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Multifactorial Inheritance with Cultural Transmission and Assortative Mating. I. Description and Basic Properties of the Unitary Models

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The familial transmission of complex developmental traits often appears to be influenced by the variation of many discrete genetic and environmental factors [1]. The transmission of such traits may be described and quantified by multifactorial models in which many factors of small effect play a role. Different formulations of polygenic or multifactorial models of continuous phenotypes or threshold characters have been described by several authors since the seminal work of Fisher [2] and are reviewed elsewhere [3, 4]. Although some formulations allow for additive environmental contributions, differences between polygenic and cultural inheritance have received little attention.

Environmental effects may sometimes simulate genetic transmission [5], but other patterns may also occur [6, 7]. Cavalli-Sforza and Feldman [6] noted that cultural inheritance may be effected directly by social learning and modeling or by parents teaching their offspring certain customs and preferences about diet, environment, and other activities. They emphasize the plasticity of genotypes in response to the environment and deal explicitly with the effect of parental phenotypes on the development of the same phenotype in the offspring. Cavalli-Sforza and Feldman recognize that other genetic and environmental factors besides the phenotype of the parent may influence the development of a child, but their model only deals with direct phenotype-to-phenotype effects.

In contrast, Morton [8] and Rao et al. [9, 10] describe linear models of familial resemblance which are primarily concerned with family members reared contemporaneously. Their models allow for environmental effects common to children reared together ("common environment") which may increase phenotypic resemblance. Their original model [9] also allows for a correlation between midparent-genotype and common environment but does not allow for direct cultural transmission from parent to offspring. Their later model [10] allows for some types of nongenetic transmission between generations at equilibrium, but the properties of such models have not been systematically described.

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To further our understanding of the transmission of complex developmental traits like height, weight, blood pressure, diabetes, hypercholesterolemia, intelligence, and psychiatric phenotypes, more comprehensive models are needed in which the effect of both polygenic and cultural inheritance are specified in pedigrees extended over several generations. In addition, the effects of nonrandom mating must be taken into account since such traits often show strong assortative mating [11–13].

In 1918, Fisher presented an extensive treatment of assortative mating for polygenic traits. An annotated account of Fisher's paper has been presented by Moran and Smith [14], and many of his results have been derived using a more elementary method by Crow and Felsenstein [15]. These treatments are only valid for polygenic traits without cultural transmission. Therefore, the extension of the multifactorial model to include cultural transmission requires a generalization of Fisher's treatment of assortative mating.

In this paper we will describe a general model of multifactorial inheritance with cultural transmission and assortative mating. For clarity we will distinguish among the *polygenic model* in which there is no cultural transmission, the *cultural model* in which there is no genetic transmission, and other *multifactorial models* in which both genetic and cultural transmission are possible. We allow for threshold effects, common environment, maternal and paternal effects, and assortative mating without selection. We follow the distinctions among assortative mating, selective mating, and inbreeding described by Lewontin et al. [16]. The effects of selective mating on the polygenic model have been considered by Wilson [17], and Wilson's critique of Fisher's model of assortative mating has been criticized elsewhere [18]. In our model and that of Fisher, gene frequencies do not change with assortative mating by definition. In addition, we assume that the overall frequencies of cultural factors relevant to the development of a trait do not change, rather only the distribution of these events between and within families is changed. A positive phenotypic correlation between mates induces a positive correlation between the transmissible cultural factors of the mates as well as between the genes relevant to the trait. This leads to an increase in the variance of the trait in the population which depends on the extent to which cultural factors are inherited.

We are concerned with both the cultural and the genetic factors which influence the transmission of a trait from parent to offspring since a priori assumptions about their relative contributions may obscure rather than enhance our understanding. It is necessary to provide a model which allows for both types of transmission simultaneously, rather than assume for convenience that cultural factors are negligible. However, for many traits there is insufficient data to estimate both heritability and the effect of cultural inheritance. Accordingly, we introduce the *unitary model* which does not try to further partition the transmissible variance, but which does allow for hypothesis testing and for approximate estimation of the combined importance of genetic and cultural factors.

DESCRIPTION OF THE MODELS

The following assumptions define the general multifactorial model and are more general than many prior formulations of this model: (1) A quantitative character P may

be partitioned as $P = A + B + E$, where A and B denote all the effects of genetic and cultural factors transmitted from parent to offspring, respectively, and E denotes all other effects which are usually random environmental influences, with the covariance (A,E) and covariance (B,E) equal to 0. (2) The transmissible factors act additively and each has small effect relative to the total phenotypic variance. (3) There are many transmissible factors so that P may be assumed to be normally distributed. We assume, further, that the joint distribution of A, B, and E is a trivariate normal. (4) In order to allow for both biological inheritance through DNA and cultural inheritance, no a priori assumptions are made about transmission probabilities or heritability.

In order to allow for assortative mating based on direct phenotypic preferences as in Fisher's first model ([14], page 50), we introduce two additional assumptions: (5) Assortative mating is recurrent (i.e., occurs every generation) and is based directly on phenotypic preferences. (6) The phenotypic correlation between mates is of constant magnitude through generations.

The causal system of transmission from parent to offspring for the general multifactorial model will be explicated in detail in the second paper in this series [19]. Briefly, the path coefficient from the additive genetic value (A) of a parent to the additive genetic value (A) of his offspring is 1/2 since the parent contributes precisely 1/2 of his genes. The path coefficient from the transmissible cultural value (B) of a parent to (B) of his offspring is denoted by β and will be discussed in detail below. The primary correlation m between the parental phenotypes induces correlations between the A and B of the parents. In addition, even if A and B within the same individual are uncorrelated in a random mating population, a nonzero correlation w is present at equilibrium if assortative mating occurs. This formulation of the multifactorial model is described by four parameters: $h^2 = V_A/V_P$, $b^2 = V_B/V_P$, β, and m, where V denotes the variance of the subscripted variable. Other parameters may be introduced to describe phenomena such as a common environment of rearing or a primary gene-culture covariance.

The correlations between relatives in terms of the parameters of the model can be determined by the use of path analysis [20–22] to provide a system of nonlinear equations for parameter estimation. To assess the effect of assortative mating on a random mating population, it is first necessary to compute the changes in variance of A and B which result from the assortative mating. The parameters at equilibrium can then be related to the random mating parameters, and the equilibrium correlations to the random mating ones. It is then possible to partition the equilibrium variance to determine the proportion attributable to assortative mating.

We will treat three submodels of the general model in what follows, and defer the analysis of the full model to a companion paper. The first submodel is the *cultural model*, which results if $V_A = 0$. The second is the usual *polygenic model* which results if $V_B = 0$. The third is termed the *pseudopolygenic model* and results when $\beta = 1/2$ in the general model. Each of the three models is characterized as unitary since transmission is determined by a single variable (viz. B, A, or $T = A + B$). Each of these models is in turn a special case of the *unitary model* which is defined below. In addition to their own usefulness, the development of these models is requisite for the general treatment.

DESCRIPTION OF THE CULTURAL MODEL

In the cultural model, we assume that $V_A = 0$, so that $P = B + E$ with $\text{cov}(B,E) = 0$. In a random mating population, the path coefficient β is equal to the correlation coefficient between the B of a parent and the B of his offspring. Once assortative mating occurs, the variance is increased in successive generations until equilibrium is reached. In the multiple regression equation of an offspring on his parents, it is assumed that the partial regression coefficient β' is unaffected by assortative mating:

$$B'_{n+1} = \beta' B'_{(M)n} + \beta' B'_{(F)n} + f'_n F'_n, \tag{1}$$

where M denotes the mother, F denotes the father, F'_n is the residual term corresponding to fluctuation about midparent cultural value, and f'_n is the partial regression coefficient for F'_n. It is shown later that in an equilibrium population the regression coefficient β' and the path coefficient β are equal. We use a prime ($'$) here to emphasize that these are the concrete (unstandardized) variables related by regression coefficients rather than path coefficients, although we assume, for convenience, that all variables have zero mean. The corresponding path equation is given by

$$B_{n+1} = \beta_n B_{(M)n} + \beta_n B_{(F)n} + f_n F_n, \tag{1.1}$$

where

$$\beta_n = \beta' \frac{\sigma_{B'n}}{\sigma_{B'n+1}} \text{ and } f_n = \frac{f'_n \sigma_{F'n}}{\sigma_{B'n+1}}.$$

Consider the path diagram displayed in figure 1 where n denotes the generation number, m the phenotypic correlation between mates, and r_{BBn} the correlation between the cultural transmissible factors of mates in generation n. For simplicity, uncorrelated residual terms are not shown since they do not contribute to familial resemblance. Although we assume that m is recurrent and of constant magnitude through generations, r_{BBn} depends on n because the ratio of the variance of transmissible factors V_{Bn} to the total phenotypic variance V_{Pn} changes with recurrent assortative mating until equilibrium is reached. Specifically, from figure 1, the correlation between the transmissible factors is $r_{BBn} = mb_n^2$.

Assumptions 5 and 6 may be changed if the correlation between mates is not based directly on the phenotype. Fisher's treatment of the relationship between m (Fisher's μ) and r_{BB} (the nongenetic analogue to Fisher's A) is valid even for cultural transmission. Only the case of direct phenotypic preference will be presented here, but other patterns are easily elaborated [19].

Changes in Variance Due to Assortative Mating

By taking the covariance of the right hand side of equation (1) with itself, and recalling that $\text{COV}(B'_{n+1}, B'_{n+1}) = V_{B'n+1}$, we have

$$V_{B'n+1} = \beta'^2 V_{B'(M)n} + \beta'^2 V_{B'(F)n} + 2\beta'^2 r_{B'B'n} \sigma_{B'(M)n} \sigma_{B'(F)n} + f'_n V_{Fn}'. \tag{2}$$

Consider first a random mating population at equilibrium denoted here as generation o, with $V_{B'n+1} = V_{B'(M)n} = V_{B'(F)n} = V_{B'o}$ and with $\text{COV}(B_{(M)n}, B_{(F)n}) = 0$. Equation (2) reduces to

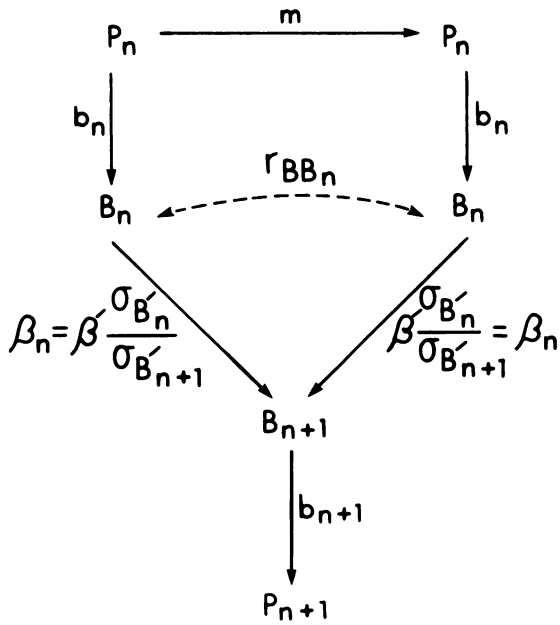

FIG. 1.—Parental determination of an offspring's phenotype for the cultural model. The generation number of the parents and the offspring are denoted by n and $n + 1$, respectively. The double-headed arrow indicates the correlation induced by the primary correlation m.

$$V_{B'_o} = 2\beta'^2 V_{B'_o} + f_o'^2 V_{F'_o}, \tag{2.1}$$

so that $f_o' \sigma_{F'_o} = (1 - 2\beta'^2)^{\frac{1}{2}} \sigma_{B'_o}$. (2.2)

Now suppose that assortative mating occurs and affects only the variance of transmissible factors (assumption 7), and specifically that $V_{F'_n} = V_{F'_o}$ and $f_n' = f_o'$ for all n, so that $f_n' \sigma_{F'_n} = f_o' \sigma_{F'_o}$. From equation (1) and (2.2), the change in the variance of transmissible factors from recurrent assortative mating is seen to follow the recursion relation

$$V_{B'_{n+1}} = 2\beta'^2 (1 + r_{B'B'_n}) V_{B'_n} + (1 - 2\beta'^2) V_{B'_o}. \tag{3}$$

Under recurrent assortative mating for a multifactorial trait, equilibrium is defined by the generations where $V_{B'_n} = V_{B'_{n+1}} = V_{B'}$. Quantities not indexed by generations are assumed to be at equilibrium under assortative mating, and quantities subscripted by o refer to their random mating values. At equilibrium, equation (3) yields

$$V_{B'} = \frac{1 - 2\beta'^2}{1 - 2\beta'^2 (1 + r_{B'B'})} V_{B'_o}. \tag{3.1}$$

When the assortative mating population is at equilibrium, the path coefficient β from the B of a parent to the B of his offspring is $\beta = \beta' (\sigma_{B'})/(\sigma_{B'}) = \beta'$. In the random mating population, $\beta_o = \beta' (\sigma_{B'_o})/(\sigma_{B'_o}) = \beta'$, and we have the remarkable fact that the path coefficient β is the same under both equilibrium conditions and equals the

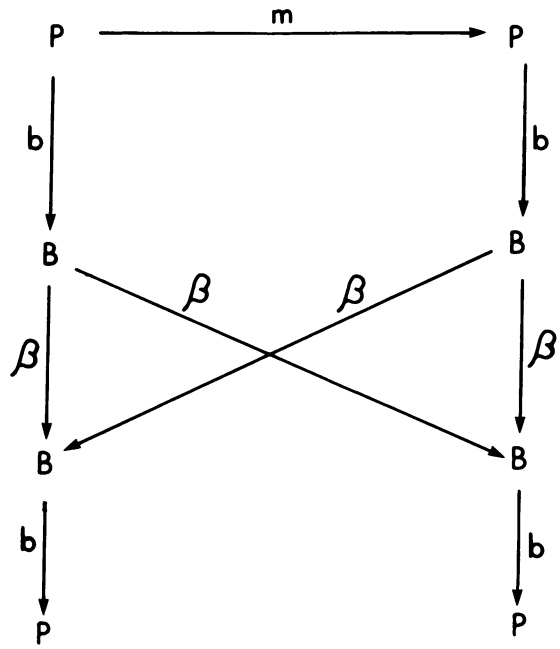

FIG. 2.—Path diagram depicting parent-offspring and sibling-sibling correlations at equilibrium for the cultural model.

partial regression coefficient β'. This is analogous to the fact that the path coefficient between the genic values of parent and offspring is 1/2 in an equilibrium population, whether it is mating randomly or assortatively ([22], p. 268). Since $\beta' = \beta$ and $r_{B'B'} = r_{BB}$, there will be no confusion in returning to the convention of representing both standardized and unstandardized quantities by the same symbol without the use of cumbersome primes.

The change in variance of transmissible factors, $\Delta V_{B'}$, depends on the sign of the correlation between mates, increasing if the correlation is positive and decreasing if the correlation is negative. This may be seen from equation (3.1) or (3.2), noting that $(1 + r_{BB})$ is always positive:

$$\frac{\Delta V_B}{V_{Bo}} = \frac{V_B}{V_{Bo}} - 1 = \frac{2\beta^2 r_{BB}}{1 - 2\beta^2 (1 + r_{BB})}. \tag{3.2}$$

Given that assortative mating only changes the variance of transmissible factors (assumption 7), and specifically that $V_E = V_{Eo} = V_{En}$, the phenotypic variance at equilibrium is seen to depend on β, r_{BB}, and V_{Po}:

$$V_P = V_B + V_E = V_{Po} + \Delta V_B = V_{Po} + \frac{2\beta^2 r_{BB} V_{Bo}}{1 - 2\beta^2 (1 + r_{BB})}. \tag{4}$$

Relationship between Parameters under Random and Assortative Mating

The path diagram in figure 2 depicts the relationship between mates, parent-offspring pairs, and siblings at equilibrium. Residual terms are omitted since they do not

contribute to familial resemblance. By path analysis, it can be shown that at equilibrium under assortative mating,

$$r_{BB} = mb^2, \tag{5}$$

where $b^2 = \dfrac{V_B}{V_P}$,

$$r_{po} = \beta(1 + m)\, b^2, \tag{6}$$

where r_{po} = parent-offspring correlation, and

$$r_{oo} = 2\beta^2 (1 + r_{BB})\, b^2, \tag{7}$$

where r_{oo} = correlation of two sibs each reared by the same parents. Under random mating, $m = 0$ and equations (6) and (7) reduce to

$$r_{po}{}^* = \beta b_o{}^2 \tag{6.1}$$

and

$$r_{oo}{}^* = 2\beta^2 b_o{}^2. \tag{7.1}$$

Equation (6) may be rearranged to give a useful expression for the proportion of the total variance due to cultural factors:

$$b^2 = \frac{r_{po}}{\beta (1 + m)}. \tag{8}$$

From equations (5) and (8), the correlation between transmissible factors of mates is

$$r_{BB} = \frac{m r_{po}}{\beta (1 + m)}. \tag{9}$$

Further, from straightforward applications of equations (3) through (9), the random mating parameters $r_{po}{}^*$, $r_{oo}{}^*$, and V_{Po} are given by

$$r_{po}{}^* = \frac{(1 + m) r_{po} (1 - 2\beta^2) - 2\beta m r_{po}{}^2}{(1 + m)^2 (1 - 2\beta^2) - 2m r_{po}{}^2}, \tag{10}$$

$$r_{oo}{}^* = 2\beta r_{po}{}^*, \text{ and} \tag{11}$$

$$V_{Po} = \left[1 - \frac{2m r^2{}_{po}}{(1 + m)^2 (1 - 2\beta^2)}\right] V_P. \tag{12}$$

These equations may in turn be solved for the assortative mating parameters at equilibrium, yielding

$$r_{po} = (1 + m) \frac{1 - 2\beta^2 - \{(1 - 2\beta^2)\,[1 - 2\beta^2 - 8m r_{po}{}^* (\beta - r_{po}{}^*)]\}^{\frac{1}{2}}}{4m (\beta - r_{po}{}^*)}, \tag{13}$$

$$r_{oo} = \frac{2\beta r_{po}}{1 + m}\left[1 + \frac{m r_{po}}{\beta(1 + m)}\right], \text{ and} \tag{14}$$

$$V_P = \left[\frac{(1 - 2\beta^2)(1 + m)^2}{(1 - 2\beta^2)(1 + m)^2 - 2m r_{po}{}^2}\right] V_{Po}. \tag{15}$$

If $r_{po}{}^* = \beta$, the expressions given in equations (13) to (15) simplify, and the

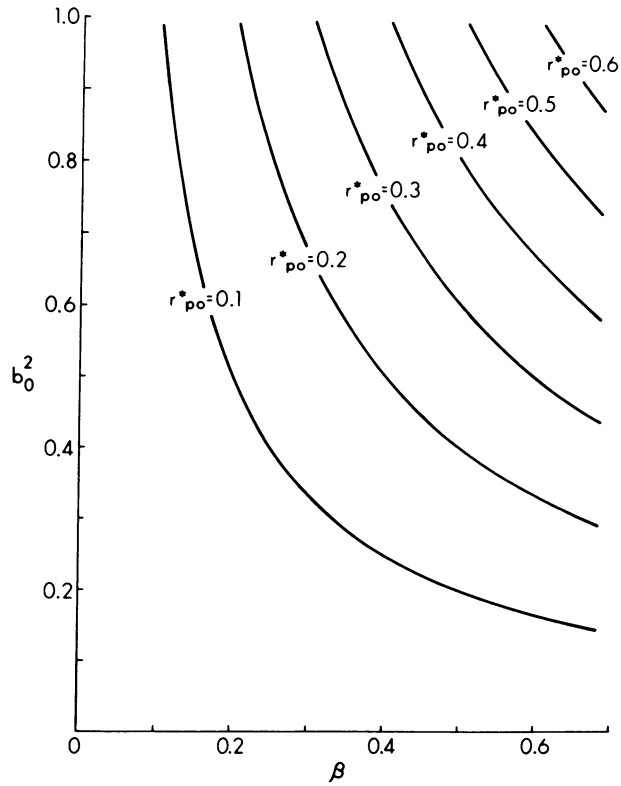

FIG. 3.—Relationship between β and b_o^2 in a random mating population for the indicated parent-offspring correlations.

parameters assume their maximal value:

$$r_{po} = \beta(1 + m), \tag{13.1}$$

$$r_{oo} = 2\beta^2(1 + m), \text{ and} \tag{14.1}$$

$$V_P = \frac{1 - 2\beta^2}{1 - 2\beta^2(1 + m)} V_{Po}. \tag{15.1}$$

From equation (6.1), it can be seen that $b_o = 1$ when $r_{po}^* = \beta$, so that $r_{BBn} = m$ for all n. Thus, the largest increases in parameter values will occur when β, m, and b_o^2 are high.

Properties of β

The relationship between β and b^2 is illustrated in terms of random mating parameters in figure 3 and in terms of assortative mating parameters in figure 4. The correlation m is fixed at .4, and the random mating values of β are shown for various values of r_{po} in a population at equilibrium. Equation (10) is first used to calculate r_{po}^* from β, r_{po} and $m = .4$, and then b^2 is found by dividing this value of r_{po}^* by β. Figures 3 and 4 demonstrate that for the cultural model the phenotypic parent-offspring

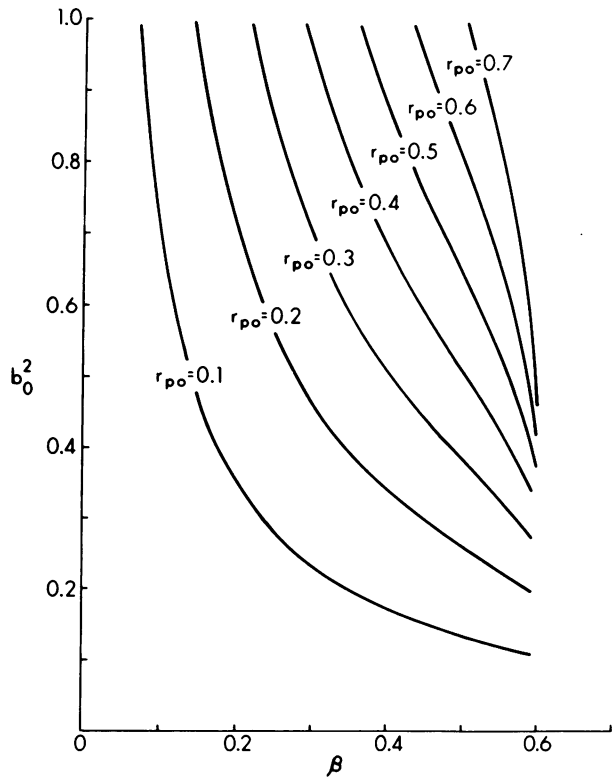

FIG. 4.—Relationship between β and b_o^2 in a population with $m = 0.4$ for the indicated parent-offspring correlations.

correlation may be a poor indicator of the importance of transmissible factors. If β is small, then r_{po} may be small with b^2 large, and if β is large, then r_{po} may be large with b^2 small. Indeed, the effectiveness of a parent as a teacher or model for social learning is a crucial consideration in the assessment of familial factors.

Although β is not fixed at 1/2 as is the path coefficient for the polygenic model, other considerations restrict the range of β and the correlation between parents and offspring.

In order for the value of the parent-offspring correlation in equation (13) to be real, the term under the radical must not be negative; that is, $(1 - 2\beta^2)\,[1 - 2\beta^2 - 8mr_{po}{}^*(\beta - r_{po}{}^*)] \geq 0$. Therefore, solving for the roots of β, an upper bound for $|\beta|$ is given by

$$|\beta| \leq -2m|r_{po}{}^*| + [1/2 + 4m|r_{po}{}^*|(1 + m)]^{\frac{1}{2}}. \tag{16}$$

A tedious computation shows that equation (16) is also equivalent to the constraints that

$$|r_{po}| \leq 2(1 + m)\,|r_{po}{}^*| \tag{16.1}$$

or

$$b^2 \leq 2b_o{}^2. \tag{16.2}$$

The requirement that the phenotypic and cultural variance reach a finite equilibrium point also places constraints on the magnitude of β. Rearranging equation (3.1), we observe that

$$\frac{V_{Bo}}{V_B} = \frac{1 - 2\beta^2(1 + mb^2)}{1 - 2\beta^2}. \tag{17}$$

Noting that the ratio of V_{Bo} to V_B becomes small and that b approaches 1 as V_B increases, we obtain the bound that

$$|\beta| < \frac{1}{\sqrt{2(1 + m)}}. \tag{18}$$

In the random mating population we must have $|\beta| \leq 1/\sqrt{2}$, so that values of $|\beta|$ greater than those given by either equations (16) or (18) or $1/\sqrt{2}$ are not possible. As β approaches this latter bound, the phenotypic variance approaches infinity. Thus, at the upper bound with $\beta = 1/\sqrt{2(1 + m)}$ and $b^2 = 1$, the parent-offspring correlation, obtained from equation (13.1) would be $[(1 + m)/2]^{\frac{1}{2}}$, a value much larger than those usually observed in practice. Also, if selection pressure prevented too large an increase in the phenotypic variance, it could reduce β in succeeding generations. With cultural inheritance β might vary from generation to generation as well as from family to family. Accordingly, if families with high β were selected against, β would decrease.

The Approach to Equilibrium

The approach to equilibrium under recurrent assortative mating depends on the magnitudes of β and m and is usually quite rapid for both cultural and biological inheritance. Equation (3) can be used to determine the number of generations required to reach equilibrium as shown in figure 5. In figure 5 the parameters m and r_{po} are fixed at .4 and .5, respectively, and equilibrium variances were chosen to be 1.0. The phenotypic variance through succeeding generations is plotted for various values of β with $n = 0$ denoting the random mating generation. Even with $\beta < 1/2$, equilibrium is closely approached within a few generations. Accordingly, assumption 6 (that the phenotypic correlation between mates is constant through generations) is not particularly restrictive.

Maternal and Paternal Effects

In the presence of cultural inheritance, β need not be the same for each parent. In many contemporary cultures the mother tends to have a greater responsibility in child rearing so that the correlation between transmissible factors may be greater between mother and child than between father and child. In some traits, such as birth weight, uterine environment may play a mediating role in the transmission of the trait and separate correlations between mother and child (r_{MO}) and between father and child (r_{FO}) are needed. Accordingly, let β_M and β_F denote the mother-child and father-child path coefficient between transmissible factors, respectively.

Equation (1) then becomes

$$B_{n+1} = \beta_M B_{(M)n} + \beta_F B_{(F)n} + (1 - \beta_M{}^2 - \beta_F{}^2)^{\frac{1}{2}} (\sigma_{Bo}/\sigma_{Fo}) F_n, \tag{19}$$

so that

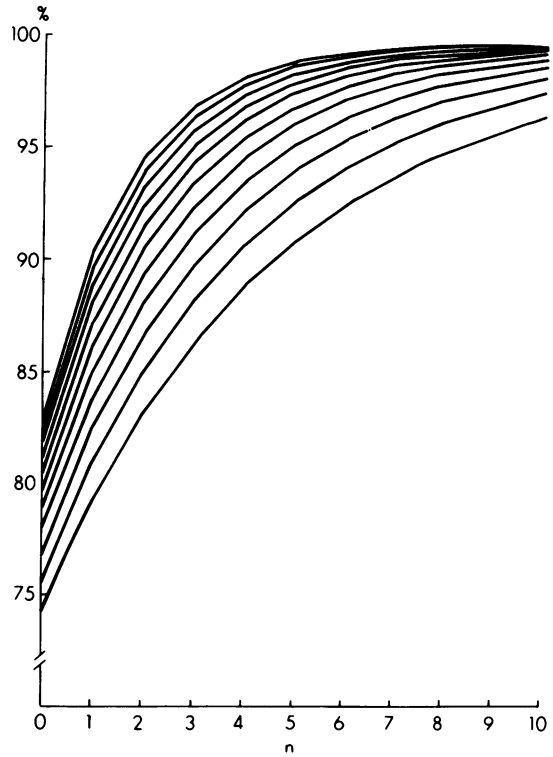

FIG. 5.—Approach to equilibrium through 10 generations expressed in terms of percentage of the final equilibrium value of the population variance. m is fixed at .4, r_{po} is fixed at .5, and β varies from .45 for the top curve to .55 for the bottom one.

$$V_B = \frac{1 - \beta_M^2 - \beta_F^2}{1 - (\beta_M^2 + \beta_F^2 + 2\beta_M\beta_F r_{BB})} V_{Bo}, \tag{20}$$

and

$$V_P = V_{Po} + \frac{(\beta_M^2 + \beta_F^2)\,\sigma_B}{1 - (\beta_M^2 + \beta_F^2 + 2\beta_M\beta_F r_{BB})} V_{Bo}. \tag{21}$$

In the random mating population, the father-offspring correlation r_{Fo}* is equal to $\beta_F b_o^2$, and the mother-offspring correlation is $\beta_M b_o^2$. Other formulae may be easily modified by substituting $(\beta_M + \beta_F)$ for 2β, $(\beta_M^2 + \beta_F^2)$ for $2\beta^2$, and $(\beta_M^2 + \beta_F^2 + 2\beta_M\beta_F r_{BB})$ for $2\beta^2(1 + r_{BB})$. Additional extensions in which β depends on both the sex of the parent and the sex of the child, as suggested by some for juvenile delinquency [23], are easily elaborated.

THE POLYGENIC MODEL

The special case of the general multifactorial model in which all transmissible factors are additive genes is termed the polygenic model. In this case, the above derivations are valid with β fixed at 1/2 and B replaced by A.

In this setting, the regression of an offspring on his parents which is given in equation (1) becomes

$$A_{n+1} = \tfrac{1}{2}A_n + \tfrac{1}{2}A_n + \frac{\sqrt{2}}{2}(\sigma_{Ao} / \sigma_S) S, \tag{22}$$

where S is the segregation from mid-parent genotype. Similarly, setting $\beta = 1/2$ in equations (3.1) and (4), the variances under assortative mating at equilibrium are

$$V_A = \frac{V_{Ao}}{1 - r_{AA}}, \tag{23}$$

and

$$V_P = V_{Po} + \frac{r_{AA}}{1 - r_{AA}} V_{Ao}. \tag{24}$$

These expressions agree with equations (4.8.11) and (4.9.6) of Crow and Kimura [24] with a large number of effective genes. Their derivation depends heavily upon the assumption of polygenic inheritance, whereas in the preceding derivation, the only genetic assumption required is that the path coefficient from A of a parent to A of his offspring is 1/2.

THE PSEUDOPOLYGENIC MODEL

Now consider the special case of the general model with $\beta = 1/2$. Equations (1) and (22) may be combined to yield

$$Q_{n+1} = \tfrac{1}{2}Q_n + \tfrac{1}{2}Q_n + \tfrac{1}{2}\sqrt{2}\,\sigma_{Qo} F', \tag{25}$$

where F' has variance 1, and Q_i is given by $Q_i = A_i + B_i$. The earlier arguments may be again applied to show

$$V_Q = \frac{V_{Qo}}{1 - r_{QQ}}, \text{ and} \tag{26}$$

$$V_P = V_{Po} + \frac{r_{QQ}}{1 - r_{QQ}} V_{Qo}, \tag{27}$$

where

$$q^2 = \frac{V_Q}{V_P}, \text{ and} \tag{28}$$

$$r_{QQ} = mq^2. \tag{29}$$

In the general case we could define

$$T_{n+1} = A_{n+1} + B_{n+1} \tag{30}$$

$$= \tfrac{1}{2}A_{(M)n} + \tfrac{1}{2}A_{(F)n} + \beta B_{(M)n} + \beta B_{(F)n} + f'F', \tag{30.1}$$

but for $\beta \neq 1/2$, the analogue of equation (25) does not hold, so that genetic and cultural factors must be considered separately [19]. The case when $\beta = 1/2$ is termed the pseudopolygenic model since all correlations between relatives in intact families (except the correlation between monozygotic twins) may be expressed in terms of m and q^2. As a result, b^2 and h^2 cannot be estimated separately unless separation data are

present. Since the pseudopolygenic model mimics genetic inheritance in intact families, the a priori assumption of a polygenic model is untestable. Techniques, such as those for the resolution of heterogeneity described by Reich et al. [25–27], which are valid for the pseudopolygenic model, are therefore desirable.

THE UNITARY MODEL

To analyze each of the above models in a uniform way, we introduce a new model, the unitary model, where we assume $P = T + E$, and that the regression of an offspring on his parents is given by

$$T_{n+1} = \tau T_{(M)n} + \tau T_{(F)n} + \sqrt{1 - 2\tau^2}\,(\sigma_{To}/\sigma_{Fo})\,\mathrm{F} \tag{31}$$

so that

$$V_T = \frac{(1 - 2\tau^2)V_{To}}{1 - 2\tau^2\,(1 + r_{TT})}, \text{ and} \tag{32}$$

$$V_P = V_{Po} + \frac{2\tau^2 r_{TT} V_{To}}{1 - 2\tau^2\,(1 + r_{TT})}, \tag{33}$$

where

$$t^2 = \frac{V_T}{V_P}, \text{ and} \tag{34}$$

$$r_{TT} = mt^2. \tag{35}$$

Submodels of the unitary model include: (1) $\tau = \beta, T = B$ (the cultural model); (2) $\tau = 1/2, T = A$ (the polygenic model); and (3) $\tau = 1/2, T = A + B$ (the pseudopolygenic model).

We have:

$$r_{po} = \tau(1 + m)\,t^2, \tag{36}$$

$$r_{oo} = 2\tau^2(1 + r_{TT})t^2, \tag{37}$$

$$r_{po}{}^* = \frac{(1 + m)r_{po}\,(1 - 2\tau^2) - 2\tau m r_{po}{}^2}{(1 + m)^2\,(1 - 2\tau^2) - 2m r_{po}{}^2}, \tag{38}$$

$$r_{oo}{}^* = 2\tau r_{po}{}^*, \tag{39}$$

$$V_{Po} = \left[1 - \frac{2m\,r_{po}{}^2}{(1 + m)^2\,(1 - 2\tau^2)}\right] V_P, \tag{40}$$

$$r_{po} = (1+m)\left(\frac{1 - 2\tau^2 - \{(1 - 2\tau^2)\,[1 - 2\tau^2 - 8m r_{po}{}^*(\tau - r_{po}{}^*)]\}^{\frac{1}{2}}}{4m\,(\tau - r_{po}{}^*)}\right), \tag{41}$$

$$r_{oo} = \frac{2\tau r_{po}}{1 + m}\left[1 + \frac{m r_{po}}{\tau(1 + m)}\right], \text{ and} \tag{42}$$

$$V_P = \left[\frac{(1 - 2\tau^2)\,(1 + m)^2}{(1 - 2\tau^2)\,(1 + m)^2 - 2m r_{po}{}^2}\right] V_{Po}. \tag{43}$$

EXTENSIONS OF THE UNITARY MODEL

Common Environment

It follows from equations (36) and (37) that if τ is equal to 1/2 (as in the polygenic or pseudopolygenic model) or is less than 1/2 (as may occur with cultural inheritance), then full sibling pairs cannot be more similar than parent-offspring pairs. However, r_{oo} is often observed to be greater than r_{po}. From the polygenic model the increased similarity of full sibs is often attributed to the effects of dominance deviation [24] since this increases r_{oo} and not r_{po}. However, Morton [8] describes the notion of dominance deviation for polygenes as "farfetched" and cites data showing that dominance decreases with small gene effect. Many early investigators did not allow for the resemblance between sibs to be due in part to the similarity of their shared environment and large dominance effects were postulated (e.g., in 1918 Fisher estimated the dominance variance for height as 25%). In studying environmentally influenced traits in humans where environments cannot be experimentally randomized, the effect of common or shared environmental influences cannot be ignored. Rao et al. [28] found correlations for height and weight to decline from .572 and .547 for dizygous twins to .127 and .120 for other full sibs born 20 years apart and noted a steady decline related to age differences. Genetic factors cannot explain such effects, whereas common environment does seem to offer a plausible explanation for this. Some formulations incorporate a variable "common environment" with a path to each sibling [8, 9, 22]. We introduce a similar but slightly more flexible model which minimizes the number of assumptions involved.

In the path diagram depicted in figure 6, we introduce a correlation c between the relevant environmental influences to which siblings are exposed. Several possible paths which could induce such a correlation are indicated by dotted paths but only the correlation c is retained. The nature of the correlation is ambiguous in this formulation, but it is seldom practical to obtain the data necessary to distinguish between the alternative interpretations unless c and e are large. It should be noted that this formulation also implies that there is no change in variance due to environmental factors with assortative mating, so that c remains unchanged, although the standardized regression coefficient e decreases as V_P increases with assortative mating.

In the random mating populations the correlation between full siblings is

$$r_{oo}^* = 2\tau^2 t_o^2 + c e_o^2, \tag{44}$$

so that

$$c = \left[r_{oo} - 2\tau \left(1 + \frac{m r_{po}}{(1+m)} \right) \frac{r_{po}}{1+m} \right] \frac{1}{e^2}. \tag{45}$$

It should be noted that c may differ in different groups such as monozygous twins, dizygous twins, and full sibs. Furthermore, being reared apart does not assure that $c = 0$. Estimates of c may be made directly by use of an index of the relevant environment as suggested by Morton and Rao et al. [8–10], or estimated using observed correlations between relatives.

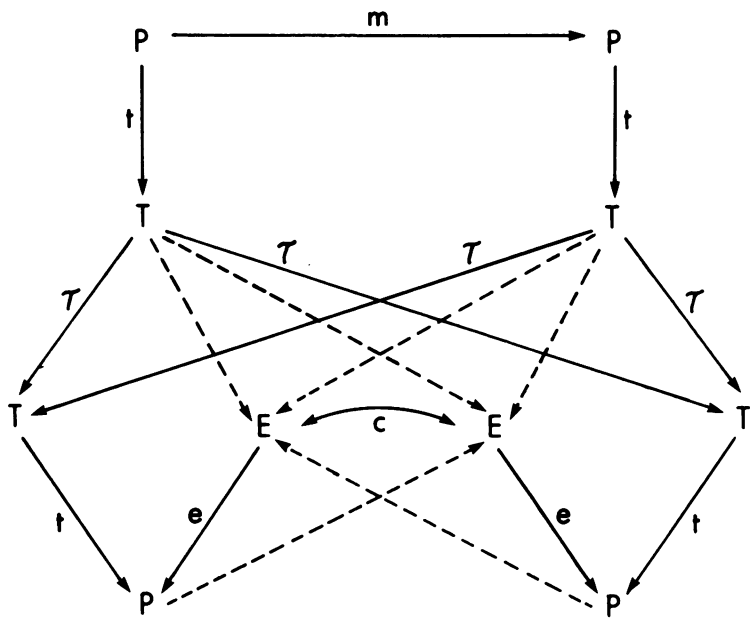

FIG. 6.—Path diagram depicting sibling-sibling correlation when a correlation between nontransmissible environmental values is present. Dashed lines = possible sources of the correlation between the E's.

Threshold Characters

The multifactorial model for threshold characters has been described by Crittenden [29], Falconer [30], Reich et al. [25–27], and Curnow and Smith [4]. Here it is assumed that the manifestation of a dichotomous trait is determined by a normally distributed variable X, termed the liability to develop the trait, with $X = T + E$; that is, transmission of liability from parent to offspring is described by the unitary model. It is further assumed that affectational status is determined by a threshold so that an individual is "affected" (i.e., manifests a trait, if his score on X is above the threshold and "unaffected" otherwise).

The correlations between individuals are then the correlations between the underlying liability scale X and may be estimated by the tetrachoric correlation coefficient [25, 26]. The normal deviate X_p of the threshold may be estimated from the population prevalence K_p.

The random mating correlations may be obtained from equations (38) and (39), and the random mating prevalence may be obtained from the change in variance given by equation (40). Letting $X_p{}^*$ denote the normal deviate for the random mating prevalence $K_p{}^*$ we have

$$X_p{}^* = X_p \left[\frac{(1 - 2\tau^2)(1 + m)^2}{(1 - 2\tau^2)(1 + m)^2 - 2mr_{po}{}^2} \right]^{\frac{1}{2}} \tag{46}$$

The value of $K_p{}^*$ can then be obtained from the complementary distribution function of the unit normal random variable evaluated at $X_p{}^*$.

The increase in population prevalence due to assortative mating is illustrated in

figure 7 with K_p* equal to 1% and m equal to .4. The maximum value of τ is given by equation (16) for r_{po}* less than .2988 and by equation (18) for r_{po}* greater than .2988. In the latter case, the curves approach the asymptote determined by equation (18) corresponding to an infinite increase in the variance. Figure 7 shows that the increase in K_p is small for small τ, increases monotonically with τ, and can be quite substantial for moderately large τ. In the case of the polygenic or pseudopolygenic models τ is .5.

RESEMBLANCE BETWEEN OTHER RELATIVES

Information about first-degree relatives and mates in one population is insufficient to estimate τ. Accordingly, information about multiple classes of relatives or heterogeneous populations is needed in order to estimate the parameters of the unitary model. Estimation of τ and the correlation between relatives will be described for the ancestors and descendants of parents-offspring (vertical relatives) and of full sibs (full collateral relatives).

Vertical Relatives

The path diagram for the grandparent-grandchild correlation r_{go} is shown in figure 8. The correlation is

$$r_{go} = \tau^2(1 + m)\,(1 + r_{TT})t^2. \tag{47}$$

In general, the correlation between ancestors and descendants of parent-offspring pairs after n additional generations is given by the formula

$$r_{Vn} = (r_{po})\tau^{n-1}\,(1 + r_{TT})^{n-1} = \tau^n(1 + m)\,(1 + r_{TT})^{n-1}t^2 \tag{48}$$

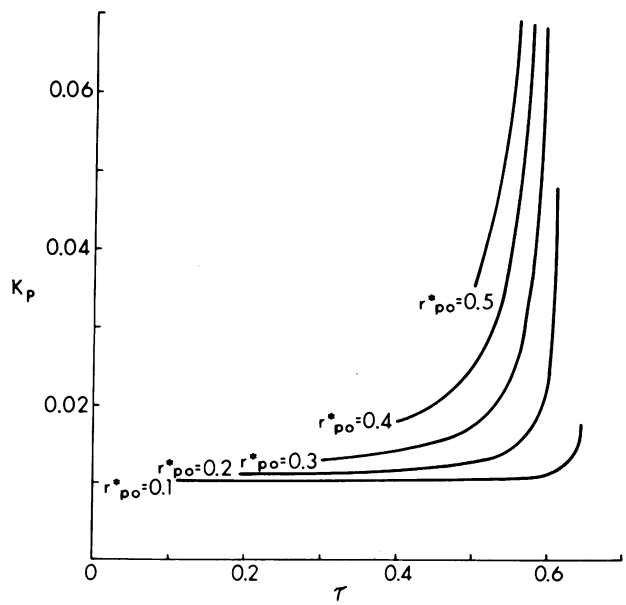

FIG. 7.—Increase in the population prevalence K_p in terms of τ for a threshold character with a random mating prevalence of 1%, m = .4 and the indicated values of r_{po}*.

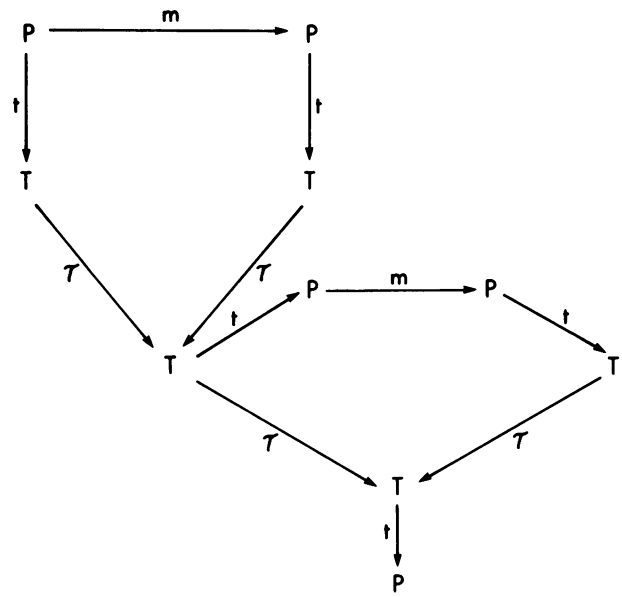

FIG. 8.—Path diagram depicting grandparent-grandchild correlation.

where $r_{V1} = r_{po}$, $r_{V2} = r_{go}$, r_{V3} is the great-grandparent correlation, etc. Note that if τ is negative, values of the trait will oscillate, and for a dichotomous trait, may appear to "skip generations."

Recalling equations (35) and (36), rearrangement of equation (48) gives a useful general expression for τ when $n > 1$,

$$\tau = \left(\frac{r_{Vn}}{r_{po}}\right)^{1/n-1} - \frac{mr_{po}}{1+m}. \tag{49}$$

Full Collateral Relatives

Similar formulae may be developed for descendants of full sibs. The path diagram for first-cousins and for uncles (aunts) and nephews (nieces) allowing for the common environment of the siblings is shown in figure 9. The correlation between uncle (aunt) and nephew (niece) is

$$r_{C2} = r_{U/N} = 2\tau^3(1 + r_{TT})^2t^2 + ce^2m\tau t^2, \tag{50}$$

and the correlation between first-cousins is

$$r_{C3} = 2\tau^4(1 + r_{TT})^3t^2 + ce^2m^2\tau^2t^4. \tag{51}$$

The contribution of common environment to the resemblance of descendants of full sibs may require some comment. The effect of environmental factors E are not transmissible by definition; nongenetic factors which are transmitted between generations are included in T. However, the correlation between the home environments of sibs induces a greater correlation between the transmissible values of the mates of a

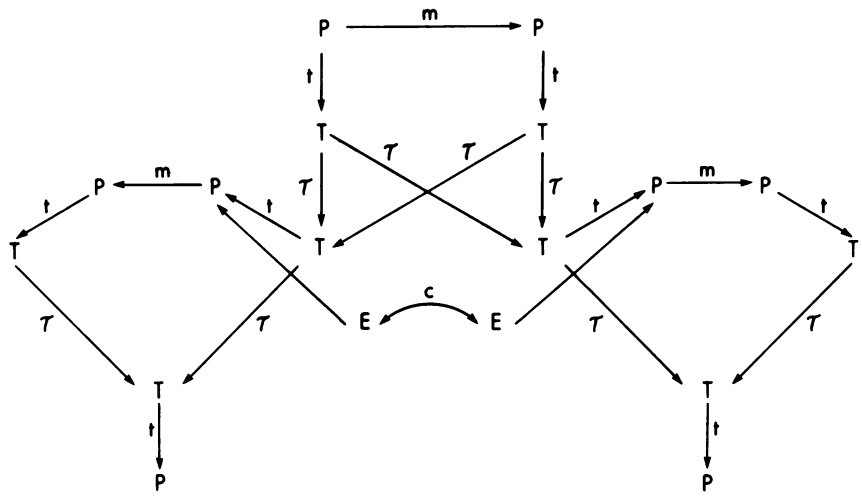

FIG. 9.—Path diagram depicting the first-cousin and uncle-nephew correlation.

pair of sibs than would be present otherwise. This increased resemblance is of course transmitted, as shown in figure 9, and is so specified in the formulae here. Regardless, the influence is small except when c, e, m, τ, and t are all high. When common environment is negligible, the correlation between collateral relatives in intact families is given in general as

$$r_{Cn} = 2\tau^{n+1}(1 + r_{TT})^n t^2 \tag{52}$$

where n is the degree of genetic relationship so that $n = 1$ for full sibs, 2 for uncle (aunt) and nephew (niece), 3 for first cousins or great uncle/nephew, etc. From equation (52), an estimate of τ similar to that obtained with vertical relatives is given by

$$\tau = \left[\frac{r_{Cn}(1 + m)}{2r_{po}}\right]^{1/n} - \frac{mr_{po}}{1 + m}. \tag{53}$$

However, if common environment is not negligible, distinctions are necessary between collateral pairs in which they are descendants of only one or each of two sibs (e.g., great uncle vs. first cousin). In general, the correlation between an individual and the descendants of his full sib is

$$r_{Cn} = \{2\tau^{n+1}(1 + r_{TT})^n t^2 + ce^2[\tau^{n-1}mt^2(1 + r_{TT})^{n-2}]\} \tag{52.1}$$

where n, the degree of genetic relationship, is 2 or greater so that $C1$ denotes full sibs, $C2$ denotes uncle and nephew, $C3$ denotes great uncle, etc. In contrast, the correlation between descendants of each of a pair of full sibs is given as

$$r_{Cn} = \{2\tau^{n+1}(1 + r_{TT})^n t^2 + ce^2[\tau^{n-1}m^2t^4(1 + r_{TT})^{n-3}]\} \tag{52.2}$$

where n, the degree of genetic relationship, is 3 or greater so that $C1$ and $C2$ are the same as in (52.1), but $C3$ denotes first cousins, $C4$ first cousins once removed, $C5$ second cousins, etc.

PROPERTIES OF THE UNITARY MODEL

The unitary model may be viewed as an approximation to the general model of combined polygenic and cultural inheritance which is defined in the beginning of this paper and developed fully elsewhere by Cloninger et al. [19]. We know that the two models are equivalent in the three special cases treated above: the cultural model, the polygenic model, and the pseudopolygenic model. The general model is parameterized by β, m, $b^2 = V_B/V_p$, $h^2 = V_A/V_p$ and c, and V_T is given by $V_T = b^2 + h^2 + 2wbh$, where w is the correlation between A and B within the same individual.

A natural question to address is how β and V_T of the general model relate to τ and V_T of the unitary model. We are investigating this question by generating observations under the general model and then fitting the unitary model to the simulated data. The results of one such simulation are shown in figures 10 and 11.

The parameters of the full model were chosen to be $V_T = .8$, $m = .3$, $c = .0$, together with the indicated values of β. Sets of correlations (specifically, parent-offspring, grandparent-grandchild, sibling, uncle, nephew, first and second cousin, and first and second cousins once removed) were generated for b^2 from 0 to .8 in increments of .1 and then fitted to the unitary model. As noted in figure 10, V_T was underestimated for all data sets (except for those where we know the answer is exact). To date, the claim that V_T obtained from the unitary model is conservative has been borne out by our simulations. It appears that V_T is underestimated by approximately $2wbh$, and other simulations have shown that V_T is very close to the true values when m is small.

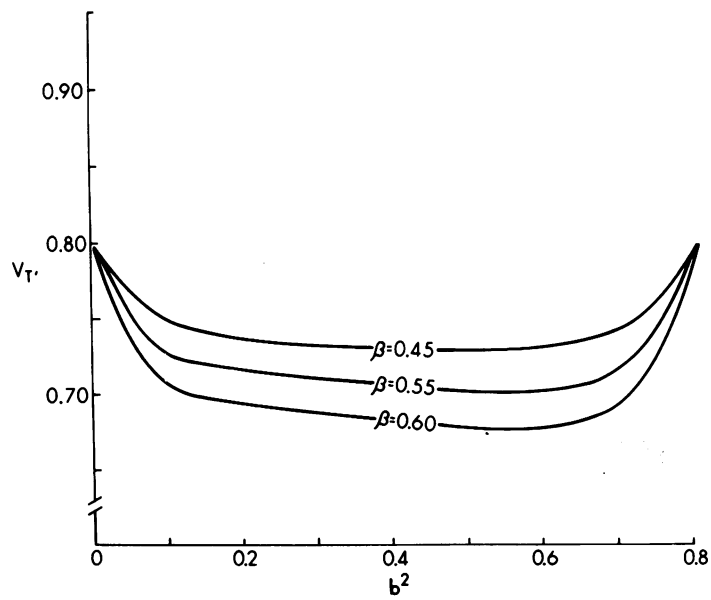

FIG. 10.—Values of the total transmissible variance V_T of the unitary model at the point of best fit to data simulated under the full model. The true V_T was .8 and data sets were generated with $b^2 = .1$ to $b^2 = .8$, with $h^2 = .8 - b^2 - 2bhw$ and with the indicated values of β. w is defined in reference [19].

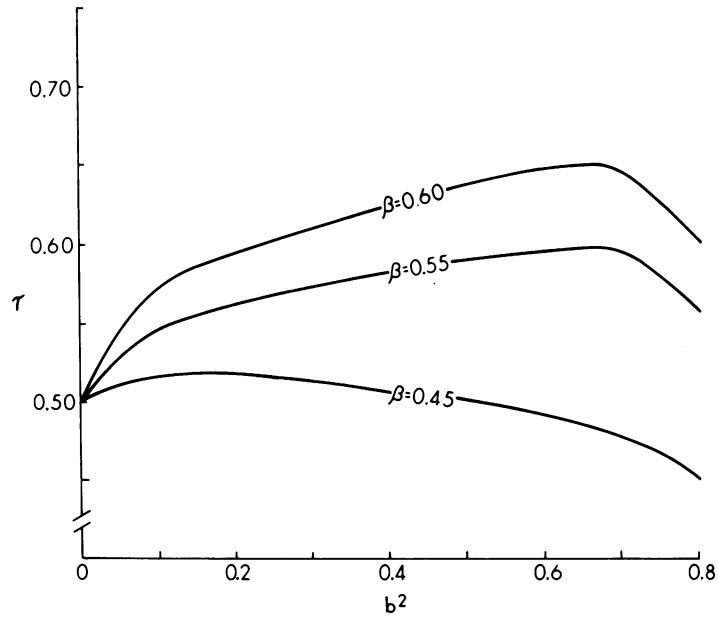

FIG. 11.—Values of the parameter τ in the unitary model at the point of best fit for the same data sets as in figure 10.

The relationship between τ and β is more complicated, as indicated in figure 11. When $V_B = .8$, we know $\tau = \beta$, and when $V_B = 0$, we know $\tau = .5$. Depending upon the relative proportions of V_A and V_B, τ may be an overestimate or underestimate of β. However, when m is 0, a good approximation of τ is given by $\tau = .5\ (V_A)/(V_T) + \beta\ (V_B)/(V_T)$. (54)

Finally, the observation that $\tau = .5$ when $V_B = 0$ allows the testing of the hypothesis that there is no cultural transmission. If $\tau \neq .5$, then V_B cannot be zero. However, as indicated by the pseudopolygenic model, if τ is 1/2, we cannot conclude the V_A is nonzero. The most important point to note is that the test of $V_B = 0$ suggested here is more powerful in detecting cultural factors than the usual chi-square goodness of fit test against an unspecified alternative which is often used to test the fit of the polygenic model to a data set.

APPLICATIONS

The unitary model contains four parameters (viz. τ, m, t, and c), so that estimates of at least four correlations are necessary for parameter estimation. A FORTRAN program TAU, available upon request, is used to provide maximum likelihood estimates of the parameters from reported correlations. This program applies the inverse hyperbolic tangent transformation, $f(x) = \frac{1}{2} \ln (1 + x)/(1 - x)$, to each product moment estimator $\hat{r}$, and ignoring the bias $r/2(N - 1)$, it assumes

$$f(\hat{r}) \sim \mathbf{N}\left(f(r), \frac{1}{N - 3}\right), \tag{55}$$

where N is the number of pairs used to estimate the correlation r.

Given observations $\bar{r}_1, \bar{r}_2, \ldots \bar{r}_k$ and parameter values $\underline{\theta}$, the log likelihood of the data set is given by

$$L = \tfrac{1}{2} \sum_{i=1}^{k} \{f(\bar{r}_i) - f[r_i(\underline{\theta})]\}^2 (N_i - 3), \tag{56}$$

where $r_i(\underline{\theta})$ is the correlation predicted from the model. The general purpose likelihood searching program MAXLIK [31] is then used to obtain maximum likelihood estimates of the parameters $\underline{\theta}$ by maximizing L. At the point of best fit, note that $2L \sim \chi^2_\nu$, where ν is k minus the number of estimated parameters.

Testing of hypotheses involving linear constraints on the parameters may then be performed using a likelihood ratio test [32] by first obtaining the likelihood L_1, without any constraints, and then obtaining the likelihood L_o by imposing the constraints. The asymptotic distribution of

$$-2\ln\left(\frac{L_o}{L_1}\right) = -2(\ln L_o - \ln L_1) \tag{57}$$

is known to be a chi-square distribution with ν degrees of freedom when ν linear constraints are used.

Unfortunately, a paucity of data is reported on remote relatives. This no doubt reflects the fact that under the polygenic model only estimates of m, r_{po}, and r_{oo} are necessary to estimate heritability and dominance deviation. Information on other classes of relatives is not necessary and statistically less efficient. However, once nonrandom environmental factors are considered, such data are not adequate.

Human Stature

Although the necessary correlations are not available to apply this method directly, the two data sets displayed in table 1 reflect different degrees of assortative mating and allow the estimation of τ and t^2 from the two parent-offspring correlations. The first data set is that collected by Pearson and Lee [33] from the population of English university students and was analyzed by Fisher [2], who estimated the random mating

TABLE 1

REPORTED CORRELATIONS FOR HUMAN STATURE FROM TWO DATA SETS ANALYZED

	PARAMETER	OBSERVED	NO.	PREDICTED $\tau = .604, t_o^2 = .484$	PREDICTED $\tau = .5, t_o^2 = .7281$
Pearson and Lee [33] ...	m	.2804	1,000	.2835	.2953
	r_{po}	.5066	4,886	.5064	.5029
Galton [34]	m	.0931	200	.0848	.0070
	r_{po}	.3355	937	.3379	.3671

NOTE.—Predicted correlations at the points of best fit for the unitary model and the unitary model with $\tau = 0.5$ are displayed in the last two columns.

heritability to be 75% and the dominance deviation to be 25% of the variance. The second data set consists of 200 families collected from the upper middle class by Galton [34], the correlations being those reported by Pearson [35] with the sexes combined. The correlation m was found to be .2804 in the first data set and .0931 in the second. Assuming that t_o^2 and τ would be the same for each population, the two parent-offspring correlations allow estimation of the random mating parameters using equation (41). This was done using a maximum likelihood search.

The values of the correlations at the point of best fit for both data sets are displayed in table 1 when τ and t_o^2 are estimated simultaneously. When τ was fixed at .5, t_o^2 was estimated to be .7281, a value consistent with Fisher's analysis of Pearson and Lee's data. This value contrasts with the value of $t_o^2 = .484$ from the unitary model. Thus, the unitary model supports the hypothesis that familial factors are less important than previously indicated.

The likelihood ratio test of $\tau = .5$ is not significant ($\chi^2_1 = 3.04$), despite the difference in the two parameter sets and the large sample sizes. This results from the two population method used, since estimation is only possible because of the differential effect on the change of variance in the two populations. (In a random mating population $r_{po}^* = \tau t_o^2$, so that if two random mating populations were sampled, the two parameters could not be estimated.) For this reason, we do not recommend this method, but present it only to indicate that the existing data may be described adequately with our model and that the conclusion that transmission of human stature is in toto genetic should be investigated further.

The observed sibling correlations are .5433 and .4004 for the two data sets, and using $\tau = .604$, $t_o^2 = .484$, and $c = 0$, the predicted correlations are .5651 and .3929, respectively, so that common environmental effects (or dominance) would be negligible. For $\tau = .5$, the predicted correlations with $c = 0$ are .4773 and .3664, respectively, so that common environment (or dominance) would be required to explain the sibling correlations.

DISCUSSION

Until recently, human genetics has paid little attention to the importance of nonrandom environment in the etiology or the transmission of quantitative phenotypes. Current investigations support the observation that many behavioral traits are highly familial and are suggestive of parent to offspring transmission. However, as the pseudopolygenic model indicates, such observations cannot be construed as proof for genetic transmission when observations are made only within intact families. It is necessary to allow for both genetic and cultural transmission, as well as a systematic effect due to a shared environment of rearing, before an observed pattern of familial correlations may be used to determine the mode of transmission of a complex trait.

The cultural model we propose is patterned after the polygenic model. Transmission from parent to offspring is determined by the additive effect of many cultural events, with the degree of parental determination measured by β. However, for β greater than .5, this model differs in two qualitative aspects from the polygenic model. First, the coefficient $2\beta^2$ in the sibling correlation is greater than the corresponding β in the parent-offspring correlation, so that r_{oo}^* is greater than r_{po}^*, and even when assortative

mating occurs, r_{oo} can be greater than or equal to r_{po}. For many traits, the observed sibling correlations are "too large," so that, in contrast to the cultural model, the polygenic model requires either a large dominance deviation or a large nontransmissible common environmental effect to explain the data.

Second, the cultural model predicts that second and third degree relatives are more similar than would be expected under the polygenic model. The observation that distant relatives are "too alike" is suggestive of cultural transmission, and neither dominance nor nontransmissible common environment can offer a satisfactory explanation. On the other hand, where β is less than .5, transmissible factors may play a major role in the etiology of the trait even though reported correlations are low.

Our treatment of assortative mating is general in that the derivations are valid even for cultural transmission. In the second paper of this series, these results will be used to include assortative mating for the general multifactorial model in the case of direct phenotypic assortment. The implementation of other types of assortative mating, such as indirect phenotypic assortment or social homogamy, can easily be accomplished by paralleling arguments used for the polygenic model together with our results which allow for cultural transmission. When threshold characters are considered, another possibility is that assortment is for the dichotomous phenotypes rather than for the underlying liability values. We are currently investigating the differences in these two different approaches.

One further question requires some comment. If the hypothesis that $V_B = 0$ is rejected, what is the minimum V_B that can explain the data? One answer to this can be obtained by fixing β in the general model [19] at its maximum value to provide a lower bound for V_B. The precision of such an estimate could itself then be tested with further simulation.

The unitary model we propose provides a common model to parameterize the cultural, the polygenic, and the pseudopolygenic models and provides an approximation to the general model. Further simulations are needed to evaluate the operating characteristics of this model more fully, but our work to date strongly supports the usefulness of this model when observations are available only for relatives reared within intact nuclear families.

The unitary model provides a framework to investigate many important questions concerning the transmission of a trait without requiring an estimate of heritability. In particular, issues such as index construction, the detection of relevant environments, the detection of heterogeneous subforms of a trait, and the detection of a single major locus may be approached within the context of this model. Observations within intact families are easily collected and studies may be replicated to test hypotheses generated in an a posteriori manner. This is in contrast to separation data where observations made on certain types of relatives, for example MZ twins reared apart, are rare and essentially nonreplicable.

Furthermore, the fitting of a model to separation data requires the assumption of common parameters in quite different rearing environments. If such assumptions are not made, new parameters are introduced with each new type of observation and the model is underdetermined. In contrast, the unitary model uses only one type of rearing structure (viz., the intact family). For behavioral traits the event of being reared in a

broken home or of being a twin may itself be an important factor in the development of the trait. Accordingly, investigation of the transmission of the trait using the unitary model provides a feasible and efficient resolution of the problem.

In the unitary model no assumption is made as to what proportion of T is genetic, and if h^2 denotes the heritability of the trait, we know only that $h^2 \leq t^2$. Indeed, if the mode of transmission of a trait is entirely cultural, we could have a large t^2 with $h^2 = 0$. Many investigators make the a priori assumption that $\tau = 1/2$ and $t^2 = h^2$, or comment that they are measuring t^2, but still fix τ at 1/2. Figures 3 and 4 have shown how erroneous such assumptions may be. Our model provides an explicit test for the presence of cultural inheritance ($\tau \neq 1/2$) and is a necessary first step in the analysis of any complex developmental trait observed in intact nuclear families. Failure to allow for cultural inheritance precludes its discovery and may lead to spurious results.

SUMMARY

A general linear model of familial resemblance is described which allows for cultural transmission from parent to offspring, polygenic inheritance, phenotypic assortative mating, common environment, maternal and paternal effects, and threshold effects. Three special cases are described in detail which are particularly useful when data are only available about a few classes of relatives reared in intact families. The cultural model, the polygenic model, and the pseudopolygenic model share the common feature that all factors which are transmitted from parent to offspring may be represented by one parameter without any loss of information. We introduce a new model, termed the unitary model, which includes these models and is appropriate when combined genetic and cultural transmission is present and when data are available only for individuals reared in intact nuclear families. The basic properties of these models are explored using path analysis and computer simulation, including description of the relationship between parameters under random and assortative mating, rate of approach to equilibrium, and constraints on the magnitude of the parameters.

General formulae for familial resemblance in extended pedigrees are given for any ancestor or descendant of either vertical or collateral relatives. Estimation procedures are described and a FORTRAN program TAU, available upon request, is used to provide maximum likelihood estimates of the parameters from reported correlations. A powerful test for detecting the presence of cultural transmission is suggested and applied to simulated data and to data sets reported by others for human stature, for which cultural transmission is suggested. In addition, it is shown that there is no need to postulate dominance to account for available data about height.

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