Evolution, 55(2), 2001, pp. 423–435

NATURAL SELECTION AND QUANTITATIVE GENETICS OF LIFE-HISTORY TRAITS IN WESTERN WOMEN: A TWIN STUDY

KATHERINE M. KIRK,[1] SIMON P. BLOMBERG,[1,2,3] DAVID L. DUFFY,[1] ANDREW C. HEATH,[4,5] IAN P. F. OWENS,[2,5] AND NICK G. MARTIN[1]

[1]*Genetic Epidemiology Laboratory, Queensland Institute for Medical Research, Brisbane, Queensland 4029, Australia*
[2]*Department of Zoology and Entomology, University of Queensland, Brisbane, Queensland 4072, Australia*
[4]*Department of Psychiatry, Washington University School of Medicine, St. Louis, Missouri 63110*
[5]*Department of Biology, Imperial College at Silwood Park, Ascot, Berkshire SL5 7PY, United Kingdom*

Abstract.—Whether contemporary human populations are still evolving as a result of natural selection has been hotly debated. For natural selection to cause evolutionary change in a trait, variation in the trait must be correlated with fitness and be genetically heritable and there must be no genetic constraints to evolution. These conditions have rarely been tested in human populations. In this study, data from a large twin cohort were used to assess whether selection will cause a change among women in a contemporary Western population for three life-history traits: age at menarche, age at first reproduction, and age at menopause. We control for temporal variation in fecundity (the ''baby boom'' phenomenon) and differences between women in educational background and religious affiliation. University-educated women have 35% lower fitness than those with less than seven years education, and Roman Catholic women have about 20% higher fitness than those of other religions. Although these differences were significant, education and religion only accounted for 2% and 1% of variance in fitness, respectively. Using structural equation modeling, we reveal significant genetic influences for all three life-history traits, with heritability estimates of 0.50, 0.23, and 0.45, respectively. However, strong genetic covariation with reproductive fitness could only be demonstrated for age at first reproduction, with much weaker covariation for age at menopause and no significant covariation for age at menarche. Selection may, therefore, lead to the evolution of earlier age at first reproduction in this population. We also estimate substantial heritable variation in fitness itself, with approximately 39% of the variance attributable to additive genetic effects, the remainder consisting of unique environmental effects and small effects from education and religion. We discuss mechanisms that could be maintaining such a high heritability for fitness. Most likely is that selection is now acting on different traits from which it did in pre-industrial human populations.

Key words.—Fitness, genetic correlation, heritability, humans, life histories, natural selection, twins.

Received March 22, 2000. Accepted September 18, 2000.

Evolution resulting from natural selection is sometimes invoked to explain puzzling aspects of human life history such as delayed reproduction, small litter size, and senescence (e.g., Darwin 1874; Medawar 1952; Williams 1957; Fisher 1958; Hamilton 1966; Short 1976). In most of these cases the inference is that the life history of modern humans has been molded by evolution due to selection that occurred in the past. This much is relatively uncontroversial among evolutionary biologists. What is more contentious is whether human life-history traits are still under selection and, in particular, whether recent changes in human culture have led to new forms of selection that will now lead to changes in the life history of some human populations (Rose et al. 1984; Jones 1993).

To predict whether evolution resulting from natural selection will cause a response in life-history traits, it is necessary (at minimum) to determine: (1) whether there are covariances between fitness and the traits; (2) whether there are significant underlying additive genetic variations in the traits; and (3) the extent and sign of genetic covariances among traits (see Falconer and Mackay 1996; Lynch and Walsh 1998). Previous studies of human life-history traits have tended to focus on one or other of the conditions for evolution via natural selection, rather than tackle all three simultaneously. For instance, although Borgehoff Mulder (1989) was able to demonstrate a correlation between age at menarche and lifetime reproductive success in a study of the Kipsigis, the heritability of the trait could not be tested. Similarly, Käär et al. (1996) used historical records to demonstrate a link between age at first reproduction and fitness in pre-industrial populations in northern Finland, but could not tell whether age at first reproduction was heritable because the pedigree of those populations could not be reconstructed. More recent studies, however, have found significant genetic influence on variation in measures of fertility among Danish twins (Kohler et al. 1999; Kohler and Christensen 2000) and in a U.S. kinship sample (Rodgers and Doughty 2000).

Conversely, there is ample evidence that a range of human morphological traits and congenital diseases are heritable, but there is little evidence that they are associated with differences in fitness. The two most conspicuous exceptions to this pattern are studies of selection on birth weight and sickle-cell anaemia. For both these traits there is good evidence that the trait is both heritable and that it affects fitness (see Bodmer and Cavalli-Sforza 1976). Thus, although both these traits have become textbook examples of natural selection in humans, they are rare exceptions rather than the rule.

The reason that simultaneous estimates of phenotypic fitness correlations, heritabilities, and genetic covariance matrices are so rare for humans is that such estimates require a long-term study of the reproductive and genetic characteristics of a population. This sort of data is very rare for any organism. To overcome this problem, we use data on twins from the Australian Twin Registry. The use of twin data has

[3] Present address: Department of Zoology, University of Wisconsin-Madison, 430 Lincoln Drive, Madison, Wisconsin 53706-1381.

both advantages and disadvantages (Bulmer 1970; Rowe 1994). The main advantage is that, because human twins can be either monozygous (MZ, identical) or dizygous (DZ, fraternal), we can use comparisons between the two types of twins to estimate the extent to which a trait is genetically heritable. In brief, because MZ are genetically identical, whereas DZ twins share, on average, only half their genes, any phenotypic differences between MZ twins must be due to differences in their environment, whereas phenotypic differences between DZ twins may be due to either differences in their environment or differences in their genes. Thus, the proportion of phenotypic variation that is heritable in the broad sense can be estimated as a function of the ratio of variance within pairs of MZ twins versus variance within pairs of DZ twins. This is what is known as the ''classical twin-method'' for estimating heritability (see Eaves et al. 1978; Martin et al. 1978). Narrow-sense heritability, which is what we investigate in this study, can be estimated by the use of maximum-likelihood models (see Neale and Cardon 1992).

This study had three specific aims: (1) to test whether phenotypic differences among women in life-history traits are associated with differences in lifetime reproductive success and fitness (which we measure as the intrinsic rate of increase *r*; see below); (2) to test whether any of the life-history traits correlated with fitness are genetically heritable; and (3) to examine the genetic covariance among life-history traits. To address these aims, we used data on approximately 1200 pairs of female twins to estimate selection gradients, heritabilities, coefficients of additive genetic variance, and genetic correlations for age at menarche, age at first reproduction, and age at menopause. We then used these parameters to predict whether selection will lead to change in the life-history traits of women in the study population. We use structural modeling to take into account the potentially confounding environmental and maternal effects, including temporal variation in the rate of fecundity in the Australian population (most notably the ''baby boom'' period); differences between women in educational background, and differences between women in religious affiliation.

Materials and Methods

Sample

Subjects for this study are female twins aged 45 years and over, who were originally recruited for participation in one or more health and lifestyle studies from the Australian National Health and Medical Research Council Twin Registry (ATR), a volunteer register begun in 1978, which has more than 30,000 twin pairs enrolled and in various stages of active contact. The first health and lifestyle questionnaire study, conducted in 1980–1982, was sent to all available twin pairs aged over 18 at that time (that is, born prior to 1965), and responses from 3808 pairs of twins and 567 singles were received. The second study, conducted in 1988–1990 and focussing on those twins who responded to the original survey, obtained follow-up data from 6327 individuals. A further questionnaire, also concerned with health and lifestyle issues, was mailed between 1993 and 1995 to all eligible twins over the age of 50 and registered with the ATR, including but not limited to those who had participated in the other two health and lifestyle studies. In each of the three questionnaires, the study participants were asked to provide information on the number, sex, and dates of birth and death of their children and the timing of three life-history events: age at menarche, age at first reproduction, and age at menopause. To estimate the role of maternal and/or cultural effects (Mousseau and Fox 1998a,b), information on each individual's religious affiliation and level of education was also collected. Educational background is represented by six categories ($\leq$ 7 years, 8–10 years, or 11–12 years postsecondary apprenticeship or certificate; postsecondary technical or teaching diploma; and university degree or higher), whereas religious affiliation was collapsed into four categories (no religion, non-Catholic Christian [e.g., Protestant, Orthodox, Evangelical, or Fundamentalist], Catholic Christian, and other religion [non-Christian]).

To estimate fitness (measured as *r*) of individual twins, we needed to be confident that each twin who we studied had completed their reproduction. Because many of the subjects were surveyed at multiple timepoints, it was possible to empirically determine an age at which this could be assumed to have occurred. Examination of the cumulative distribution of ages at last reproduction indicated that a cut-off age of 45 years was appropriate for this sample (because only six of 3418 women reported reproducing after this age), resulting in a study sample of 2710 women (1001 pairs and 708 singles) for whom reproductive fitness data were available. Where it was impossible to determine with absolute certainty whether a particular participant had children (e.g., noncompletion of reproductive history questionnaire items across all studies in which she had participated), reproductive fitness for that case was assigned missing-value status.

Studies using self-report data are reliant on the ability of participants to recall major life events accurately. To test the validity of this assumption, we have used the longitudinal nature of our study to, initially, record major life-history events as they occur and then, subsequently, measure the accuracy of the participants' recall. Consistency of recall of events like age at menarche has been shown to be generally high (Treloar 1974; Treloar and Martin 1990). However, the correlation between true age at menarche and recalled age of menarche was found to be 0.75 when the participants are questioned approximately 19 years after the event, but reduced to 0.60 after 39 years (Damon and Bajema 1974). To minimize the effect of this declining correlation, the data analyzed for each of the three life-history traits was taken from the earliest report by the participants after the event of interest.

Zygosity

The zygosity of twins was determined on the basis of responses to standard questions about physical similarity and the degree to which others confused them with one another. This method has been shown to give at least 95% agreement with diagnosis based on extensive blood typing (Martin and Martin 1975; Eaves et al. 1989). More recently, a subsample of 198 same-sex pairs from this cohort were typed for 11 independent highly polymorphic markers in the course of an

asthma study, with no errors in previous zygosity diagnosis detected (Duffy 1994).

Measurement of Fitness

The choice of an appropriate measure of fitness is fundamental to life-history studies (Kozlowski 1993). Although a universal definition of fitness is probably unattainable (Stearns 1992), to measure natural selection, fitness must be appropriately defined for individuals in such a way as to be consistent with population genetic theory and the equations for dynamic evolutionary change (de Jong 1994). For a diploid, age-structured, increasing or decreasing population under weak, density-independent selection and at the stable age distribution, an appropriate measure of population fitness is the intrinsic rate of increase (r), or equivalently, the finite rate of increase ($\lambda = e^r$; Charlesworth 1980; Lande 1982a). If natural selection acts to maximize λ, then the fitness of each female within the population can be calculated as a function of her life-history phenotype in such a way as to discount the genetic contribution of each offspring to the next generation by a factor λ (Lande 1982b; Lenski and Service 1982; Kawecki and Stearns 1993), according to the equation

$$w(z) = \lambda \int_0^\infty \lambda^{-x} m(x) l(x), \qquad (1)$$

where $w(z)$ is the fitness of life-history phenotype z, λ is the finite rate of population increase, x is age, $m(x)$ is the number of offspring at each age (divided by two for diploid organisms), and $l(x)$ is the survival function for individuals. An analogous formula defined for genotypes in a single-locus genetic model has been shown to be a fitness measure in populations at genetic equilibrium (Charlesworth 1980). The above formula for individual fitness has the advantage that the mean fitness calculated for individuals in a large, random sample drawn from the population will be equal to the population fitness (Lenski and Service 1982), which justifies the use of this measure in regression analyses to detect natural selection (Lande and Arnold 1983; Arnold and Wade 1984a,b). This is not a property of some other proposed measures of fitness for individuals in age-structured populations (e.g., McGraw and Caswell 1996; T. J. Kawecki, pers. comm.).

Demographic data to calculate λ for the twins' cohort were available from demographic information on the natural increase in the Australian population from 1901 to 1998 (Australian Bureau of Statistics 2000). Women in the study were in their reproductive years from 1920 to 1983, during which time the rate of natural increase in the Australian population varied from a high of 15 per 1000 population to a low of 6.9 per 1000, as shown in Figure 1. Because the variation in the rate of natural increase over these six decades was substantial, we used a value of λ for each woman corresponding to that of the mean year in which she had children. This value was used in all subsequent calculations of individual fitness, using the above equation.

Selection

Exploratory analysis of the data was performed by implementing nonparametric regression of fitness on trait values to visualize the form of selection on individual traits. The method used was a cubic spline approximation technique using the FORTRAN program GLMS (Schluter 1988). Maximum-likelihood methods were used to perform multivariate linear regression of fitness on trait values (Lande and Arnold 1983; Arnold and Wade 1984a,b). In each case, statistical significance was evaluated by testing the significance of the difference when individual regression parameters were removed.

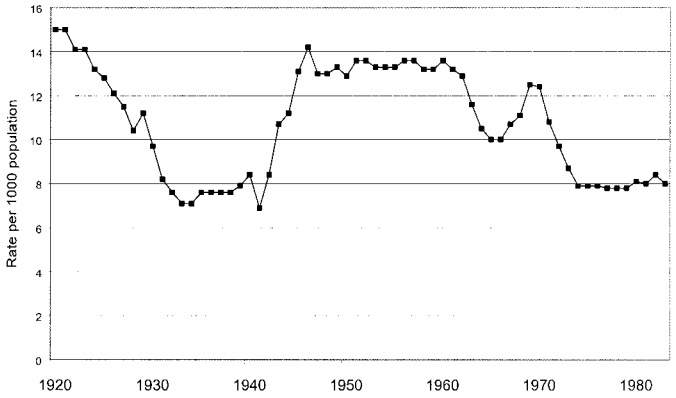

FIG. 1. Growth in the Australian population between 1920 and 1983 due to natural increase (excluding immigration). Data from Australian Bureau of Statistics (2000).

Inheritance

Inspection of the data on reproductive fitness indicated that approximately 10% of the women in the sample never had children, resulting in a reproductive fitness value of zero for these cases. In addition, the method of response for the question pertaining to age at menarche (whole years) resulted in data comprising a small number of discrete categories. Because measurements of these two variables had serious departures from normality, we decided to convert them into categories that could be treated as arising from the imposition of arbitrary thresholds on an underlying, normally distributed, liability dimension. For consistency, we did the same with the other variables so that covariance between all measures could be described in terms of polychoric correlations (correlations calculated between categorical variables under normal distribution theory; Olsson 1979). Consequently, the variables of interest were transformed to ordinal measures for input into structural equation modeling, with reproductive fitness being divided into deciles and age at first reproduction into five categories ($\leq$19, 20–24, 25–29, 30–34, and $\geq$35 years). Previous analyses of age at menarche in this dataset found evidence of a quadratic relationship with reproductive fitness—those having menarche near the mean age (13 years) having the greatest number of children by age 26 and those having menarche either earlier or later having fewer, suggestive of stabilizing selection (Martin and Treloar 1991). We therefore considered age at menarche as a variable consisting of six categories representing the square of the deviation of an individual's age at menarche from the median age at menarche (13 years), expressed as (age at menarche $- \mu)^2$. Age at menopause was divided into six categories

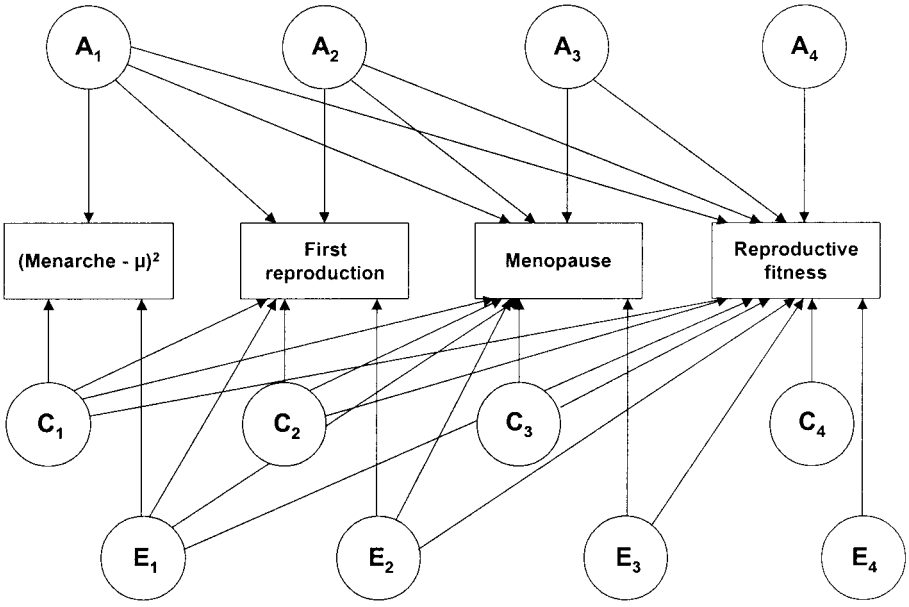

FIG. 2. Path diagram of a full Cholesky decomposition model, depicting latent genetic and environmental influences (circles) on four variables. Additive genetic (A_1, A_2, A_3, and A_4), shared environment (C_1, C_2, C_3, and C_4) and unique environment (E_1, E_2, E_3, and E_4) factors are shown.

(≤34, 35–39, 40–44, 45–49, 50–54, and ≥55 years). Censoring of data occurred where women reported having had hysterectomies, bilateral oophorectomies, or hormone replacement therapy prior to the onset of menopause. However, in cases where these interventions occurred after the age of 44 years or if a participant had reached the age of at least 45 years without the onset of menopause, promotion to the next age bracket was used to minimize loss of information.

Our data on educational background and religious affiliation could not be treated as ordinal and were included in the structural equation models as moderator variables (Neale 1998), with each category assumed to moderate the observed variables for a given individual (fitness, age at menopause, etc.) if true for that individual. This means that the influences of the individual categories (e.g., ≤7 years education vs. 11–12 years education) on each observed variable may be considered separately and tested for equivalence.

Although significant twin correlations establish the fact that there is familial aggregation for the measures of interest, they do not distinguish between the possible mechanisms by which this arises. Potential sources of variation include three (additive genetic effects, A; nonadditive genetic effects, D; and shared environment, C) that make family members more alike than random individuals and one (unique environment, E) that introduces differences between family members. Structural equation modeling may be used to determine which combination of the above parameters provides the simplest explanation for the observed pattern of MZ and DZ twin correlations, with the limitation that shared environmental and nonadditive genetic effects are generally confounded in a study of twins reared together. For example, a much greater resemblance of MZ twins than DZ twins for a given trait of interest indicates the presence of genetic influences on that trait, because DZ twins share, on average, only half their genes and MZ twins are genetically identical. In contrast, an absence of genetic influences would be indicated by the degree of similarity between MZ twins being the same as the degree of similarity between DZ twins. If this degree of similarity were substantial, the presence of shared environmental effects could be inferred. The most parsimonious model is determined by comparing the relative goodness-of-fit of models as assessed by the likelihood-ratio χ^2 (Neale and Cardon 1992).

Extension to multivariate analysis allows the determination not only of the sources of covariation, but also the pattern or structure in which these differentially influence the covarying measures. The preliminary structural equation model investigated was a Cholesky decomposition of the variance/covariance matrix into additive genetic, shared environment, and unique environment effects, shown in Figure 2. In this model, A_1 is an additive genetic factor that may influence the first and subsequent observed variables (i.e., [age at menarche $-$ μ]^2, age at first reproduction, age at menopause, and reproductive fitness), whereas A_2 may load on only the second and subsequent observed variables, A_3 is constrained to influence only the third and fourth variables in the model, and A_4 acts only on the last variable. Similar restrictions apply to shared environment factors C_1, C_2, C_3, and C_4 and to unique environment factors E_1, E_2, E_3, and E_4. The observed variable reproductive fitness was placed in the final position in the model, because we are interested in how much of the additive genetic, shared environmental, and unique environmental influences on reproductive fitness are accounted for by factors also influencing the other three variables (i.e., A_1, A_2, A_3, C_1, C_2, C_3, E_1, E_2, and E_3). This initial model does not test any specific hypothesis, but is used as a standard against which submodels may be tested for goodness-of-fit. Genetic factor submodels can be used to test specific hypotheses about the relationships between individual observed variables. One such submodel, the independent

TABLE 1. Mean trait values by zygosity for reproductive fitness and age at menarche, first reproduction, and menopause, with 95% confidence intervals estimated by maximum-likelihood methods. Estimates were obtained by taking into account twin structure of the data. Individuals are members of twin pairs for whom reproductive fitness could be calculated for at least one twin (628 MZ complete pairs, 130 MZ incomplete pairs, 373 DZ complete pairs, 578 DZ incomplete, or opposite-sex pairs).

Trait	Zygosity of twins		
	MZ (95% CI)	DZ (95% CI)	*P*-value ($\bar{x}_{MZ} = \bar{x}_{DZ}$)
Age at menarche	13.04	13.08	0.56
(1373 MZ, 1310 DZ individuals)	(12.94–13.14)	(12.99–13.16)	
(Age at menarche $-$ μ)^2	2.20	2.22	0.84
(1373 MZ, 1310 DZ individuals)	(2.01–2.40)	(2.06–2.40)	
Age at first reproduction	24.66	24.78	0.52
(1258 MZ; 1187 DZ individuals)	(24.38–24.93)	(24.53–25.04)	
Age at menopause	48.88	47.11	0.22
(746 MZ; 739 DZ individuals)	(48.49–49.27)	(46.73–47.48)	
Reproductive Fitness	1.03	1.04	0.88
(1384 MZ; 1326 DZ individuals)	(1.00–1.07)	(1.01–1.08)	

MZ and DZ refer to monozygous and dizygous twins, respectively. 95% CI refers to 95% confidence intervals. μ, median age at menarche (13 years).

pathway model (Kendler et al. 1987), proposes that the covariance between the four observed variables can be attributed to a single set of additive genetic (A_C), nonadditive genetic (D_C), and nonshared environmental effects (E_C) common to some or all of the observed variables, with the remainder of the variance due to specific additive genetic, shared environment, and nonshared environment effects acting separately on each observed variable. Univariate and multivariate analyses were conducted using maximum-likelihood methods for raw ordinal data implemented in Mx version 1.47c (Neale 1999).

RESULTS

An important preliminary to genetic analysis is to check that MZ and DZ twins are sampled from the same population (Jinks and Fulker 1970). The mean values and standard deviations of life-history traits for MZ and DZ twins are presented in Table 1. No significant difference between MZ and DZ twins was observed for any of the traits, with both MZ and DZ twin groups having mean fitness very close to that of the general population.

Selection

Figure 3 illustrates the relationship between reproductive fitness and the three life-history traits being examined. Age at first reproduction and age at menopause appear to have a nearly linear relationship with fitness, with early age at reproduction and later menopause correlated with higher fitness, while age at menarche demonstrates the aforementioned quadratic relationship with fitness (Martin and Treloar 1991). Bootstrap methods were used to estimate 95% confidence intervals for Figure 3, but have been omitted from the graphs because they were too close to the fitness estimates to be distinguished on the scales used (typically $\pm$ 0.01).

Mean values for life-history traits and fitness by education and religious affiliation are given in Table 2. Religious affiliation was found to influence reproductive fitness, with those identifying as Roman Catholic having substantially higher values (20% above the population average), whereas those reporting no religious affiliation had the lowest reproductive fitness. There were no significant differences between education levels for our menarche measure, although there was a trend to later menopause in more educated women (as has been reported by others; Luoto et al. 1994; Do et al. 1998, 2000), with university-educated women having menopause almost two years later than those with only primary education ($P = 0.013$). However, the most substantial education effects in this sample were observed for both the age at which a woman had her first child and her overall reproductive fitness. Women with less than eight years of formal education tended to have their first child earlier and have overall higher fitness values. Those with university education, in contrast, had their first child comparatively later and had lower mean fitness. Although there is a clear trend toward lower fitness with higher education ($\chi^2 = 26.703$, df = 1, $P < 0.001$), it accounts for only 2.3% of variance in fitness. Similarly, although differences in fitness by religious affiliation are highly significant, they account for only 1.4% of the variance.

Contributions to Fitness

Multivariate regression of the life-history traits with reproductive fitness is shown in Table 3. Because age at first reproduction takes on a missing value for women who have not had children, the multivariate analysis has been performed twice. The first analysis excludes age at first reproduction, whereas the second variable includes age at first reproduction but, due to listwise deletion, excludes those women who had no children. This dual analysis is also useful in exploring the impact of the substantial negative correlation between age at first reproduction and reproductive fitness ($r = -0.41$).

In the first model (which excludes age at first reproduction), age at menopause, education, and religious affiliation are all significantly associated with reproductive fitness. The square of the difference between observed and mean age at menarche approaches significance, whereas age at menarche itself appears unrelated. The second model (including age at first reproduction) shows that, among women who had children, fitness is still significantly related to age at menopause and

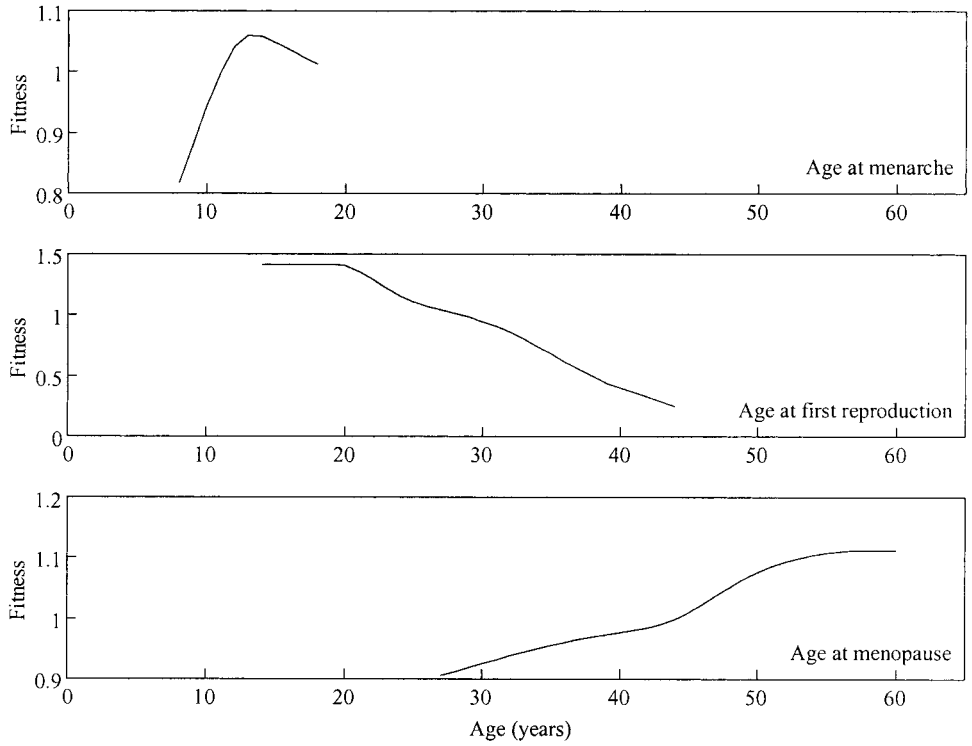

FIG. 3. Nonlinear regression (cubic spline; Schluter 1988) of fitness on age at menarche, age at first reproduction, and age at menopause in a sample of Australian twins.

religious affiliation. However, the inclusion of age at first reproduction in the model has resulted in education becoming a nonsignificant predictor, suggesting that the impact of education on fitness was limited to its effect on the age at which a woman had her first child.

Inheritance

Univariate analysis

Polychoric correlations for MZ and DZ twin pairs are shown in Table 4. For each trait there was a significant correlation for both MZ and DZ twins, with the correlation in each case being stronger for MZ twins than for DZ twins.

Table 5 shows the proportion of variance in each life-history trait that is attributable to each source, based on univariate structural equation modeling. Because the DZ twin correlation (r_{DZ}) is less than half the MZ twin correlation (r_{MZ}), we fitted ADE models for (age at menarche $-\mu$)^2 and fitness and estimated nonzero values for A and D, although neither was significant because of the strong negative correlation between these two estimates (-0.95). For this reason, very large twin samples are required to obtain significant estimates of even very large amounts of dominance (Martin et al. 1978). It is important to note, however, that the joint estimate of genetic variance (A + D) is highly significant for each variable. For age at first reproduction and age at menopause, r_{DZ} is greater than half r_{MZ}, so we have fitted ACE models to these variables. Variance in age at first reproduction can be explained in a univariate model by only unique environmental effects and either additive genetic or shared environmental effects (these two alternatives could not be resolved), whereas additive genetic effects plus unique environmental effects are required to adequately model the sources of variance in age at menopause.

It is worthwhile noting that the proportions of variance in Table 5 are those remaining *after* the contributions to variance by education and religious affiliation have been accounted for. Educational background and religious affiliation might be expected to contribute to shared environment C, and would have masked the nonadditive genetic effect observed for fitness (due to the confounding of shared environment and nonadditive genetic effects) if not modeled separately. However, familial aggregation for education attainment in this sample is largely genetic in origin (Baker et al. 1996), so correcting for it may result in an underestimate of the genetic variance.

Multivariate analysis

Cross-twin, cross-trait correlations for the four variables of interest are shown in Table 6 for MZ and DZ twin pairs. As expected from Table 3, the major cross-trait correlation observed is between age at first reproduction and reproductive fitness, with a smaller correlation between age at menopause and fitness.

As for univariate analysis, the pattern of MZ and DZ cross-correlations can be used to provide insight into the genetic and environmental influences on the traits of interest. However, in the multivariate case more information (and therefore statistical power) is available in the form of the cross-twin, cross-trait elements. It is these terms that allow the determination of how genetic and environmental effects differentially influence the covarying measures of interest.

TABLE 2. Mean values for life-history traits by (A) educational background and (B) religious affiliation, with number of individuals in each category in parentheses. Percentages of variance in life-history traits accounted for by education and religious affiliation are also shown, with *P*-values from chi-square tests of statistical significance.

(A) Religious affiliation

Trait	Observed means—religious affiliation categories: No religion (486)	Non-Catholic Christian (3957)	Roman Catholic (1336)	Other religion (219)	% variance	*P*-value
(Age at menarche − μ)²	2.02	2.14	2.08	2.03	0.0	0.863
Age at first reproduction	25.64	24.73	24.55	24.83	0.2	0.257
Age at menopause	48.45	48.55	48.18	48.30	0.2	0.349
Fitness	**0.86**	**1.01**	**1.20**	**0.99**	**1.4**	**<0.001**

(B) Educational background

Trait	Observed means—education categories: <7 years (257)	8–10 years (2002)	11–12 years (1607)	App/Cert[1] (907)	Diploma (795)	University (476)	% variance	*P*-value
(Age at menarche − μ)²	1.98	2.18	1.99	2.32	2.01	2.03	0.2	0.293
Age at first reproduction	**23.78**	**23.84**	**24.58**	**25.75**	**25.99**	**27.08**	**7.2**	**<0.001**
Age at menopause	47.47	48.49	48.36	48.81	48.38	49.35	0.6	0.120
Fitness	**1.22**	**1.07**	**1.01**	**0.95**	**1.02**	**0.87**	**2.3**	**<0.001**

μ = median age at menarche (13 years)

[1] App/Cert, postsecondary education to apprenticeship or certificate level. Diploma, postsecondary education to diploma level (technical or teachers' college).

Figure 4 represents the ACE Cholesky decomposition for the variables in this study, with paths accounting for less than 1% of the variance in any variable omitted for simplicity. In this multivariate model, educational attainment accounts for 7% of the variance in age at first reproduction and 2% of the variance in reproductive fitness, while a further 1% of the variance in fitness can be attributed to religious affiliation. Shared environmental effects were found to contribute significantly to the variance between individuals for only age at first reproduction and age at menopause, with no other significant shared environmental correlations between variables ($\Delta\chi^2 = 2.10$, df = 8, $P = 0.978$). However, significant additive genetic effects were found for all four variables. Additive genetic and unique influences on age at first reproduction (A_1 and E_1) were found also to have a strong influence on reproductive fitness. Approximately 42% of the additive genetic and 18% of the unique environment influences on reproductive fitness also affect age at first reproduction. As expected, these influences are in opposite directions on the two variables, because earlier age at first reproduction increases reproductive fitness. The additive genetic effects acting on age at menopause make a minor contribution to variance in fitness (4% of variance, not significant).

An alternative, more restrictive model (independent pathway model; Kendler et al. 1987) has been shown to be able to include both C and D effects while maintaining a model fit invariant to the order of the observed variables, provided that the pattern of effects on the observed variables is different (D. L. Duffy, pers. comm.). The independent pathway model presented in Figure 5 consists of a set of additive genetic (A_C), nonadditive genetic (D_C), and nonshared environmental effects (E_C) common to some or all of the observed variables, with specific additive genetic, shared environment, and nonshared environment effects acting separately on each observed variable. This independent pathway model is not significantly different from the atheoretical Cholesky model in Figure 4 in terms of fit to the data ($\Delta\chi^2 = 9.557$, df = 10, $P = 0.480$) and has the advantages of testing a specific hypothesis.

It can be seen from Figure 5 that, under the independent pathway model, the common additive genetic effect A_C accounts for very little of the variance in age at menopause

TABLE 3. Results from multivariate regression analysis of life-history traits with reproductive fitness. Only individuals with complete education, religious affiliation, and reproductive history information could be included in the analysis ($n = 1459$ for model not including age at first reproduction; $n = 1330$ for model including age at first reproduction). Correlations (Corr) ± standard error (SE), percent of variance in fitness accounted for, and statistical significance in regression analysis are presented.

Trait	Corr ± SE	Model not including age at first reproduction: % variance	*P*-value	Model including age at first reproduction: % variance	*P*-value
Age at menarche	0.01 ± 0.03	0.00	0.815	0.00	0.841
(Age at menarche − μ)²	−0.05 ± 0.02	0.27	0.133	0.14	0.128
Age at first reproduction	−0.41 ± 0.02	—	—	15.93	<0.001
Age at menopause	0.07 ± 0.03	0.41	0.003	0.65	0.001
Educational background	—	1.51	<0.001	0.28	0.455
Religious affiliation	—	2.08	<0.001	4.18	<0.001

μ = median age at menarche (13 years).

TABLE 4. Univariate polychoric correlations between twins for monozygotic (MZ) and dizygotic (DZ) pairs, calculated by maximum-likelihood methods and corrected for the effects of educational background and religious affiliation. n_{pair} represents the number of twin pairs with complete information for trait of interest.

Trait	Monozygotic twins			Dizygotic twins		
	r_{MZ}	95% CI	n_{pair}	r_{DZ}	95% CI	n_{pair}
Fitness	0.42	(0.35–0.49)	627	0.19	(0.09–0.29)	374
(Age at menarche − μ)²	0.51	(0.46–0.55)	696	0.17	(0.09–0.25)	421
Age at first reproduction	0.39	(0.32–0.45)	555	0.28	(0.20–0.37)	313
Age at menopause	0.52	(0.44–0.58)	429	0.29	(0.16–0.41)	251

95% CI refers to 95% confidence intervals. μ = median age at menarche (13 years).

(approximately 1%, significant) or (age at menarche − μ)² (<0.3%, not significant). However, it accounts for nearly half the total additive genetic variance on age at first reproduction and almost all of the additive genetic effect on fitness. A similar pattern is observed for nonshared environment (E_C), with little or no nonshared environmental effects on fitness that do not also act on age at first reproduction (E_4). The strong nonadditive genetic effect on (age at menarche − μ)² can be clearly seen (D_C), but it has only a minor impact on reproductive fitness (<0.5% of variance, not significant). The genetic correlations between all four variables of interest are summarized in Table 7, with 95% confidence intervals.

DISCUSSION

Our analyses revealed that variations in all three life-history traits were heritable to some extent, with broad heritability estimates of 50% for (age at menarche − μ)², 23% for age at first reproduction, and 45% for age at menopause (age at menarche itself has previously been estimated in this sample to have a broad heritability of 0.61–0.68; Treloar and Martin 1990). Age at reproduction and age at menopause were both significantly correlated with fitness, although genetic covariation with reproductive fitness was quite weak for age at menopause. These results suggest that, over time, selection may cause the evolution of earlier age at first reproduction and perhaps later age at menopause in this population. Analysis of the quadratic term (age at menarche − μ)², on the other hand, found no evidence for selection, due to the absence of a significant genetic correlation of this variable with fitness.

One of Fisher's hypotheses associated with his theory of dominance (Fisher 1958) predicted that traits closely associated with fitness should have a significant dominance variance component, both due to the erosion of the additive component of variance and the evolution of directional dominance (see also Haldane 1932). It is interesting, therefore, that the only trait for which we detected any evidence of nonadditive genetic variance was age at menarche, echoing an earlier analysis of this trait in a subset of the data (Treloar and Martin 1990). Although we could not detect natural selection on this variable in the contemporary postindustrialized population we studied, other studies suggest strongly that age at menarche was/is under selection in several pre- or nonindustrialized populations (e.g., Critescu 1975; Aghajanian 1981; Gubhaju 1983; Borgehoff Mulder 1989).

Age at menarche may have been correlated with fitness in the ancestral populations from which the contemporary Australian population has been drawn, with a relaxation of selection occurring due to cultural change. It should be noted, however, that an earlier analysis of a subset of these data, using cruder measures of reproductive success, did find significant relationships with age at menarche, suggesting maximum fitness at the median age (13 years), falling off at both older and younger ages at menarche (Martin and Treloar 1991). The same relationship is still apparent here, although the maximum-likelihood estimate, taking relatedness of subjects into account, is no longer significant. Life-history traits are usually assumed to be relatively closely correlated with measures of fitness, especially when compared to other sorts of traits (Gustafsson 1986; Mousseau and Roff 1976; Roff 1992; Stearns 1992; Kruuk et al. 2000; Merila and Sheldon 2000). However, in a benign environment many individuals

TABLE 5. Univariate structural equation modeling of life-history traits and reproductive fitness, corrected for effects of educational background and religious affiliation. A and D represent proportions of variance in each life-history trait attributable to additive and nonadditive genetic influences on the trait of interest, respectively, whereas C and E represent proportion of variance attributable shared and nonshared environmental effects, respectively.

Trait	Factors in structural model				
	A	D	A + D	C	E
(Age at menarche − μ)²	0.19 (0.00–0.51)	0.32 (0.00–0.55)	0.51 (0.46–0.55)	—	0.49 (0.45–0.54)
Age at first reproduction	0.21 (0.00–0.42)	—	0.21 (0.00–0.42)	0.18 (0.00–0.36)	0.61 (0.55–0.68)
Age at menopause	0.44 (0.17–0.58)	—	0.44 (0.17–0.58)	0.07 (0.00–0.31)	0.49 (0.41–0.56)
Fitness	0.36 (0.00–0.48)	0.06 (0.00–0.46)	0.42 (0.49–0.35)	—	0.58 (0.51–0.65)

μ = median age at menarche (13 years).

TABLE 6. Table of cross-twin cross-trait correlations for (age at menarche − μ)², age at first child, age at menopause, and fitness, where μ is the median age at menarche, 13 years. Twin correlations for individual traits are shown in bold type. Results for monozygotic twins appear above the main diagonal and dizygotic twins below the main diagonal.

		Twin 1				Twin 2			
		(Menarche − μ)²	First reproduction	Menopause	Fitness	(Menarche − μ)²	First reproduction	Menopause	Fitness
Twin 1	(Menarche − μ)²		0.01	−0.14	−0.11	**0.51**	0.03	−0.12	0.02
	First reproduction	−0.02		0.04	−0.47	0.03	**0.40**	−0.03	−0.24
	Menopause	0.02	−0.06		0.13	−0.10	0.02	**0.52**	0.04
	Fitness	0.03	−0.45	0.05		−0.07	−0.22	0.15	**0.41**
Twin 2	(Menarche − μ)²	**−0.17**	0.02	−0.08	−0.08		0.02	−0.08	−0.02
	First reproduction	0.05	**0.29**	−0.02	−0.10	0.00		−0.08	−0.54
	Menopause	−0.09	−0.02	**0.28**	−0.04	−0.08	−0.07		0.12
	Fitness	−0.04	−0.15	−0.02	**0.18**	−0.03	−0.40	0.13	

may obtain an opportunity to reproduce, regardless of the adaptedness of their phenotype, leading to a breakdown in the correlation between variation in life history and variation in fitness. Indeed there is evidence for relaxed mortality selection on human birth weight (Ulizzi and Terrenato 1987). The greater degree of protein polymorphism in humans in comparison to chimpanzees (*Pan troglodytes*) has also been used to suggest that it resulted from relaxed natural selection during the Pleistocene (Takahata 1993). We suspect that the traits most closely linked to fitness in postindustrialized human populations are now behavioral/psychological traits, although this has been difficult to demonstrate (e.g., Eaves et al. 1990; Mealey and Segal 1993).

Our finding that age at first reproduction is correlated with fitness may appear hardly surprising given that age at first reproduction is a term in the formula we used to calculate fitness. However, the fact that we also find a correlation between age at first breeding and completed family size, Charlesworth's (1980) recommended measure of fitness, suggests strongly that this association is not due to autocorrelation. Rather, our structural models suggest that the covariance between age at first reproduction and fitness is due to an interaction between genetic and maternal/environmental effects. Multivariate regression analysis suggests, for example, that the majority of the small but statistically significant effect of educational attainment on our measure of fitness is mediated through its effect on age at first reproduction, most noticeably in the late reproduction and lower fitness among

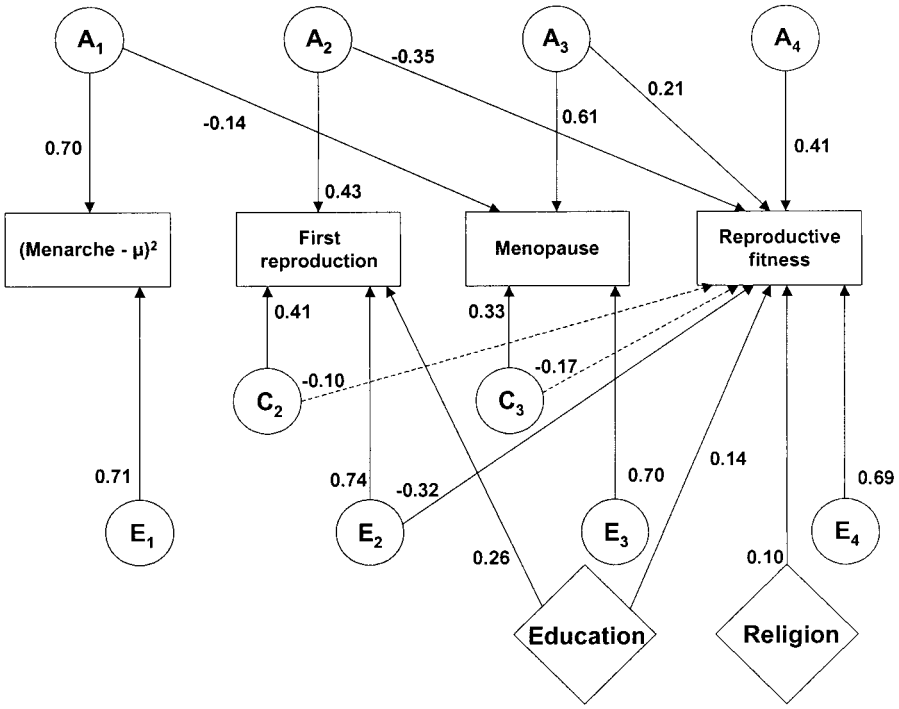

FIG. 4. Path diagram of full Cholesky decomposition model on (age at menarche − μ)², age at first reproduction, age at menopause, and reproductive fitness. Numbers by paths are path coefficients and must be squared to obtain proportions of variance of the measured variable accounted for by the latent variable. All latent variables have unit variance. Paths accounting for less than 1% of the variance in an observed variable have been omitted for simplicity; nonstatistically significant paths are shown as dashed lines. Moderator variables (education, religious affiliation) are shown as diamonds.

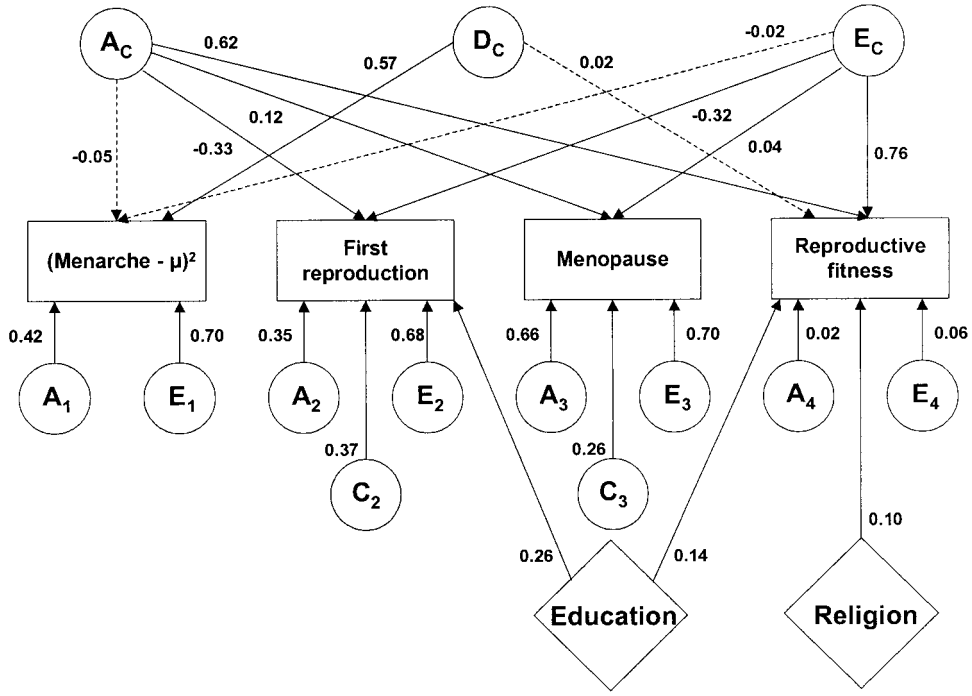

FIG. 5. Path diagram of an independent pathway model, showing (age at menarche $-\mu)^2$, first reproduction, age at menopause, and reproductive fitness. Latent variables whose effects are common to more than one observed variable are denoted by a subscript c, and effects specific to individual variables are indicated by numeric subscripts. Other notation as in Figure 4.

university-educated women, and earlier reproduction and higher fitness among women with less than seven years of formal education. Religious affiliation, on the other hand, seems to influence reproductive fitness but not age at first reproduction. Structural equation modeling indicates that the majority of the additive genetic and nonshared environmental effects on fitness also influence the age at which a woman first reproduces. These common genetic influences are not of equal strength, however, with age at first reproduction having a much smaller heritability than fitness (23% vs. 39%). Additionally, approximately half the additive genetic effect on age at first reproduction is actually due to other influences not shared with our measure of fitness (Fig. 5).

The robust genetic covariance between age at first breeding and fitness supports the theories that early reproduction is favored in increasing populations (e.g., Charlesworth 1980; Lande 1982a) and that, in iteroparous organisms, early reproduction maximizes the number of opportunities for reproduction over a lifetime, provided there is no trade-off between early and late reproduction. Women who delay reproduction until their mid to late 30s are relatively unlikely to have enough time to produce a large family before they reach menopause. Our conclusion that there is a selective advantage associated with earlier age at first reproduction in our population of modern Australian women is also interesting in the light of a recent study of natural selection in three pre-industrial Western human populations (Käär et al. 1996), which also found evidence of relationships between age at first reproduction and family size. Although it is difficult to compare our study with that of Käär et al. (1996) because our databases were obtained in such different ways (theirs from several-hundred-year-old church records of births, and ours from self-reported questionnaires), it is plausible that there has been a real decrease in the selection gradient on age at first reproduction in historical times. Improvements in diet, sanitation, and medical treatment asso-

TABLE 7. Maximum-likelihood estimates of genetic correlations between (age at menarche $-\mu)^2$, age at first reproduction, age at menopause, and reproductive fitness. Heritability estimates are shown on the diagonal and 95% confidence intervals are in parentheses.

	(Age at menarche $-\mu)^2$	Age at first reproduction	Age at menopause	Reproductive fitness
(Age at menarche $-\mu)^2$	0.51 (0.46–0.55)			
Age at first reproduction	0.02 (−0.03–0.06)	0.24 (0.08–0.43)		
Age at menopause	−0.01 (−0.03–0.01)	−0.04 (−0.08–−0.01)	0.45 (0.16–0.58)	
Reproductive fitness	−0.03 (−0.10–0.05)	−0.21 (−0.27–−0.15)	0.08 (0.01–0.14)	0.39 (0.33–0.46)

ciated with industrialization may mean that women in Western culture now lead longer, healthier lives and can reproduce more successfully at older ages than before. Furthermore, the introduction of effective contraception in the early 1960's has provided women with the potential for much greater control over their family size and the timing of births. We tentatively suggest that these cultural changes may have resulted in more relaxed selection on age at first reproduction, although far more comparisons between pre- and postindustrialized populations would be needed to test this idea rigorously.

Heritability of Fitness

Our analyses also suggest that variation in fitness itself appears to be partly heritable, with multivariate modeling estimating that approximately 39% of the variance (95% confidence interval = 33–46%) is attributable to additive genetic effects, the remainder consisting of unique environmental effects and some suggestion of a small, nonadditive genetic effect. This finding is intriguing because many theoretical frameworks suggest that, in populations at equilibrium, fitness should have almost zero heritability and very low additive variance due to the erosive effects of selection (see reviews by Gustafsson 1986; Charlesworth 1987; Jones 1987; Mousseau and Roff 1987; Frank and Slatkin 1992; Burt 1995, 2000; Merila and Sheldon 1999; Walsh and Lynch 2000).

Why then is our estimate of the heritability of fitness so high? Several explanations are possible. One explanation, based on the line of argument in the previous section, is that there is heritable variation in fitness because the population is not at equilibrium. If the cultural environment has changed so that behavioral traits now have a greater influence on fitness than do life-history traits, the additive variation in fitness may be due to differences between individuals with respect to behavioral variation. At present, we do not know which, if any, behavioral traits are correlated with fitness or reproductive success in this population. However, Kohler et al. (1999) hypothesize that ''genetic influences on fertility are most relevant when the number of children results from a deliberate and conscious decision, and when social norms and economic conditions allow a relatively broad range of life-course alternatives.'' Also, previous studies have identified associations between behavioral traits and crude estimates of fitness (see Eaves et al. 1989, 1990), although these associations have not been tested using the approach developed here.

Alternatively, the additive changes in fitness may be due to perturbation of gene-environment (G $\times$ E) interactions. Genetic variance (and therefore heritability) will decline to zero only ''in the absence of perturbing forces'' (Houle 1992). If the recent changes in the cultural environment have moved postindustrialized populations to another position along the reaction norm for fitness, this may result in an increase in the additive-genetic component of variance, even though the underlying genetic causes of fitness remain unaltered (see Lewontin [1974] for a discussion of the important effects of G $\times$ E interaction on genetic variance, and Rodgers et al. [2000] for a discussion of potential sources of perturbation).

These two types of explanation for the high heritability of fitness complement a newly emerging framework for understanding the quantitative genetic basis of variation in fitness and fitness-related traits (see Houle 1991, 1998; Price and Schluter 1991; Schluter et al. 1991; Fowler et al. 1992; Burt 1995, 2000; Pomiankowski and Møller 1995; Rowe and Houle 1996; Merila and Sheldon 1999). This framework is based on the idea that the infamously low heritability of fitness-related traits (e.g., Gustafsson 1986; Mousseau and Roff 1987) is not necessarily due to low additive genetic variation per se, but due to strong environmental, maternal, or nonadditive effects (heritability being the ratio between variance due to additive genetic sources and variance due to all other sources; see Price and Schluter 1991; Pomiankowski and Møller 1995). It has been argued, for instance, that fitness-related traits are likely to be influenced by many more loci than many morphological traits (Houle 1991, 1992, 1998; Houle et al. 1996) and that fitness-related traits may be subject to strong G $\times$ E interactions (Schluter et al. 1991; Rowe and Houle 1996). Indeed, such views have recently received support from studies showing that, although the heritabilities of fitness and fitness-related traits are usually low, the corresponding coefficients of variance of additive genetic variance are often rather high (e.g., Pomiankowski and Møller 1995; Kruuk et al. 2000; Merila and Sheldon 2000). Such work is relevant to the results of our twin analyses because it predicts that, because low fitness heritability is due to a masking of (rather than an absolute lack of) additive genetic variation, high heritabilities for fitness may be readily exposed by perturbation, either in the form of selection or of G $\times$ E interactions.

Finally, our finding of high fitness heritability could be dismissed as an artifact if MZ twins were atypical with respect to their reproductive ecology (this would exaggerate the extent to which MZ twins resembled one another). However, this is not the case because in our sample there is no significant difference in mean or variance between the fitness (or completed family size) of MZ and the fitness of DZ twins (Table 1), with both zygosity groups having mean fitness values very close to the expected value of 1.00. This suggests that the high heritability of fitness that we have observed is likely to be a real phenomenon and demands explanation, perhaps by examining which behavioral/psychological traits genetically covary with fitness in Western women.

Acknowledgments

The authors thank M. Blows, T. Kawecki, J. S. Jones, R. Lande, L. Mealey, B. Sheldon, R. Slade, P. Ward, and B. Walsh for advice and/or discussions and M. Blows, A. Burt, S. Clegg, P. Dwyer, F. Frentiu, M. Minnegal, T. Mousseau, B. Sheldon, and P. Ward for comments on previous drafts of the manuscript. This research has been supported by grant 941177 from National Health and Medical Research Council, and by grants AA07535, AA07728, and AA11998 from National Institutes of Health. We also thank the twins for their participation. The Australian Twin Registry is supported by the Australian National Health and Medical Research Council.

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Corresponding Editor: T. Mousseau