

The genetics of human fertility

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## Abstract

Heritable variation in fitness—survival and reproduction—is the fuel of evolution by natural selection. Many human societies have dramatically reduced mortality before and during the prime reproductive years, making fertility a reasonably good proxy for the whole of fitness in much of our species. For this reason, empirical knowledge regarding the genetics of fertility must be an essential part of any framework for understanding past and ongoing trends in human adaptive evolution. Here we use R. A. Fisher’s analysis of human fertility as a starting point and find strong support from more recent research for his main contentions: fertility is a moderately heritable trait, where much of the genetic influences are shared with psychological characteristics.

All else being equal, heritable variation in fitness leads to future generations being more like the most reproductively successful members of previous generations. Ronald A. Fisher formalized this notion in the Fundamental Theorem of Natural Selection (FTNS): ‘the rate of increase in fitness of any organism at any time is equal to its genetic variance in fitness at that time’ [1]. A common misinterpretation of the FTNS, however, has occasionally hindered research on human fertility and quantitative traits more generally. It is worthwhile at the outset to set aside this confusion, hopefully for good.

A mistaken interpretation of the FTNS is that fitness-increasing alleles will necessarily and rapidly reach fixation in the population, thus eventually resulting in a heritability of zero (i.e., no genetic variation). In particular, to the extent that fertility is a good proxy for overall fitness, it should have a near-zero heritability. What the FTNS actually says is that the rate at which fitness increases, due solely to the effect of natural selection on allele frequencies, is equal to the population’s genetic variance in fitness [2–4]. It would probably take us too far afield to explicate why this statement of the FTNS does not in fact imply a low heritability of fitness. For our purposes it suffices to point out that Fisher’s own remarks on human fertility in the very same book presenting the FTNS were not at all consistent with its interpretation as a prediction of near-zero heritability. He did not argue for a negligible heritability of fertility; in fact, he estimated the heritability of human fertility to be about 0.40 on the basis of ancestor-descendant correlations. This estimate is reasonably close to those obtained in subsequent twin studies—from 0.24 to 0.43 for women, from 0.24 to 0.28 for men [5]. The DNA-based GREML method gives an estimate of 0.10 [6], showing that not all of the heritability of fertility is due to rare variants of evolutionarily recent origin.

Fisher went beyond the estimation of heritability to provide an intriguing account of the mechanisms by which genetic variation affects fertility in humans. He emphasized the

exceptionality of humans in post-forager societies, among whom differences in fertility might be more ascribable to differences in psychology (‘mental and moral qualities’) than to reproductive anatomy and physiology. This emphasis has proven to be quite prescient [7]. A recent GWAS of fertility has found a near-unity genetic correlation between male and female fertility [8]; this result is unintuitive if individual differences act chiefly through anatomy and physiology, in light of the physical differences between men and women, but becomes reasonable if the mediating traits are behavioral in nature. Indeed, this review will cover many behavioral correlates of fertility documented in recent studies. GWAS can now pinpoint the specific regions of the human genome harboring variation affecting fertility, and the first ‘hits’ strongly support the genetic overlap of fertility with behavior in our species; many of the polymorphic sites associated with age at first birth (an important component of overall fertility) are also strongly associated with years of education [9].

An increasing role for the psychological component of fertility since Fisher’s own day may be driving a remarkable trend in modern industrialized societies: people are having children later in life and fewer children overall [10]. In many countries this trend has reached the point of subreplacement fertility—i.e., a number of births insufficient to maintain the native-born population at its current size. This is certainly a very curious phenomenon from the standpoint of evolutionary biology. A possible explanation is that a relaxation of traditional expectations with respect to family structure have enabled the greater expression of psychological differences affecting fertility preference [11–13]. It will be useful to keep this overarching hypothesis in mind as we review the particular traits appearing to mediate the heritability of human fertility.

Our review is encapsulated in Table 1. In order for a correlate of fertility to have an evolutionary impact on the species, the phenotypic correlation must have some genetic basis, and in Table 1 we indicate whether we have been able to find evidence in the

literature for the presence of a genetic correlation. Some readers may take the term ‘fertility’ to mean the biological capacity or potential to have children (i.e., fecundity), but here we follow the demographic definition of fertility as the actual number of biological children produced over the entire lifespan.

## **Socioeconomic traits**

The most well-established correlate of fertility may be years of education, particularly in women. The prolonging of education has at least some causal effect on later age at first reproduction [14–16], which inevitably depresses total fertility in turn [5, 8]. Since the mid-twentieth century, many countries have experienced a massive rise in the proportion of the population awarded a college degree, particularly among women. Women postpone having their first child in order to pursue higher education, along with other career-enhancing opportunities [17]. This is a convincing example of a social or cultural change leading to individuals, perhaps in particular those with certain genetic dispositions, altering their reproductive behavior. Years of education and fertility do share a genetic basis. Polygenic scores for education successfully predict fertility [18], and significant genetic correlations in the expected directions have been found between years of education and various measures of fertility. The genetic correlation between years of education and age at first birth is particularly high ( $\sim 0.7$ ) [8].

It has recently been shown that this negative correlation between fertility and education is changing the genetic composition of Western populations [19, 20]. For example, the average polygenic score for education is falling over time in Iceland, as a result of more highly educated individuals having fewer children.

Most studies find intelligence to be negatively correlated with fertility [21–23], and some of these studies suggest that this is not entirely because more intelligent individuals

seek more education [24]. Raw IQ scores, however, have not declined over most of this time period but rather have increased [25]. As we have already said, educational credentials have proliferated as well. This apparent masking of genetic decline by environmental improvements illustrates the complexity of evolutionary change in our peculiar species. Nevertheless we think the authors of one relevant study justified in pointing out that ‘[i]t is remarkable to report changes in [the education polygenic score] across the several decades covered by this study. In evolutionary time, this is a blink of an eye. However, if this trend persists over many centuries, the impact could be profound’ [20, p. E730].

Some studies have found higher income to be associated with decreased fertility [26]. Others have questioned the general applicability of this finding, particularly for high-earning men [14]. The relationship may be U-shaped, with the highest- and lowest-income individuals having higher fertility. Further research is necessary. If it is true that the overall correlation between income and fertility is negative, the sign of this correlation may have undergone a reversal since the medieval and early modern period [27]. Antisocial behavior now shows a positive genetic correlation with fertility [28], and the sign of this correlation may also have undergone a reversal [29].

## **Personality and psychopathology**

We will not review the literature on personality and fertility, as this has been recently done in this journal [30]. It is worth repeating the finding of this review that studies of the personality-fertility associations are sometimes inconsistent, almost certainly as a result of low statistical power. There have been few studies of whether the observed correlations have a genetic basis [7, 8, 31], but with the growing availability of personality GWAS data there will certainly be more.

The evidence for a relationship between fertility and certain mental illnesses is more

secure. Individuals diagnosed with schizophrenia and autism spectrum disorder, particularly males, show dramatically reduced fertility [32]. One oddity is that whereas individuals diagnosed with bipolar disorder or unipolar depression show baseline or slightly reduced fertility, the genetic correlations of these two disorders with fertility are both positive [8, 33]. A possibility worthy of further investigation is that increasing liability to bipolar disorder increases fertility for some reason, until the point where further liability produces the behavioral problems leading to a diagnosis of the disorder. Consistent with this hypothesis, siblings of individuals affected by these disorders do show somewhat elevated fertility [32].

## Reproductive behavior

Intended fertility unsurprisingly affects actual fertility; those who want to have more children, tend to do so [34, 35].

The proportion of children born to unmarried parents has dramatically increased in many countries [10]. Interestingly, in times and places where marriage does predict fertility, one of the mediating pathways through which physical attractiveness leads to higher fertility appears to be a greater probability of marriage [36, 37]. Married women who are more attractive may have higher fertility even after conditioning on duration of marriage, which is consistent with female attractiveness providing cues to additional indicators of fecundity beyond age [38]. Physical attractiveness is heritable [39], but a genetic correlation with fertility has not been demonstrated to our knowledge.

Age at first sexual intercourse shows a very strong positive genetic correlation with age at first reproduction and hence a negative genetic correlation with overall fertility (i.e., those who start having sex earlier tend to end up with more children) [8, 40]. In part, an early age at first sexual intercourse may reflect a fast life history [41], consistent with

small and positive genetic correlations with age at menarche and age at voice breaking [40].

## Religion and politics

People with more conservative or traditional beliefs beget more children [42–45]. These findings combine in an interesting way with the relationships between fertility and reproductive behavior described above; social conservatism is associated with earlier and more stable marriage (a positive correlate of fertility), but also with later age at first sexual intercourse and fewer sexual partners over the lifespan (negative correlates of fertility) [46]. Overall, there is a suggestion of two different reproductive strategies proving to be successful in modern Western societies: (1) a strategy associated with socially conservative values, including a high commitment to the bearing of children within marriage; and (2) a strategy associated with antisocial behavior, early sexual experimentation, a variety of sexual partners, low educational attainment, low commitment to marriage, haphazard pregnancies, and indifference to politics. This notion of distinct lifestyles characterized in common by relatively high fertility deserves further empirical and theoretical study.

## Conclusion

R. A. Fisher was a pioneer in the study of human fertility, as in so many other fields. Using the quantitative-genetic principles that he had himself developed, he found that fertility is a moderately heritable trait. He went on to posit that much of this heritability overlaps with genetic influences on behavior. Recent research has amply confirmed these hypotheses and early findings.

Potential future directions are numerous. Table 1 suggests the importance of confirming a genetic contribution to the correlation between fertility and more traditional

Table 1: Traits that may mediate genetic influences on fertility in humans

Correlate of fertility	Sign of correlation	Genetic correlation	References
<b>Socioeconomic traits</b>			
Years of education	–	Yes	[8, 14, 18–20]
Intelligence	–	Yes	[21–24]
Income	–	No	[14, 26]
Antisocial behavior	+	Yes	[28]
<b>Psychopathology</b>			
Autism spectrum disorder	–	Yes	[8, 32]
Schizophrenia	–	No	[8, 32]
Bipolar disorder	+	Yes	[8, 32]
Depression	+	Yes	[18, 32, 33]
<b>Reproductive behavior</b>			
Intended fertility	+	No	[34, 35]
Physical attractiveness	+	No	[36–38]
Age at first sex	–	Yes	[8, 40]
<b>Religion and politics</b>			
Religiosity	+	No	[42, 43, 45]
Conservatism	+	No	[44, 45]

‘Genetic correlation,’ whether we have been able to find a study documenting a significant genetic correlation between the two traits. Some of the relationships with fertility may be nonlinear; see the text for details.

religious and political views. One would have liked an opportunity to ask Fisher for his thoughts about the psychological basis of this relationship. Fisher himself was a conservative, an English patriot, a professed Christian, and a father of nine, and it is natural to think that a mind so penetrating would have gleaned some insight if trained introspectively. GWAS of fertility should continue to increase the sample size, and one promising application of the resulting data might be testing the ‘Fisher-Muller hypothesis’ and its variants for the advantage of sexual reproduction itself [1, 47], in a manner analogous to recent studies of mostly model organisms [48, 49].

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