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Heritability and Anthropometric Influences on Human Fertility

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ABSTRACT This study researched the impact of anthropometrics and size-of-family of orientation on women's fertility by using path analysis. The data were collected as part of the anthropological study conducted in Ireland by Harvard University personnel before the Second World War. The women included in this analysis were all over age 49 and were either married or widowed at the time of the survey. Our results indicate that the heritability of fertility is moderate in this sample and that there is a tendency for heavy women to have a higher fertility. However, when anthropometrics and size-of-family of orientation were entered as independent variables in a path diagram, an insignificant portion of the variation of fertility was explained. In this Irish population, the main cause of differential fertility was cultural rather than biological. A large portion of women never married and no unmarried woman reported producing a child. Am. J. Hum. Biol. 15:16–22, 2003.

Human fertility and its many facets has been a topic of interest to human biologists and biological anthropologists. Given its holistic nature, human biology focuses on how cultural behaviors such as mating patterns, length of postpartum taboos, ritual celebrations, and environmental cyclical changes affect fertility. Indeed, there are a number of volumes and chapters written within the discipline of human biology concerning human fertility (Mealey, 2000; Rosetta and Mascie-Taylor, 1996; Ellison, 2001; Ellison and O'Rourke, 2000; Wood, 1994).

From an evolutionary perspective, biological anthropologists may study fertility asking what phenotypic traits may result in higher or lower fertility in a specific environment. For example, does a female's menarcheal age or do her anthropometric measures affect her fertility and thus her fitness? And if they do, is it because these traits have a genetic basis and they predispose the female to higher fertility, because they reflect biological plasticity in specific environments, or because they reflect differential socioeconomic status? These are not easy questions to answer. Although some of these phenotypes might have a genetic component, they are influenced by the environment. Indeed, the secular trends of both menarcheal age and weight and stature have been well established as human responses to environmental changes (Stinson, 2000).

During the 1970s and 1980s, a large number of research articles examined the association between anthropometric measurements and fertility. The assumption of many researchers was that if natural selection operates through differential fertility of women with different anthropometric traits, human populations might be evolving towards specific modal anthropometrics. Whereas some authors suggested that males (Mitton, 1975) or both genders (Muller, 1979) of intermediate height have higher fertility, others observed a higher fertility of subjects with specific head shapes (Lasker and Thomas, 1976, 1978) or with a specific infradental–menton index (a dental measure which is acknowledged to be notoriously difficult to measure accurately) (Mueller et al., 1981). Other studies have indicated that the tallest women have the highest fertility (Liljestrand et al., 1985; Martorell et al., 1981), whereas others (Scott and Bajema, 1982) report a significant association between high fertility and the body mass index (BMI). Tague (2000) notes that big females have big pelves, which could impact their fertility. Finally, other researchers found that in specific environments shorter women have higher fertility (Devi et al., 1985; Frisancho et al., 1977, 1973; Stini, 1969, 1972).

These studies were important because they demonstrated the considerable varia-

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tion in the possible link between anthropometrics and fertility. At the same time, several articles suffered from having a large Type-I statistical error because numerous null hypotheses were proposed (i.e., fertility was proposed to be correlated with a large number of anthropometric measures) and, as expected by chance, a few were rejected (Muller et al., 1981; Lasker and Thomas, 1978). Moreover, it is not justified to assume that the only explanation for the higher fertility of a specific phenotypic group is natural selection, instead of, for example, higher socioeconomic status of taller and heavier women.

Another approach to the study of fertility from an evolutionary perspective has been to research the heritability of fertility within the framework of Fisher's fundamental theorem. The theorem postulates that the mean rate of evolutionary change in mean fitness equals the additive genetic variance in fitness itself (Fisher, 1930). As Falconer (1985) explains, if a population is in equilibrium the heritability of fitness approaches 0. This results from the fact that there should be no genetic variation in fitness-related traits because the alleles conferring highest fitness should be driven quickly to fixation by natural selection. Thus, most genetic variability of fitness of populations in equilibrium is due to epistatic interactions and dominance. Fisher's fundamental theorem, however, has been the topic of a large number of articles which have attempted to validate it, to reinterpret it, or even to explain how Fisher has been misunderstood.

For example, Gustafsson (1986) examined the heritability of lifetime reproductive success of the flycatcher and reported empirical support for Fisher's theorem. Other researchers, however, indicated that if life-history traits have low heritability, this could be due to the fact that these traits (such as fertility or survivorship) are "one step further down the causal pathway from genes to phenotype," allowing for other random factors to come into play at each step (Price and Schulter, 1991). In a similar vein, Merilä and Sheldon (2000) suggest that the low heritability of fitness traits does not reflect depleted additive genetic variance, as predicted by Fisher's fundamental theorem, but reflects the disproportionately large residual variance (variance due to environmental and nonadditive genetic sources) of these traits. A similar point is presented by Houle (1992). Frank and Slatkin (1992) opine that the traditional interpretation of Fisher's theorem is incorrect, since it refers to total evolutionary change. They argue that Fisher partitioned the total change into change due to natural selection and change caused by deterioration of the environment. Given that the second term is often negative, although natural selection increases fitness, the total change in fitness is usually close to zero. Frank and Slatkin (1992) mention that when Fisher's fundamental theorem is correctly interpreted, it has proven to be disappointing, for example, to Price (1972) and Ewens (1989). For a through review of Fisher's fundamental theorem, see Lessard (1997).

Our purpose here is certainly not to review the various interpretations of Fisher's fundamental theorem, but rather to demonstrate that if the heritability of fertility or fitness-associated traits is low, it might not be only due to the traditional interpretation of Fisher's work (Falconer, 1985), but could be due to other reasons as proposed by Price and Schulter (1991) and Merilä and Sheldon (2000). Moreover, we want to point out that the traditional interpretation of Fisher's work has been called into question (Ewens, 1989; Frank and Slatkin, 1992; Lessard, 1997; Price, 1972). Be that as it may, Murphy (1999) points out, in an extensive review of the literature, that the relationship between fertility of human parents and offspring is weak, although it has increased in the last decades. Gagnon and Heyer (2001) also show that there is virtually a null impact of parents' fertility on children's fertility in a Canadian population.

The purpose of this article is to evaluate the influence of anthropometrics and size-of-family of orientation on women's fertility. We compute the heritability of fertility, and then investigate if body morphology influences fertility. We then incorporate both anthropometry and size of family of orientation by using path analysis. Path analysis is ideally suited for the construction and evaluation of models about the relationship among variables. An appealing feature of this method is that it allows one to consider correlated independent variables and that it allows the computation of U, a residual variable that contains all unknown factors which influence the dependent variable. Thus, in this article we look at the influence of phenotype and sibship size on a woman's

fitness first separately and then conjointly in path analysis.

MATERIALS AND METHODS

Dataset

The data used in this analysis constitute a subset of the data originally collected by E.A. Hooton, C. Wesley Dupertuis, and H. Dawson as part of an anthropological study of Ireland conducted by Harvard University in the 1930s. Between 1934 and 1936, Dupertuis collected anthropometric and demographic data on almost 9,000 adult males throughout the entire island of Ireland and Dawson collected similar data on almost 2,000 adult females in the western part of Ireland (Hooton et al., 1955). As used here, the term "Ireland" refers to the entire island, which today is made up of two nations, the Republic of Ireland and Northern Ireland. Michael Crawford obtained the original data cards from Peabody Museum in the 1970s and computerized a portion of the total sample. The remainder of the sample was computerized in the 1990s (see Relethford and Crawford, 1995, for further details on data collection). Although a large number of variables were collected (including religion, county of residency, anthroposcopic and anthropometric traits), the variables analyzed here are the subject's number of children, their number of siblings, and the following anthropometric measures: weight (pounds), stature (mm), acromion height (mm), dactylion height (mm), arm span (mm), biacromial breadth (mm), and sitting height (mm). An advantage of using this dataset is that we can be fairly confident of the fact that no Western contraception was employed by the Irish population. Indeed, the medical director of the Irish Family Planning Association (IFPA) informed us that no form of contraception was available in Ireland before the Second World War (S. Jones, pers. commun.).

Methodology

All the analyses presented in this article are based on a data subset which consists of women over 49 years of age and who were married or widowed. The restriction on marital status is due to the fact that no single woman is reported to have had children. The restriction of age allows us to measure completed fertility. These restrictions, however, severely limit our sample size: the number of females over 49 is 361 and, of those, 293 were married or widowed. Some anthropometric variables were not recorded in a few subjects, thus decreasing the sample size analyzed in this study to 290.

The data collected by Dawson clearly indicate that Irish women got married at a rather late age: none of the subjects in the 15–19 age group were married and 86% of those in the 20–34 age group were still single (Kennedy, 1973). Indeed, 33% and 20% of women in the 35–54 and 55+ age groups, respectively, were single. It is unfortunate that we do not have the specific age at marriage of each subject, but only summary statistics. Thus, we cannot enter the duration of marriage and age at marriage as independent variables in our analysis.

Our methodology attempts to avoid two errors frequently committed in studies on the influence of anthropometry on fertility: first, the probability of a Type-I error was frequently inflated as a result of the computation of too many tests without appropriate statistical correction. Second, the variable "number of children" is not normally distributed, but was analyzed with parametric tests. The first shortcoming can be solved by considering only one anthropometric variable or few multivariate indexes, as independent variables impacting fertility. Here we chose to use body mass index (BMI), computed as

$$BMI = \frac{weight(kg)}{height(m)^2}$$

(Frisancho, 1999).

We also performed a principal component analysis of the anthropometric variables listed above. The PC analysis was done after the data were standardized using the **R** matrix. We follow Chatfield and Collin's (1980) strong suggestion that PC analysis should be favored over factor analysis, and do not proceed further than the computation and analysis of the principal components. Weight was recorded in pounds, thus necessitating a transformation into kilograms.

The second problem has been either overlooked or has been circumvented by dividing females according to fertility and comparing their anthropometrics, which tend to be normally distributed. A better approach is to keep fertility as the dependent variable and use a nonparametric ANOVA to compare

the fertility of groups of women defined by their anthropometric measures. This is the approach we take here, by dividing women according to the quantile in which they fall: Group 1 contains women who fall at least in the 75th percentile, Group 2 the women between the 50th and the 75th percentiles, Group 3 the women between the 25th and 50th percentiles, and Group 4 the women under the 25th percentiles.

The size-of-family of orientation was computed by adding 1 to the subject's number of siblings; this variable is referred to as sibs+1. Heritability was computed as indicated by Falconer (1985) by regressing the females' fertility on sibs+1 and multiplying the regression coefficient by 2. The standard error is simply the standard error of the regression coefficient multiplied by 2 (Falconer, 1985).

Path analysis was performed by standardizing the variables and regressing completed fertility on the independent variables. Before the path diagram was constructed the correlation between the independent variables was computed to determine if it needed to be incorporated in the model (Sokal and Rohlf, 1981).

RESULTS

A Shapiro–Wilk normality test of the variables considered here shows that they are not normally distributed (fertility [$W = 0.96$, $P < 0.001$], sibs + 1 [$W = 0.98$, $P < 0.0011$], and BMI [$W = 0.97, P < 0.0002$]). Since repeated attempts to normalize the data failed, all further analysis is performed with nonparametric tests.

The hypothesis that BMI and fertility are not correlated was rejected with a Spearman test. The coefficient indicates that there is a moderate although significant positive correlation between the variables ($r_s = 0.14$, $P = 0.02$, $n = 290$). This correlation can be better understood by examining Table 1, which shows the mean number of children produced by women divided by their BMI quantiles. It is obvious that the heaviest women have the highest fertility ($\bar{Y} = 6.54$ children) and that the slimmest women have the lowest fertility ($\bar{Y} = 5.39$ children). A nonparametric ANOVA did not reject the hypothesis that women divided by BMI quantile have equal fertility (Kruskal-Wallis Test Chi-square approximation = 4.41, df = 3, $P = 0.22$). A Wilcoxon two-sample test comparing the first and fourth group failed to reject the hypothesis as well, yielding a Kruskal-Wallis statistic (Chi-square approximation) of 2.51, with $P = 0.11$. Thus, the significant correlation reflects the fact that the heaviest females have the largest fertility and the slimmest females the lowest fertility. But the mean number of children produced by these two groups is not significantly different.

Before computing the heritability of fertility, we computed the Spearman correlation between sibs+1 and fertility and obtained a significant positive coefficient. This indicates that women from large families of orientation tend to have large families themselves ($r_s = 0.15$, $P = 0.009$, $n = 290$). Indeed, a nonparametric comparison of women who had a family of orientation equal to or greater than the median of 7 sibs+1 with those who had less than the median yields a significant Kruskal-Wallis statistic (Chi-square approximation) of 10.82, with $P = 0.001$.

A number of studies have computed heritability of human fertility through mother–offspring regression. Given that the heritability of a character refers to a particular population under particular conditions, a fair amount of variation among different estimates of human fertility heritability is expected. Indeed, estimates based on mother–offspring regression range from 0.04–0.41%,

TABLE 1. Fertility of women divided by their BMI

Group	n	Mean number of children	Standard deviation
1 = women at least in the 75th percentile	72	6.54	4.1
2 = women between the 50th and 75th percentiles	72	6.50	4.2
3 = women between the 25th and 50th percentile	73	5.53	3.5
4 = women at or below the 25th percentile	72	5.30	3.8

Kruskal-Wallis Test (Chi-Square approximation) = 4.41, df = 3, $P = 0.2204$.
Two sample test (comparing groups 1 and 4. Chi-Square approximation) = 2.51, $P = 0.11$.

TABLE 2. Heritability estimates for human fertility computed from mother–offspring regression; two sources report more than one estimate

Source	h^2
Pearson et al., 1899	0.0836
	0.4192
Huestis and Maxwell, 1932	0.24
Berent, 1953	0.38
Kanter and Potter, 1954	0.22
Duncan et al., 1965	0.22
	0.04
Imaizumi et al., 1970	0.12
Schull et al., 1970	0.04
Neel and Schull, 1972	0.18
This study	0.34

shown in Table 2. The heritability computed in this study is one of the highest ones so far computed: 0.34. This estimate is certainly higher than the heritabilities of any trait associated with fitness reported by Falconer (1985:150), and is supported by the significant correlation between sibs+1 and fertility already discussed.

Given that sibs+1 and BMI are not significantly correlated ($r_s = 0.02156$, $P = 0.7239$, $n = 271$), the path diagram (Fig. 1) only shows a path from sibs+1 to fertility and another one from BMI to fertility, with no correlation between the independent variables. From the path diagram it is obvious that when the entire variation of completed fertility is considered the contribution of BMI and of sibs+1 is negligible. The R^2 is only 0.02, and the amount of residual variation is 0.98 (Sokal and Rohlf, 1981).

The principal component analysis yielded two PCs which accounted for 75% of the variation of anthropometric measures analyzed. The first PC explains 59% of the variance, has an eigenvalue of 4.13, and is a general measure of size, with high loadings from all variables. The second PC explains 16% of the variance, has an eigenvalue of 1.10, and contrasts weight and biacromial breadth with all the other variables. When these two PCs were entered into the path diagram instead of BMI, the explanatory value of the diagram barely improved. The path coefficients are 0.16 from sibs+1, 0.05 from PC1, and 0.06 from PC2 to fertility. Thus,

$$(U = 0.98 = \sqrt{1 - [(0.16)^2 + (.05)^2 + (.06)^2]}),$$

and the coefficient of determination is only increased to $R^2 = 0.04$. Thus, the multivariate approach to anthropometrics did not improve the initial BMI analysis. We only show the path diagram with BMI because it is a simpler one.

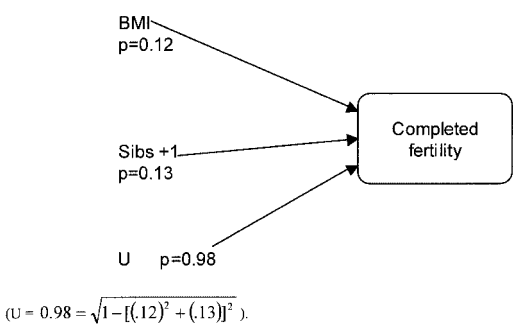

Fig. 1. Path diagram.

DISCUSSION

The effect of a woman's anthropometry on her fertility is not a simple matter to study. Even with a dataset such as this one, whose subjects were not using Western contraception and were all over age 49, we cannot definitively answer the question of whether women are heavier because they had more children or had more children because they were heavier. To answer this question, researchers should follow women's entire reproductive careers and document the subjects' weights and fertility.

The literature on the effect of a woman's anthropometrics on her fertility is extensive and fraught with methodological problems. After a large number of statistical tests were run and a few were significant, several authors concluded that natural selection was acting through differential fertility of women with different phenotypes. Although we do not want to dismiss the possibility of some differential fertility of women with certain phenotypes, we think a more likely explanation for the results of these studies is an environmentally specific distribution of fertility which stems from differential access to resources, as well as the plasticity of the human body during growth and development.

Our data indicate that there is a rather small (although significant) positive correlation between fertility and BMI. However, a

nonparametric ANOVA failed to reject the hypothesis that women divided by their quantiles have significantly different fertility. More importantly, the mean number of children produced by the heaviest and slimmest women is not significantly different. Furthermore, our estimate of h^2 is moderate, and not as low as would be predicted by looking at the estimates reported by Falconer (1985) and others who found support for Fisher's fundamental theorem (Gustaffson, 1986; Murphy, 1999). A consideration of the other estimates shown in Table 2, however, lead us to conclude that human fertility heritability is moderate in other populations, not only the one presented here. Thus, at least in some human populations, there is some detectable genetic variance of fertility.

Our analysis indicates that there is a moderate although significant correlation between family-of-orientation and fertility. However, when both sibs+1 and BMI (or two PCs of anthropometric measures) are entered as independent variables into a path diagram, their contribution to explaining variation of completed fertility is minimal. Although fertility has a moderate correlation with BMI and sibs+1, its variance is not satisfactorily explained by these two variables.

It should also be considered that the number of women who were studied here was a very small minority from those surveyed by Dawson: 70% of the women were single and a large number of them over the age of 49. Dawson and Hooton (Hooton et al., 1955) remark on the large proportion of unmarried females, as well as on the late age of marriage. If differential fertility is to be found in the Irish sample, it is between those who had the opportunity to reproduce (in this sample, that means having been married) and those who did not. Of course, we do acknowledge that some unmarried women might have borne children who were then claimed to be their mothers' offspring. But we can only work with the data as they were collected and all unmarried subjects report a fertility of 0. Although this sample of females was not practicing Western contraception, it did restrict its fertility by having a late age at marriage and by limiting the number of women who could reproduce. In this sample, at least, the main determinant of fertility appears to be of a cultural nature (ability to acquire a mate) and not biological. Our results fit nicely with Williams and Williams' (1974) reexamination of Pearson's early computation of heritability of fertility in the British peerage: that there is an overwhelming contribution of culture to fertility in human populations.

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