Evolution and Path Models in Human Behavioral Genetics

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The evolutionary implications of the path-analysis model most often used in human behavior genetics are examined. With directional selection, a model of pure vertical environmental transmission does not respond in a fully adaptive fashion. Unless the coefficients of transmission are exactly 0.50, the population mean will not equilibrate at the selective optimum over time. If there is both genetic and vertical environmental transmission, then the population mean can equilibrate at the selective optimum. In the presence of genetic transmission, vertical environmental transmission increases population fitness and has a strong effect on the rapid movement of the mean toward the selective optimum. This raises the intriguing paradox of why empirical evidence suggests that vertical environmental transmission is usually small when it possesses such important fitness properties.

KEY WORDS: genetics; evolution; cultural transmission; path analysis.

INTRODUCTION

A neglected area of human behavior genetic research is the juncture of the psychological mechanisms implied by current path models and the quantitative theory that depicts how these mechanisms may have evolved. For example, how would a mutant allele that biased learning toward imitation of one's parents fare in the process of natural selection? It is quite obvious that some species do not engage in such imitation. Oak

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trees and salmon are two examples. It is also quite obvious that higher primates imitate conspecifics (e.g., Bandura, 1969; Mineka et al., 1984). Consequently, at some point in the divergence between salmon and Homo sapiens, genes must have mutated and changed in frequency to transfigure the nervous system to permit imitation in one species but not the other.

From this perspective, the data-analytic models in human behavior genetics should pass a minimal test of "evolutionary robustness." The model, along with the substantive conclusions generated from assessing the fit of the model to observed data, must be plausible in terms of its response to natural selection, its ability to equilibrate over time, etc. Despite well-developed theories for the evolution of cultural transmission (Boyd and Richerson, 1985; Cavalli-Sforza and Feldman, 1973, 1981; Karlin, 1979; Lumsden and Wilson, 1981), the evolutionary properties of the model used most often in behavioral genetics to predict parent–offspring resemblance have not been fully explicated for the audience of this journal. Not only is this type of research required as a part of model validation, but the predictions from the models may point to the evolutionary history behind behavioral traits. The present article is an attempt to redress this omission.

In developing a formula for the evolution of familial transmission, the generic approach of Karlin (1979) is followed. Here, two types of models are used—the first for selection and the second for transmission. The goal of these two types of models is to develop a series of recursion equations that give a variable at time \( t + 1 \) as a function of variables at time \( t \). In the selection model, we begin with a population of offspring. In the process of maturation, these offspring are subject to natural selection which alters the distribution of phenotypes and genotypes when the offspring reach reproductive age. In the transmission model, the selected offspring, now adults, transmit genes and act as models that their offspring imitate.

The actual models of selection and transmission used here were first explicated as "complex transmission" by Feldman and Cavalli-Sforza (1977) and bear similarity to the models put forward by Karlin (1979) and Kirkpatrick and Lande (1989) among others. The following two sections present the salient points of this general model. However, to keep the overall model relatively simple, the following assumptions are made: (1) all gene action is additive; (2) mating is random; (3) before selection begins, the population is at an equilibrium for means, covariances, and variances; (4) in the initial equilibrium population, all variables are multivariate normal; (5) no gender and age differences exist in variables or parameters; (6) generations are discrete; (7) there is no mu-
tetation; (8) in the equilibrium population, the variables are scaled to have means of 0.0; and (9) the genetic system is polygenic, with an infinite number of loci with equal allele frequencies at each locus.

MODELS OF SELECTION AND TRANSMISSION

Selection

Let $p_o$, $p_m$, and $p_f$ denote the phenotypes of an offspring, mother, and father, respectively. The typical path model that includes cultural transmission expresses the offspring’s phenotype as a function of offspring genotype and offspring environment. The offspring environment is, in turn, a linear function of the parental phenotypes. Thus, we may write

$$p_o = g_o + e_o = g_o + bp_m + bp_f + u$$

(1)

Here, $b$ is a regression weight (for convenience, taken to be the same for mother and father), and $u$ is a residual.

We assume that the phenotype is distributed as a normal with mean $m$ and variance $V$. The equilibrium condition implies that in the initial population before selection

$$m = g + 2bm = g/(1 - 2b)$$

(2)

where $g$ is the mean genotypic value of the population. Under random mating, equilibrium also implies that the covariance between genotype and phenotype in offspring will equal that in parents, or

$$\text{cov}(g_o, p_o) = \text{cov}(g_m, p_m) = \text{cov}(g_f, p_f)$$

$$= \text{cov}(g, p)$$

$$= V_g + b \text{cov}(g, p)$$

(3)

$$= V_g/(1 - b)$$

where $V_g$ is the genotypic variance. Phenotypic variance will also be equal in the two generations. Hence, the variance in the initial equilibrium population will be

$$V = V_g + 2b^2V + 2b[\text{cov}(g_o, p_m) + \text{cov}(g_o, p_f)] + V_u$$

$$= [V_g + V_u + 2b \text{cov}(g, p)]/(1 - 2b^2)$$

(4)

where $V_u$ is the residual variance. For simplicity, it is assumed that variables in the equilibrium population are scaled with means of 0.0.

We now assume that the offspring in the equilibrium population are
subject to natural selection as they move into adulthood. Of course, natural selection does not necessarily imply mortality, but for the sake of illustration it is convenient to talk about selection in such terms. The selection process is modeled by a fitness (mortality) function that is proportional to a Gaussian curve with mean \( \mu \) and variance \( \sigma^2 \). That is, the fitness of those with phenotypic values of \( x \) is

\[
w(x) = \text{constant} \cdot \exp\left\{-\frac{1}{2} (x - \mu)^2/\sigma^2\right\}
\]

Strictly speaking, this type of function represents stabilizing selection in which the optimum phenotypic value is \( \mu \) (i.e., the optimum from the point of view of reproductive fitness) and \( \sigma^2 \) signifies the inverse of selection intensity. That is, large values of \( \sigma^2 \) denote weak selection and small values represent strong selection. This type of function can also be used to model the initial stages of directional selection by using a value for \( \mu \) that is remote from the population mean and examining the approach to equilibrium.

The proportion of the juvenile population that survives to reproduce is

\[
s(x) = \text{constant} \cdot V^{1/2} \int_{-\infty}^{x} w(x) \exp\left\{-\frac{1}{2}(x - m)^2/V\right\} dx
\]

\[
= \text{constant} \cdot \left[\sigma^2/(V + \sigma^2)\right]^{1/2} \exp\left\{-\frac{1}{2}(m - \mu)/(V + \sigma^2)\right\}
\]

(see Cavalli-Sforza and Feldman, 1981, Eq. 5.6.9). We define \( s(x)/\text{constant} \) as the fitness of the population.

We assume that the genotypic values \( (g) \) and phenotypic values \( (p) \) before selection are distributed as a bivariate normal with mean vector \( m, m = (g, m)' \), and covariance matrix \( W \),

\[
W = \begin{pmatrix} V_g & C \\ C & V \end{pmatrix}
\]

Let

\[
\Sigma = \begin{pmatrix} 0 & 0 \\ 0 & 1/\sigma^2 \end{pmatrix}
\]

Feldman and Cavalli-Sforza (1977) and Karlin (1979) give the covariance matrix after selection at time \( t \) as

\[
W_t^* = \left(W_t^{-1} + \Sigma\right)^{-1} = \begin{pmatrix} V_g^* & C_t^* \\ C_t^* & V_t^* \end{pmatrix}
\]
Here and in the following, we let an asterisk denote a quantity after selection.

Feldman and Cavalli-Sforza (1977) give the mean vector after selection as

\[
m_{t}^{*} = W_{t}^{*} \left( W_{t}^{-1} m_{t} + \sum \mu \right) = \left( g_{t}^{*}, m_{t}^{*} \right)
\]

where \( \mu \) is vector \((0, \mu)'\).

**Transmission**

Transmission to the next offspring generation may be modeled by using the traditional path model of parent–offspring resemblance with the substitution of variables with asterisks on the right-hand side of the structural equations. This model is reproduced in Fig. 1 and has been used by several groups of researchers (Carey and Rice, 1983; Cavalli-Sforza and Feldman, 1973; Eaves et al., 1978, 1989; Feldman and Cavalli-Sforza, 1977; Fulkner and DeFries, 1983; Loehlin, 1978).

The structural equation for the phenotype of the unselected offspring at generation \((t + 1)\) is

\[
p_{t+1} = g_{t+1} + bp_{m_{t}}^{*} + bp_{u_{t}}^{*} + u
\]

\[
= 1/2 g_{m}^{*} + 1/2 g_{t}^{*} + u_{g} + bp_{m_{t}}^{*} + bp_{u_{t}}^{*} + u
\]

(11)

![Fig. 1. A path model of vertical transmission.](image-url)
Here $\mu_g$ denotes a residual genotypic value (i.e., segregation from mid-parent) and $p^*_{mt}$ and $p^*_{tf}$ are phenotypes of mother and father in generation $t$. They have asterisks because they represent the parental phenotypes after the selection process. We assume that the genotypic mean in the offspring equals that of their selected parents. Hence, the phenotypic mean in the offspring generation will be

$$m_{t+1} = g^*_t + 2bm^*$$

(12)

where $g^*$ and $m^*$ were given above in Eq. (10). Under random mating among the adult survivors, the genotypic variance of offspring equals that of the selected parents. That is, $V_{g,t+1} = V_{g,t}$. Then the phenotypic variance in the unselected offspring will be

$$V_{t+1} = V_{g,t} + 2b^2V^*_{t} + 2bcov(g^*,p^*) + V_u$$

(13)

and the covariance between the genotype and the phenotype is

$$cov(g_p)_{t+1} = V_{g,t} + b cov(g^*,p^*)$$

(14)

The quantities on the right-hand sides of Eqs. (13) and (14) are functions of $b$, $V_u$, and the quantities in the selection equation (9). Hence, the recurrence equations may be solved as a function of three parameters for transmission ($b$, $V_g$, and $V_u$) and two parameters of selection ($\mu$, $\sigma^2$).

**RESULTS**

**Pure Cultural Transmission**

The case in which $V_g = 0$, or pure cultural transmission, is now examined. Using Eqs. (12) and (10),

$$m_{t+1} = 2bm^*_t = 2b[\alpha_t m_t + (1 - \alpha_t)\mu]$$

(15)

where $\alpha_t = \sigma^2 / (\sigma^2 + V_t)$. A similar recursion can be written for the phenotypic variance,

$$V_{t+1} = 2b^2V^*_t + V_u = 2b^2\alpha_t V_t + V_u$$

(16)

When $\sigma^2 \gg V$, then $\alpha$ will change only trivially over time and hence can be treated as a constant. Then the approximate equilibrium value for the mean ($m_e$) occurs when $m_{t+1} = m_t$, or

$$m_e = (1 - \alpha)\mu/(1 - 2\alpha b)$$

(17)

Equation (17) highlights the major problem with pure cultural transmission. In order for the population to move so that the mean reaches the selection optimum of $\mu$, then $(1 - \alpha) = (1 - 2\alpha b)$, requiring that $b =$
0.50. With $b < 0.50$, the population will equilibrate at an optimum between the initial population mean and $\mu$. With the quantity $2\alpha b > 0.50$ (which implies that $b$ is slightly greater than 0.50), Eq. (17) for equilibrium does not hold. The population mean will continuously change over time and pass the selective optimum without stopping there. Figure 2 illustrates the situation.

It is quite clear that the model of pure cultural transmission used in current behavior genetic analysis does not give an evolutionally sensible system when the constraint that $b = 0.50$ is not imposed. One remedy is to fix $b$ at 0.50 and then assess the fit of a model of pure cultural transmission. This implies that the parent–offspring correlation must be 0.50 in the absence of assortment. Observed data on intact nuclear families are sufficient to reject this hypothesis under the condition that test measurement error is either negligible or estimable from the data.

**Mixed Transmission**

The case of both genetic and cultural transmission is now considered. To illustrate the basic properties of the model, several series of calculations were performed using the recursion equations given above. In all cases, weak directional selection with $\mu = 5$ and $\sigma^2 = 100$ was modeled. Under these conditions, for every 10 children left by an individual two standard deviations below the mean, 12 children would be left by an individual two standard deviations above the mean. It was

![Fig. 2. Change in population mean in response to selection under pure cultural transmission.](image-url)
assumed that, in the absence of cultural transmission, the population distribution was a standard normal, i.e., \(V_g + V_u = 1.0\).

The fitness of the populations was expressed as fitness relative to the fitness of the initial, equilibrium population in which cultural transmission was absent. Two initial heritabilities were used—\(V_g = 0.20\) and \(V_g = 0.40\)—and \(b\) was set to values of 0.0, 0.15, 0.30, and 0.45. Figure 3 presents the relative fitness and the means of these populations over time.

It is readily apparent that the main influence of cultural transmission is to change the mean of the distribution. Compared to pure genetic transmission, a small amount of cultural transmission provides a more rapid approach to the selection optimum under conditions of both low and moderate heritability. Despite the increase in variance induced by cultural transmission (which will tend to reduce fitness), relative fitness also increases with \(b\). If we consider the fact that selection is weak, the actual difference in fitness is quite striking, particularly at the early generations. If the intensity of selection were stronger, then these differences would be magnified. It is clear from these calculations that any weak mechanism that induced offspring to imitate their parents would be favored under directional selection and should increase in frequency.

Fig. 3. Change in population fitness and population mean in response to selection with initial heritability of 0.20 (a) and 0.40 (b).
DISCUSSION

How does the model fare from an evolutionary perspective? If the model were to be graded, it would not get an F; at the same time, however, it does not deserve an A. Of particular importance is the specific case of pure cultural transmission. This model makes little evolutionary sense without the specific constraint that \( b = 0.50 \). Yet in testing a model of pure cultural transmission, \( b \) is always permitted to be a free parameter. In effect, this is testing a system that is unlikely to have evolved.

Perhaps the most important observation to emerge from this analysis is the fact that moderate amounts of cultural transmission are indeed adaptive in response to natural selection. Not only does a system with moderate cultural transmission have better fitness than one with only genetic transmission, but cultural transmission dramatically increases the rapidity with which the population mean moves toward the optimum phenotype. In the presence of directional selection, mechanisms that would promote weak imitation of parents would be adaptive and, hence, should increase.

Why, then, do we not find more evidence of cultural transmission? The calculations given above suggest that adoptive parent–offspring correlations in the range of 0.20 to 0.40 would be reasonable, yet with the possible exception of intelligence (Bouchard and McGue, 1981), such high correlations have not been reported in the adoption literature. There are many potential reasons for the discrepancy between the empirical observations and the theoretical conclusions, and in the face of such incongruence, the empirical observations must be favored. This gives two nonexclusive conclusions: (1) the path analytical model is wrong; and/or (2) the evolutionary model is wrong. Each of these possibilities is now discussed.

In terms of the evolutionary model, it is possible that stabilizing selection played a more important role than directional selection in the immediate evolutionary history of the traits most often examined in behavior genetic research. Once the population mean reaches the selective optimum, the implications of stabilizing selection can be clearly stated without abstruse algebra—mechanisms that reduce variance will be favored over those that increase variance. Because cultural transmission increases variance, it will be disadvantageous under pure stabilizing selection. But at the same time, so will other mechanisms that increase variance, such as assortative mating and sibling interactions. This leads to an intriguing prediction. If stabilizing selection had been (or is) important for a behavioral trait, then that trait should have little cultural
transmission, assortative mating, or any other psychological mechanism that increases phenotypic variance. It is interesting that some traits with small marital correlations (e.g., most personality traits) also have little vertical transmission, whereas other traits with moderate spousal correlations (e.g., intelligence) do have some adoptive parent–offspring resemblance. More rigorous empirical research is required to test whether this generalization holds over a wide variety of traits.

In terms of the path model, there may be several ways of improving the way cultural transmission is modeled. First, the models employed by Feldman and Cavalli-Sforza (1979), Rao et al. (1979), Rice et al. (1978), and Cloninger et al. (1979) model cultural transmission in terms of a latent variable that impinges upon parental phenotypes and is transmitted to an analogous latent variable in offspring. Such a model appears to have satisfactory properties in reaching equilibrium over time (see Cloninger et al., 1979; Rice et al., 1978). Behavior geneticists must consider alternative modes of cultural transmission.

Second, another method for improving path models comes from empirical research on primate imitation. It is indisputable from observation of human behavior that members of Homo sapiens are not constrained to imitate the behavior of only mother and father. Other conspecifics are also imitated. Perhaps a generalized mechanism for imitation evolved instead of a specific one for imitating one’s parents. In this case, we may write the environment of an offspring as a linear function of \( n \) cultural parents, \( n > 2 \), that include not only nuclear family parents but also a large number of other role models. Let \( p \) denote an \( (n \times 1) \) vector of phenotypic values and \( b \) denote an \( (n \times 1) \) vector of weights. Then the environmental value of an offspring may be written as

\[
e_{o} = b'p + \mu_{eo}
\]

In order for the phenotypic variance to equilibrate over time, the values in \( b \) must be small as \( n \) increases. For simplicity, let all the values in \( b \) be identical and let the correlation matrix for \( p \) be diagonal. Then the phenotypic variance of the offspring will be

\[
V = V_g + nb^2V_p + 2b \sum \text{cov}(g_{os}p_t) + V_u
\]

At equilibrium, \( V = V_p \) implying that the maximum value of \( b \) is \( (1/\sqrt{n}) \). Hence, if \( n \) numbers in the hundreds (as it may if role models in books, television, and movies can act as cultural parents), then the maximum adoptive–offspring correlation may be small.

Along similar lines, if each cultural model is weighted equally so that \( b = 1/n \), then the cultural transmission variance becomes \( V/n \) or the variance of a mean. Hence, cultural transmission variance will be small
as \( n \) increases. Again, this type of cultural transmission may be very important for moving the mean of a distribution in response to selection (either natural selection or cultural selection) but will contribute very little to variance within the distribution. This may be one reason why variables such as suicide, alcohol use, and affective disorder show both heritability and marked secular trends that are too large to be accounted for by a genetic response to natural selection.

Finally, cultural selection (or simple pragmatism) may dictate a selective range of the adult population to imitate. Let \( m^*_t \) and \( V^*_t \) denote the observed mean and variance in the parental generation after natural selection. Suppose that cultural parents encourage children to behave like a select sample of the adult population of survivors. Let \( e^* \) denote the mean of these adult role models and \( s^* \) denote their variance. The parameter \( s^* \) with respect to \( V_t \) may be viewed as the reciprocal of the intensity of this role model encouragement or importance of the behavior. Large values of \( s^* \) represent an unimportant behavior, whereas small values of \( s^* \) signify a behavior where departures from the norm of \( e^* \) are not desirable. The cultural transmission variance will be a direct function of \( s^* \). Hence, behaviors that are societally encouraged will be expected to have low cultural variance.

Other approaches to the evolution of vertical transmission have been explored by Cavalli-Sforza and Feldman (1981), Boyd and Richerson (1985), and Lumsden and Wilson (1981). Selective reception of culture, oblique and horizontal transmission, and biased observational learning are but a few examples that have never been examined from the behavioral genetic perspective. The present research generates no clear answers. But it does point to the fact that we must seriously consider the evolutionary development of the psychological mechanisms of cultural transmission that are embodied in our mathematical models.

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**REFERENCES**


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