Modeling NLSY Fertility Patterns Longitudinally and Biometrically: Evolutionary, Genetic, and Social Interpretations

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Note: This outline form was extracted from a PowerPoint presentation.

Fisher's Fundamental Theorem of Natural Selection (FTNS)

- The FTNS: Fitness traits and behaviors strongly affected by natural selection will "lose" their genetic variance in the long run -- thus, fertility and fertility precursors should have little genetic variance, and thus zero heritability, if natural selection is the only process at work
- BUT IT'S NOT!!!!!
- The FTNS was mis-interpreted by some to suggest that, by definition, for fertility
  h2 = 0 -- But it doesn't!
- Hughes & Burleson (2000) suggested a number of different processes that re-introduce genetic variance into fitness traits, even while natural selection is washing it out
  - Mutation (most important)
  - Frequency-dependent selection
  - Heterozygote advantage (overdominance)
  - Sexual antagonism
  - Environmental Perturbations
- Is fertility heritable? It's an empirical question.

Is Human Fertility Heritable?

Univariate studies:

- Fisher (1930) -- sample of British aristocracy -- (possibly biased) --h2 = .40
- Mealey & Segal (1993) – Minn.
  study of twins raised apart -- h2 = .06
- Kohler & Christensen (2000) –
  Danish twin sample -- male h2 = .39 female h2 = .11
- Rodgers & Doughty (2000) –
  NLSY (US) family data -- median h2 = .33

Is Human Fertility Heritable? Time Series and Multivariate studies:

- Kohler, Rodgers, & Christensen (1999) -- time series of almost 100 years of data from Danish Twin registry -- heritabilities rose from around zero to moderate levels for cohorts born during the 1880's and the 1950's (during fertility transition)
- Kirk et al (2001) -- MV analysis of Australian twin data showed genetic correlation between a measure of fitness and both age at first reproduction and age at menopause
- Rodgers, Kohler, Kyvik & Christensen (2001) -- MV biometrical analysis of Danish twins showed genetic variance overlapped between age-at-first-pregnancy-attempt and fertility outcomes
- Conclusion from literature review: It's no longer useful to ask whether there is genetic variance in human fertility -- the answer is clearly, "Yes, often -- but not always."
- New questions: When and why?
- We need more nuanced treatment of this question, using sophisticated modeling, theoretical insights, and new data sources

The Udry (1995) Theory

- Udry suggested, on conceptual grounds (and without supporting data) that biologically-based variance can only emerge in cultural settings where there is substantial choice over fertility outcomes -- e.g., in modern contracepting cultures, but not in natural-fertility societies
- Supported by Kohler et al (1999) time-series results, in which fertility heritability rose during fertility transition, when fertility control emerged and fertility choices became more available

Method -- the Data

- The (US) National Longitudinal Survey of Youth (NLSY) contains a sample of over 12,000 individuals who were 14-21 years old at the beginning of 1979. This was a household probability sample, many households of which contained twins, full siblings, half-siblings, and even cousins
- The NLSY Youth have been followed every year or two since 1979 -- we have the year 2000 data available, in which respondents were 35-43
- The linking algorithm: Sibling status is not directly addressed, but by using information about the biological fathers of each child, we can reliably assign sibling status to around 80-90% of the sibling pairs
- Validity analyses and more than a dozen studies using these links support their legitimacy
- Result: A population-based sample of hundreds of kinship pairs who lived together in the same household in 1979, with approximately representative kinship distribution of twins, full-sibs, half-sibs, and cousins

- We restricted this study to females, by only considering female-female kinship pairs
- Sample Sizes of female-female pairs:
  - 15 MZ/DZ twins of unknown zygocity (R=.75)
  - 474 Full siblings (R=.50)
  - 10 Indeterminant, either full or half sibs (R=.375)
  - 78 Half siblings (R=.25)
  - 22 Cousins (R=.125)

Method -- the Measures

- Using fertility histories, we constructed the following four measures for each female in the NLSY:
  - Number of children born by age 20 (F20)
  - Number of children born between 20 and 25 (F25)
  - Number of children born between 25 and 30 (F30)
  - Number of children born between 30 and 35 (F35)
- These measures were positively skewed

Method – Analyses

- We fit biometrical Cholesky models to the covariances between these measures using the raw-data option in Mx
- The fertility variables were highly skewed, which made the regular maximum likelihood estimation routine in Mx problematic -- instead, we implemented the ordinal option in Mx, which estimated both biometrical parameters and also thresholds on top of a quantitative "number of children" continuum (assumed normal)
- These models were used to identify the unique and overlapping genetic, shared, and nonshared environmental variance between these four different fertility periods -- early fertility (before age 20), early middle fertility (age 20-25), middle fertility (age 25-30), and late middle fertility (age 30-35)

- We ran a univariate ACE model on each of the four fertility measures separately:

Basic ACE Univariate Model

	h2	c2	e2
F20	**.65**	.09	.26
F25	.00	**.35**	.65
F30	**.25**	.00	.75
F35	.00	.09	.91

- If we stopped here – as typical of past research – we'd just say genes affect early (pre age 20) fertility, and age 25-30 fertility, while the shared environment affects age 20-25 fertility
- And neither genes nor shared environment affect 30-35 fertility
- But with MV analysis we can model the overlapping genetic and environmental sources of variance

Advantages of MV Analysis

- Conceptual advantages are obvious
  - Richer modeling structure
  - Relations *between* variables
- Methodological advantages as well
  - Covariance structure is more complete with additional variables (i.e., more data are used)
  - Missing at random assumption allows us to "fill in" missing data

The Complete Cholesky Model -- Genetic Component (A), for one member of a kinship pair

- Results suggest two separate genetic factors
  - Early fertility shares genetic variance with late middle fertility – this genetic influence contributes positively to fertility before age 20, inhibits fertility between 30 and 35
  - Middle fertility (age 25-30) shares genetic variance in a positive direction with late middle fertility (age 30-35)

The Cholesky Sub-Model with Significant Links Included -- Shared Environmental Component (C)

- Results suggest one shared environmental factor
  - Shared between three of the four fertility periods
  - In particular, shared between early and early middle fertility periods
  - This shared environmental influence contributes positively to the first two time periods, inhibits fertility in the last time period

Summary

  - There are two genetic factors, one operating early (before age 20), the other operating later (after age 25)
  - There is one shared environmental factor, operating early
  - The negative loadings for both the early genetic and shared environment factor may be partially artifactual – what contributes to having children early, will automatically inhibit having children later, especially in low-fertility societies

Implications

- Udry's theory
  - We had a difficult time making a priori predictions from Udry's theory about early fertility
    - Is there fertility choice among very young childbearers?
    - Or is there limited choice among very young childbearers?
  - If genetic variance is an indicator of fertility choice, as Udry suggests, then greatest choice is before age 20 and between 25-30

- But note that there's also a lot of shared environmental influence before age 20, as well
- Perhaps we need to revise Udry's theory

A Tentative Theory

- Our results suggest different mechanisms underlie the two early periods (up to age 25) and the two later periods (ages 25-35) (note that explication of fertility patterns after age 35 will have to wait for future NLSY research in a few years)
- Our elaboration/revision of Udry's theory:
  - Biological influences on fertility are always there, in some latent form
  - 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 doesn't contribute to individual differences – i.e., like many morphological features (e.g., having one nose, two arms, and feet located at the end of our legs, etc.), a desire to start childbearing may lie (almost) universally genetically encoded (e.g., Miller & Rodgers, 2001)
  - Important Point: Once there, this genetic structure can never disappear. By definition, any change (e.g., through mutation) will by definition be mal-adaptive.
  - But this part of the theory is not about individual differences, and that's what we've been modeling
  - Where do the individual differences come from? Gene-gene interactions, and gene-environment interactions
  - Example of gene-gene interactions: Given genes that lead to high intelligence, a person still wants to start childbearing, but may be willing to wait because education is so stimulating (or, alternatively, intelligence gives them knowledge that allows them to wait)
  - Example of gene-environment interactions: a woman reaching pubertal maturity in a mate-rich environment can more easily respond to the childbearing mandate than one who's environment has been robbed of mates by, for example, war
  - This is where Udry's theory begins to apply – Individual differences caused by gene-gene or gene-environment interactions are only relevant in settings in which reproductive choice is relevant, and can be realized
  - Our revision: Modeling genetic or environmental variance underlying fertility is actually reflecting individual differences in these other sources with which fertility interacts
  - Postponing childbearing may not be mal-adaptive for some, if quality ultimately results in increased fitness – with quality leveraged through these types of interactions
  - How did this process get started? Perhaps through some type of frequency-dependent selection, supporting a k-selected reproductive strategy

Final Conclusions

- If this type of process is going on, where would we expect the genetic variance reflecting individual differences to occur?

In early childbearing, where those gene-gene and gene-environment interactions are first realized, just where we found them
And the environmental influences are there as well

- These genetic influences would logically be different types of biological/genetic processes than those influencing later childbearing, as we found