results. As predicted, there appear to be different sources of genetic variance for early and later childbearing. We were not able to clearly predict whether there was fertility-choice for early childbearing or not; these results suggest that there is choice, but that it derives from a different biological source than for later childbearing (as noted above). Further, the prediction that shared environmental influences would be stronger for early fertility than for later fertility matched the results; in fact, it completely disappeared for fertility past age 25 (except for the negative coefficient previously discussed).

We note that, despite having a somewhat underpowered study (underpowered, at least, to test statistical hypotheses associated with individual coefficients in our multivariate model), the most preferred overall model was quite consistent with our predictions. Further, at a higher scientific level, the model makes a simple, coherent, and theoretically defensible statement about the genetic and environmental processes that underlie the pattern of relationships across these different age periods within the life course.

These findings suggest that there are three primary age-related patterns of influence on fertility. Before age 20, both genetic and shared environmental influences are substantial. The shared environmental variance overlaps into the early-mid fertility period (age 20–25), but the early-fertility genetic influence did not overlap into that category. We interpret the shared environmental variance as reflecting the effect of the nuclear family on values and behaviors, initially strong but mostly disappearing by the mid-twenties. We interpret the early-fertility genetic variance as reflecting the effect of genes on an adolescents’ ability to regulate their own sexual and reproductive behaviors. For fertility after age 20 there is a new and shared source of genetic influence leading to differences in fertility outcomes. We interpret this as the basic “mature” genetic motivation for children. Miller and Rodgers (2001) presented a framework in which the neural/genetic basis for nurturant bonding plays a central role in this type of motivation. The second factor in Fig. 3a is suggestive of individual differences in this source of fertility behavior. It applies, further, during the early-mid and middle fertility periods, the predicted periods of greatest choice and fertility flexibility. Those in this period have mature—and relatively unconstrained by society—fertility options, but do not yet have concerns over the upper biological limits of female fertility.

In considering the mechanisms that might bring about these patterns, we find the literature on gene–environment interactions to provide considerable guidance (see, e.g., Rowe 2002; Rutter and Silberg 2002). But we enter this discussion with some broader reflections, and will return to genetic interactions in a moment. First, we posit that biological influences on human fertility are always there, in some latent form, with both canalized genetic properties that support having children despite genetic variability, as well as phenotypic plasticity that allows childbearing in relation to many different individual desires and environmental contingencies. Some of these biological influences are, of course, not directly related to fertility desires themselves. Obviously, selection for sexual activity, for example, leads directly (even if not necessarily

consciously) to fertility (e.g., Potts 1997; Turke 1989). Potts and Turke argued that a desire for copulation was sufficient, and that a desire to conceive has no particularly important role in understanding human fertility. But in contrast to these arguments, we have found past evidence of genetic influence on fertility desires and motivation (e.g., Rodgers and Doughty 2000; Pasta and Miller 2000). Even more focused on this point, Rodgers et al. (2001b) demonstrated overlapping genetic variance between a measure of fertility motivation and achieved fertility using a multivariate design with Danish twin data. Thus, based on these past findings, we believe there are genetically based individual differences in motivation for human fertility.

However, we assume that much of the genetic basis for wanting children—i.e., to begin childbearing, or to have at least one child—may lie in the part of the genome that does not contribute to individual differences. This is where canalization may come in. Similar to many morphological features (e.g., having one nose, two arms, and feet located at the end of our legs, etc.), a desire to bear children may lie (almost) universally genetically encoded (e.g., Miller and Rodgers 2001). An important point is to note that such a genetic structure can never disappear. Motivation for childbearing operates directly on fitness, and by definition, any change (e.g., through mutation) will be maladaptive.

But the modeling in the current study is in relation to individual differences, that is, to variability in childbearing patterns. In other words, we can speculate about canalization and its role in reproduction, but phenotypic plasticity is the process that has potentially influenced our empirical findings. From where do these individual differences emerge? The obvious source would appear to be gene–environment and gene–gene interactions. How might these function? As an example of a gene–gene interaction, given genes for high intelligence, a woman may be more willing to delay expression of her genetic desire for childbearing in order to pursue higher educational goals. Further, consistent with the tenets of life history theory, such a woman may have fitness-based mechanisms to effectively trade off the quantity against the quality of her children (e.g., she may be smart enough to help them avoid accidents or disease, and thus support them to live to adulthood and to reproduce successfully).

As an example of a gene–environment interaction, both theory and empirical evidence suggest that stress in the family environment inhabited by young children may trigger genetic responses—which may in turn trigger precocious sexual and nurturant behavior (e.g., Repetti et al. 2002). As Miller and Pasta (2000, p. 216) stated, “In early family environments, the relative absence of affection is a signal to the developing child to be alert to alternative sources of affection. One good way to achieve such a source is to mature earlier.... Turning on sexuality, however, also necessitates preparing the child for the largely inevitable consequence of childbearing by also turning on nurturance.” Kohler and Rodgers (2003) showed empirically how education can be a mediating influence on heritability, which supports the gene–environment interaction interpretation. To summarize, there are obvious interactions both between genes and other genes and between genes and environments that can help explain the individual differences in fertility reflected in the behavioral genetic variances of our models.

In relation to Udry’s (1996) theory, such gene–gene or gene–environment interactions may only occur in settings in which the “other side”—the other genetic, or other environmental influence—has the potential to be expressed. No matter how much intelligence a woman has, if educational opportunities are limited then the choice to postpone childbearing to pursue education may be irrelevant. Or, if most family environments in a certain culture did not exhibit the type of stress that would begin the process leading to precocious sexuality, then individual differences in fertility related to these processes would not emerge—even though their potential was there, reflected in (unexpressed) genetic variability. Thus, we propose that individual differences in fertility are often reflections of individual differences in other processes that

interact with fertility, including education, employment, intelligence, stress in the family environment, etc.

In relation to life history theory, our findings support a very simple and cogent theoretical statement. The complexities of human reproduction lead to a nuanced explanatory model that operates differently at different ages. It is no surprise that genetic and shared environmental processes are not fixed across ages; it would be a surprise if they were. At a basic level, though, the processes are not all that complex. That a simple model to explain those patterns has emerged, and that it is consistent with biosocial thinking about how these processes should happen, provides leverage for future research, both empirical and theoretical.

In light of our empirical findings, it makes sense that the relevant genetic or environmental processes that result in individual differences among females in motivation for childbearing, and in fertility outcomes, would be different for those younger than age 20, and for those older than 20. A small amount of previous research has been done to identify and model such processes; in addition to the Kohler and Rodgers (2003) work that considered the interaction between education and fertility, Neiss et al. (2002) developed models of the interaction between education, income, and age-at-first-childbearing. Further development of such gene–gene and gene–environment interaction models would appear to show great promise for future research attempting to understand genetic and environmental influences on fertility.

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References

- Carey G. Cholesky problems. *Behav Genet* 2005;35:653–665. [PubMed: 16184491]
- Carroll, SB. *Endless forms most beautiful: the new science of Evo Devo*. Norton; New York: 2005.
- Center for Human Resource Research. *NLY handbook*, 2003. U.S. Department of Labor; Washington: 2003.
- Christensen K, Kohler H-P, Gasso O, Olsen J, Vaupel JW, Rodgers JL. The correlation of fecundity among twins: evidence of a genetic effect on fertility? *Epidemiology* 2003;14:60–64. [PubMed: 12500047]
- Doughty, D.; Rodgers, JL. Behavior genetic modeling of menarche in U.S. females. In: Rodgers, JL.; Rowe, D.; Miller, WB., editors. *Genetic influences on fertility and sexuality*. Kluwer Academic Publishers; Boston: 2000.
- Dunne MP, Martin NG, Statham DJ, Slutske WS, Dinwiddie SH, Bucholz KK, Madden PA, Heath AC. Genetic and environmental contributions to variance in age at first sexual intercourse. *Psychol Sci* 1997;8:1–6.
- Falconer, DS. *Introduction to quantitative genetics*. Longman; New York: 1979.
- Figueredo AJ, Vasquez G, Brumbach BH, Schneider SMR. The heritability of life history strategy: The K-factor, covitality, and personality. *Soc Biol*. In press
- Fisher, RA. *The genetical theory of natural selection*. Oxford; Clarendon: 1930.
- Flatt T. The evolutionary genetics of canalization. *Q Rev Biol* 2005;80:287–295. [PubMed: 16250465]
- Geary DC. Sexual selection and human life history. *Adv Child Dev Behav* 2002;30:41–101. [PubMed: 12402672]
- Geronimus AT. Teenage childbearing and social and reproductive disadvantage: the evolution of complex questions and the demise of simple answers. *Fam Relat* 1991;40:463–471.
- Geronimus AT, Korenman RS. The socioeconomic consequences of teen childbearing reconsidered. *Q J Econ* 1992;107:1187–1214.

- Houle D. Comparing evolvability and variability of quantitative traits. *Genetics* 1992;130:195–204. [PubMed: 1732160]
- Hughes, KA.; Burleson, MH. Evolutionary causes and consequences of variation in fertility and other fitness traits. In: Rodgers, JL.; Rowe, DC.; Miller, WB., editors. *Genetic influences on human fertility and sexuality*. Kluwer; Boston: 2000. p. 7-33.
- Johnson AE, Rodgers JL. The impact of having children on the lives of women: the effects of children questionnaire. *J Appl Soc Psychol* 2006;36:2685–2714.
- Kallan JE. Race, intervening variables, and two components of low birth weight. *Demography* 1993;30:489–506. [PubMed: 8405611]
- Kirk KM, Blomberg SP, Duffy DL, Heath AC, Owens IPF, Martin NG. Natural selection and quantitative genetics of life history traits in western women: a twin study. *Evolution* 2000;55:423–435. [PubMed: 11308097]
- Kohler, H-P.; Christensen, K. Genetic influences on fertility behavior: findings from a Danish twin study. In: Rodgers, JL.; Rowe, DC.; Miller, WB., editors. *Genetic influences on human fertility and sexuality*. Kluwer; Boston: 2000. p. 67-84.
- Kohler, H-P.; Rodgers, JL. Education, fertility, and heritability: explaining a paradox. In: Wachter, KW.; Bulatao, RA., editors. *Offspring: human fertility behavior in biodemographic perspective*. Washington, DC; National Academies Press: 2003.
- Kohler H-P, Rodgers JL, Christensen K. Is fertility behavior in our genes: findings from a Danish twin study. *Popul Dev Rev* 1999;25:253–288.
- Kohler H-P, Rodgers JL, Christensen K. Between nurture and nature: the shifting determinants of female fertility in Danish twin cohorts. *Soc Biol* 2002;49:218–248. [PubMed: 14652919]
- Kuhnert P, Do K-A. Fitting genetic models to twin data with binary and ordinal outcomes: a simulation study to compare structural equation modeling and Bayesian hierarchical models. *Behav Genet* 2003;33:441–452. [PubMed: 14574143]
- Little, RJ.; Rubin, DB. *Statistical analysis with missing data*. Wiley; New York: 2002.
- Lyons MJ, True WR, Eisen SA, Goldberg J, Meyer JM, Faraone S, Eaves L, Tsuang MT. Differential heritability of adult and juvenile antisocial traits. *Arch Genet Psychiatry* 1995;52:906–915.
- Mealey L, Segal NL. Heritable and environmental variables affect reproduction-related behaviors, but not ultimate reproductive success. *Pers Individ Dif* 1993;14:783–794.
- Mehta PD, Neale MC, Flay BR. Squeezing interval change from ordinal panel data: latent growth curves with ordinal outcomes. *Psychol Meth* 2004;9:301–333.
- Miles DR, Carey G. Genetic and environmental architecture on human aggression. *J Pers Soc Psychol* 1997;72:207–217. [PubMed: 9008382]
- Miller, WB.; Pasta, DJ. Early family environment, reproductive strategy, and contraceptive behavior: testing a genetic hypothesis. In: Rodgers, JL.; Rowe, DC.; Miller, WB., editors. *Genetic influences on human fertility and sexuality*. Kluwer Academic Press; Boston: 2000. p. 183-230.
- Miller WB, Pasta DJ, MacMurray J, Chiu C, Wu H, Comings DE. Dopamine receptors are associated with ge at first sexual intercourse. *J Biosoc Sci* 1999;31:43–54. [PubMed: 10081236]
- Miller, WB.; Pasta, DJ.; MacMurray, J.; Muhleman, D.; Comings, DE. Genetic influences on childbearing motivation: further testing a theoretical framework. In: Rodgers, JL.; Rowe, DC.; Miller, WB., editors. *Genetic influences on human fertility and sexuality*. Kluwer Academic Press; Boston: 2000. p. 35-66.
- Miller, WB.; Rodgers, JL. The ontogeny of human bonding systems: evolutionary origins, neural bases, and psychological manifestations. Kluwer; Boston: 2001.
- Moffitt TE. Adolescent-limited and life-course-persistent antisocial behavior: a developmental taxonomy. *Psychol Rev* 1993;100:674–701. [PubMed: 8255953]
- Neale, MC.; Boker, SM.; Xie, G.; Maes, HH. *Mx: Statistical modeling*. Vol. 6. Department of Psychiatry, VCU; Richmond, VA: 2003.
- Neale, MC.; Cardon, LR. *Methodology for genetic studies of twins and families*. Kluwer Academic Publishers; Dordrecht, L: 1992.
- Neiss M, Rowe DC, Rodgers JL. Does education mediate the relationship between IQ and age of first birth? A behavioural genetic analysis. *J Biosoc Sci* 2002;34:259–275. [PubMed: 11926458]

- Pasta, DJ.; Miller, WB. A heritability study of childbearing motivation. In: Rodgers, JL.; Rowe, DC.; Miller, WB., editors. Genetic influences on human fertility and sexuality. Kluwer Academic Press; Boston: 2000. p. 107-120.
- Posthuma D, de Geus EJC, Neale MC, Hulshoff Pol HE, Baare WEC, Kahn RS, Boomsma D. Multivariate genetic analysis of brain structure in an extended twin design. *Behav Genet* 2000;30:311–318. [PubMed: 11206086]
- Potts M. Sex and the birth rate. *Popul Dev Rev* 1997;23:1–40.
- Repetti RL, Taylor SE, Seeman TE. Risky families: family social environments and the mental and physical health of offspring. *Psychol Bull* 2002;128:330–366. [PubMed: 11931522]
- Retherford RD, Sewell WH. How intelligence affects fertility. *Intelligence* 1989;13:169–185.
- Rodgers JL. NLSY-Youth linking algorithm. 1996Unpublished document
- Rodgers, JL. The development of sexuality in adolescence. In: Friedman, SB.; Fisher, M.; Schonberg, SK.; Alderman, EM., editors. Comprehensive adolescent health care. Vol. 2. Quality Medical Publishing, Inc.; St. Louis: 1998.
- Rodgers JL, Buster M, Rowe DC. Genetic and environmental influences on delinquency: DF analysis of NLSY kinship data. *J Quant Criminol* 2001a;17:145–168.
- Rodgers, JL.; Doughty, D. Genetic and environmental influences on fertility expectations and outcomes using NLSY kinship data. In: Rodgers, JL.; Rowe, D.; Miller, WB., editors. Genetic influences on fertility and sexuality. Kluwer Academic Publishers; Boston: 2000.
- Rodgers JL, Hughes K, Kohler H-P, Christensen K, Doughty D, Rowe D, Miller WB. Genetic influence helps explain variation in fertility outcomes: a review of the recent behavioral and molecular genetic literature. *Curr Dir Psychol Sci* 2001b;10:184–188.
- Rodgers JL, Kohler H-P, Kyvik K, Christensen K. Genes affect human fertility via fertility motivation: findings from a contemporary Danish twin study. *Demography* 2001c;38:29–42. [PubMed: 11227843]
- Rodgers JL, Rowe DC. Adolescent sexual activity and mildly deviant behavior: Sibling and friendship effects. *J Fam Issues* 1991;11:274–293.
- Rodgers JL, Rowe DC, Buster M. Nature, nurture, and first sexual intercourse: Fitting behavioural genetic models to NLSY kinship data. *J Biosoc Sci* 1999;31:29–41. [PubMed: 10081235]
- Rowe, DC. Environmental and genetic influence on pubertal development: evolutionary life history traits?. In: Rodgers, JL.; Rowe, D.; Miller, WB., editors. Genetic influences on fertility and sexuality. Kluwer Academic Publishers; Boston: 2000.
- Rowe, DC. Assessing genotype–environment interactions and correlations in the postgenomic era. In: Plomin, R.; DeFries, JC.; Craig, IW.; McGuffin, P., editors. Behavioral genetics in the postgenomic era. APA; Washington DC: 2002. p. 71-86.
- Rutter, M.; Silberg, J. Gene–environment interplay in relation to emotional and behavioral disturbance. In: Fiske, ST.; Schacter, DL.; Zahn-Waxler, C., editors. Annual Review of Psychology. Annual Reviews; Palo Alto, CA: 2002.
- Sappenfield WM, Buehler JW, Binkin NJ, Hogue CJR, Strauss LT, Smith JC. Differences in neonatal and postnatal mortality by race, birth weight, and gestational age. *N Engl J Med* 1987;326:1522–1526.
- Self SG, Liang K-Y. Asymptotic properties of maximum likelihood estimators and likelihood ratio tests under nonstandard conditions. *J Am Stat Assoc* 1987;82:605–610.
- Tambs K, Sundet JM, Magnus P, Berg K. Genetic and environmental contributions to the covariance between occupational status, educational attainment, and IQ: A study of twins. *Behav Genet* 1989;19:505–522.
- Trumbetta, SL.; Gottesman, II. Endophenotypes for marital status in the NAS-NRC twin registry. In: Rodgers, JL.; Rowe, D.; Miller, WB., editors. Genetic influences on fertility and sexuality. Kluwer Academic Publishers; Boston: 2000.
- Turke PW. Evolution and the demand for children. *Popul Dev Rev* 1989;15:61–90.
- Udry, JR. Biosocial models of low-fertility societies. In: Casterline, JB.; Lee, RD.; Foote, KA., editors. Fertility in the United States: new patterns, new theories. The Population Council; New York: 1996.
- Van de Kaa DJ. Europe's second demographic transition. *Popul Bull* 1987;42:1–57. [PubMed: 12268395]

- van den Oord EJCG, Rowe DC. Racial differences in birth health risk: a quantitative genetic approach. *Demography* 2000;37:285–298. [PubMed: 10953804]
- Vigil JM, Geary DC, Byrd-Craven J. A life history assessment of early childhood sexual abuse in women. *Dev Psychol* 2005;41:553–561. [PubMed: 15910162]
- West-Eberhard, MJ. *Developmental plasticity and evolution*. Oxford University Press; 2003.
- Williams LA, Williams BJ. A re-examination of the heritability of fertility in the British peerage. *Soc Biol* 1974;21:225–231. [PubMed: 4450115]

Fig. 1.
A Univariate ACE model

Fig. 2.

(a) A four-variable multivariate Cholesky Model, Additive Genetic (A) component, for one member of a kinship pair, (b) A four-variable multivariate Cholesky Model, Shared Environmental (C) component, for one member of a kinship pair

Fig. 3.

(a) Best-fitting Cholesky multivariate model, additive genetic (A) part of the ACE model; the coefficients are standardized coefficients (corresponding to the unstandardized a 's in Table 6); to get h^2 and c^2 values, these coefficients would have to be squared; all individual coefficients are significant ($P < 0.05$) or marginally significant ($P < 0.10$). (b) Best-fitting Cholesky multivariate model, shared environmental (C) part of the ACE model; the coefficients are standardized coefficients (corresponding to the unstandardized c 's in Table 6); to get c^2 values, these coefficients would have to be squared; all individual coefficients are significant ($P < .05$)

Table 1
Demographic information, NLSY females with kinship linking information

<i>Race</i>									
White	824								
Black	294								
Other	71								
Missing	9								
<i>Kinship pairs</i>									
Cousins	22								
Half-siblings	10								
Ambiguous HS/FS	78								
Full siblings	474								
Twins	15								
<i>Fertility distributions</i>									
	0	1	2	3	4	Missing	Mean	SD	Skewness*
F20	998	167	24	8	0	1	0.20	0.49	2.80
F25	775	301	100	18	1	4	.48	0.72	1.49
F30	624	352	116	22	1	83	0.59	0.76	1.17
F35	610	258	74	8	1	247	0.46	0.68	1.43

* Calculated as the ratio: $\frac{\sum (x_i - \bar{x})^3}{\sigma^3}$

Table 2Univariate ACE Model components (and *P*-values)

	h^2	c^2	e^2
F20	0.65 (0.08)	0.09 (0.59)	0.26 (0.09)
F25	0.00 (1.0)	0.35 (<0.01)	0.65 (<0.01)
F30	0.22 (0.52)	0.01 (1.0)	0.77 (<0.01)
F35	0.00 (1.0)	0.08 (0.55)	0.92 (<0.01)

Note: F20 is fertility (number of births) up to age 20; F25 is fertility from age 20 up to age 25; F30 is fertility from age 25 up to age 30; F35 is fertility from age 30 up to age 35

Table 3
Polychoric kinship correlations indicative of the multivariate structure across F20, F25, F30, and F35 (sample sizes of non-missing data in parentheses)

	F20_2	F25_2	F30_2	F35_2
<i>Combined cousins, ambiguous half-siblings, and half-siblings</i>				
F20_1		0.29 (217)	−0.10 (191)	−0.13 (172)
F25_1			0.08 (191)	−0.24 (172)
F30_1				0.08 (169)
<i>Combined full-siblings and twins</i>				
F20_1		0.27 (977)	0.03 (923)	−0.22 (718)
F25_1			0.10 (922)	−0.11 (772)
F30_1				0.10 (774)

Table 4

Multivariate Cholesky ACE Model components and 95% confidence intervals (CI), full model

	Unstdized a_1 , CI	Unstdized a_2 , CI	Unstdized a_3 , CI	Unstdized a_4 , CI
F20	0.53 [-2.01, 4.85]			
F25	-0.06 [-3.52, 3.52]	0.32 [-0.06, 3.41]		
F30	-0.33 [-3.51, 3.54]	0.90 [-3.68, 3.68]	0.26 [-2.98, 3.34]	
F35	-0.45 [3.25, 3.26]	0.43 [-2.76, 2.76]	0.13 [-2.72, 2.72]	0.00 [-2.16, 2.16]
	Unstdized c_1 , CI	Unstdized c_2 , CI	Unstdized c_3 , CI	Unstdized c_4 , CI
F20	0.59 [.05, 4.13]			
F25	1.07 [.22, 4.06]	-0.01 [-2.52, 2.52]		
F30	0.17 [-1.01, 1.66]	-0.01 [-2.43, 2.43]	0.00 [-2.20, 2.20]	
F35	-0.44 [-2.28, -0.01]	0.00 [-.41, 1.76]	0.00 [-1.75, 1.76]	0.00 [-1.43, 1.50]

Notes: F20 is fertility (number of births) up to age 20; F25 is fertility from age 20 up to age 25; F30 is fertility from age 25 up to age 30; F35 is fertility from age 30 up to age 35; Unstdized a 's and Unstdized c 's are the estimated unstandardized coefficients associated with the full Cholesky Model

None of the a coefficients are significant in the full model; the c_1 coefficients are significant for F20, F25, and F35 (i.e., note that the CI's do not contain 0)

Table 5

Multivariate Cholesky ACE Model components and 95% confidence intervals (CI), reduced most-preferred model

	Unstdized a_1 , CI	Unstdized a_2 , CI	Unstdized a_3 , CI	Unstdized a_4 , CI
F20	0.48** [0.01, 1.26]			
F25	0.00 [-]	0.30* [-0.00, 3.18]		
F30	-0.45** [-3.23, -0.02]	0.69 [-3.33, 3.33]	0.17 [-2.97, 2.97]	
F35	-0.32* [-3.18, 0.00]	0.14 [-1.71, 1.95]	0.00 [-]	0.00 [-]
	Unstdized c_1 , CI	Unstdized c_2 , CI	Unstdized c_3 , CI	Unstdized c_4 , CI
F20	0.69** [0.03, 0.86]			
F25	0.92** [0.04, 3.99]	0.00 [-]		
F30	0.00 [-]	0.00 [-]	0.00 [-]	
F35	-0.48** [-2.19, -0.38]	0.00 [-]	0.00 [-]	0.00 [-]

Notes: F20 is fertility (number of births) up to age 20; F25 is fertility from age 20 up to age 25; F30 is fertility from age 25 up to age 30; F35 is fertility from age 30 up to age 35; Unstdized a 's and Unstdized c 's are the estimated unstandardized coefficients associated with the reduced Cholesky Model

** indicates significant coefficients (i.e., the 95% CI does not contain 0)

* indicates marginal significance (i.e., the 90% CI does not contain 0)

Table 6

Multivariate Cholesky ACE reduced model, threshold estimates

	Expected thresholds [*]			
	1 Child	2Child	3Child	4Child
F20	0.96	1.90	2.42	3.74
F25	0.39	1.28	2.11	2.54
F30	0.15	1.15	2.03	2.33
F35	0.36	1.34	2.29	3.04

* Thresholds were constrained to be the same across all kinship groups. Thresholds were also constrained to be the same for each sibling measurement (e.g., Thresholds for Kin 1 F20 = Thresholds Kin 2 F20)

FIML-estimated polychoric correlations, estimated by the unrestricted saturated multivariate normality model, for five kinship categories

[illegible]

[illegible]

Note: The order of the variables is F20_Kin1, F25Kin1, F30Kin1, F20_Kin2, F25Kin2, F30Kin2, F35Kin2