

## A Theory of Developmental Change in Quantitative Phenotypes Applied to Cognitive Development

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*A model is presented for the changes in familial resemblance as a function of age. The model allows for separate developmental components of genetic and environmental effects and for the influence of earlier phenotypic values on current measurements. Genetic and environmental effects may be specific to occasions or constant over time. Expected covariances are derived within individuals and between relatives measured at different ages. Parameter substitution shows that models with different assumptions about the mechanism of development yield different predictions for temporal changes in family resemblance. The application of the model is illustrated by the analysis of published longitudinal data on cognitive development. The data suggest that the continuity of cognitive performance over time and the increase in heritability with age reflect the cumulative long-term effects of a single set of genes expressed throughout development. The quality of the shared environment changes from family to family over time but appears to exercise a long-term effect on cognitive development.*

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**KEY WORDS:** development; aging; path analysis; heritability; family resemblance; quantitative inheritance; cognition; twins.

### INTRODUCTION

Few human traits, if any, are constant at all ages. People get taller as they get older, their blood pressures increase, and they are able to solve more complex problems. If such changes are merely constant for all mem-

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bers of a population, they are of little consequence for the analysis of family resemblance and shed little light on the processes of physical, physiological, or cognitive development. The average age trends in quantitative traits may be removed by appropriate regression or age-banding techniques and genetic analysis may proceed along conventional lines.

Individual differences in the speed and pattern of development, however, are not removed by such age-correction techniques. Such differences remain a source of misspecification in classical models for family resemblance by making the correlations between relatives a function of age differences (e.g., Eaves, 1978).

Of still greater importance is the fact that individual differences in development provide the raw material from which we may construct models for the developmental process. This basic conception has inspired several longitudinal studies of family resemblance (e.g., Wilson, 1972, 1978, 1983; Fischbein, 1981; Hay and O'Brien, 1981) in the hope that familial correlations in the patterns of change and changes in the pattern of familial correlations may yield further insight about the effects of genes and environment on development.

These studies have generated valuable data about the importance of genetic factors in development but have lacked any explicit and testable theory that relates observed age changes in family resemblance to a particular model for the developmental process. Quantitative geneticists have been little help because such scant attention as they have paid to developmental effects has consisted in fitting empirical parameters to age changes in the correlation between relatives (e.g., Eaves, 1978) or estimating separate "heritabilities" for adults and juveniles (Rao *et al.*, 1976; Young *et al.*, 1980). As long as geneticists only *describe* change, epidemiologists and developmental psychologists will gain little from genetics about phenomena as diverse as the "tracking" of blood-pressure measurements and longitudinal changes in parent-offspring correlations for cognitive test scores.

We present the elements of a theoretical model for development which, while it is still far from all-embracing, may yield greater insight about the consequences of different kinds of developmental mechanisms for age-dependent observations in human families. The model may be applied with the greatest power in the analysis of longitudinal family data but will also be helpful in representing the effects of age on the covariances between relatives in cross-sectional kinship studies (e.g., Corey *et al.*, 1985).

## THE MODEL

The basic model is given in Fig. 1. The figure specifies both genetic and environmental components of development. In this paper we make

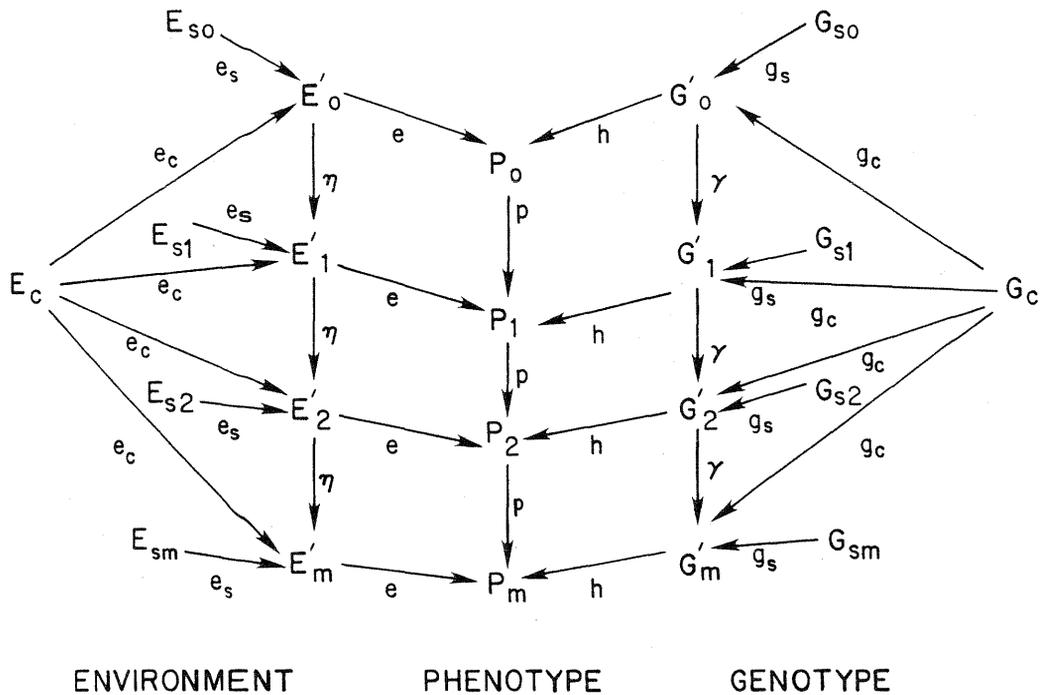


Fig. 1. Model for longitudinal measures of a continuous trait.

the (nontrivial) assumption that genes and environment are independent, with the result that phenotypic variances and covariances are simply the sum of their genetic and environmental components.

The path model describes the state of the phenotype,  $P_m$ , at time  $m$  as a function of antecedent genetic and environmental causes which, ultimately, may be traced to an original state,  $P_0$ , the time at which the genetic and environmental effects were first expressed. This “time of onset” may be assumed *ex hypothesi* or estimated from actual data. The phenotype at any age is influenced directly by a latent gene product,  $G'$ , with levels at a given occasion  $m$  represented in Fig. 1 by  $G'_m$ . In addition, the phenotype at any time may be influenced directly by the phenotype at the immediately preceding time, i.e.,  $P_m$  may be affected by  $P_{m-1}$ . The regressions of  $P_m$  on  $G'_m$  and  $P_{m-1}$  are  $h$  and  $p$ , respectively. The regressions are assumed to be constant over time, but the variances of the phenotypic values and gene-product levels will change systematically during development. We assume, without loss of generality, that the variances of the phenotype and the values of  $G'$  are unity at  $m = 0$ .

Underlying variation in levels of the intervening gene product are two possible kinds of genetic variation. Some genetic effects may be specific to each occasion. That is, there may be some genetic effects expressed at a given time which are adaptive to that time and that time only. Behavioral development, for example, is characterized by particular fears

that are expressed only at certain times. Other genes may exert a continuous and pleiotropic effect on every occasion during development. The model represents genes specific to time  $m$  by the standardized latent variable  $G_{sm}$ . The standardized latent variable,  $G_c$ , represents those genetic effects which are common to all occasions. The regressions of  $G'_m$  on  $G_{sm}$  and  $G_c$  are  $g_s$  and  $g_c$ , respectively. These regressions are assumed to be constant throughout development in our treatment and  $g_c^2 + g_s^2 = 1$ . At  $m = 0$ , therefore a proportion  $\rho_g = g_c^2$  of the variance in  $G'$  is due to genes which exercise a *direct* pleiotropic effect on subsequent phenotypic values.

The model treats development as a longitudinal process of incorporating new genetic and environmental effects into the phenotype. Figure 1 specifies three kinds of developmental continuity between adjacent occasions. The parameter  $p$  represents the direct effect of the prior state of the phenotype on the present value. This parameter would be important for those variables for which what happens now is a direct consequence of what has just happened. For example, high scores on a previous psychological test may lead directly to greater success on retesting; a person who has been told that he or she has high blood pressure might display the physiological signs of anxiety on remeasurement. The second source of continuity is provided by the persistence of  $G'$  in the system over time. Although we conceive of  $G'$  as a "gene product," this should be interpreted very loosely to mean any latent variable between genes and phenotype which may persist over time. It may include specific molecular products of gene expression but should be interpreted more generally. The regression coefficient,  $\gamma$ , represents the developmental continuity of levels of  $G'$ . The third source of developmental continuity is the persistence, or "remembering," of information derived from previous environmental experiences. The latter effects are considered subsequently. In its present form, the model ignores the effects of measurement errors that are distinct from environmental differences within families. It is a simple matter to allow for errors of measurement by including a path,  $r$ , from the "true" standardized phenotype to the measured value that is an unreliable index of the true phenotype. All expected correlations would be multiplied by  $r^2$ , if the reliability is constant over time, or by the product  $r_m r_n$  if the reliabilities differ at times  $m$  and  $n$ .

The environmental components of the model are represented in Fig. 1 by specific and general environmental effects,  $E_s$  and  $E_c$ , analogous to the genetic effects  $G_s$  and  $G_c$ . The environmental analogies of the intervening gene products,  $G'$ , are the latent variables,  $E'$ , which may form the basis of temporal continuity of information derived from the environment. The regression of phenotype on the intervening environmental

variable is  $e$ . Since the initial phenotype variance is standardized to unity,  $h^2 + e^2 = 1$ . The environmental analogy of  $\gamma$  is the regression coefficient,  $\eta$ . In psychological applications,  $\eta$  represents the effects of memory for information derived from the environment. If genetic and environmental effects persist through their effects on a common pathway, we may set  $p > 0$  and allow  $\gamma = \eta = 0$  or put  $p = 0$  and set  $\gamma = \eta$ .

### Expected Covariances Between Relatives

The rules for linear combinations of random variables may be used to derive expectations for the covariances of measurements made on the same individuals at different ages (as in the longitudinal study of unrelated individuals) and for the covariances between relatives measured at different ages (as in the typical cross-sectional study of family resemblance). These expectations may be combined to generate structural models for more informative longitudinal studies of related individuals such as the longitudinal twin studies already cited.

We derive the expected genetic component of the phenotypic covariance within individuals measured on different occasions. The genetic covariance between relatives is obtained by multiplying this expectation by the appropriate genetic correlation between relatives given additive gene action and random mating. The form of the environmental components is identical with the substitution of appropriate environmental parameters  $e$ ,  $\eta$ , and  $\rho_e$  for their genetic counterparts  $h$ ,  $\gamma$ , and  $\rho_g$ , respectively.

It is convenient to represent the phenotypic values of the  $i$ th individual at a number of equally spaced occasions  $0 \dots m$  by the  $m + 1$  element vector  $\mathbf{p}_i$ . Let  $\mathbf{C}$  be the  $m + 1$  square matrix of expected genetic covariances within individuals such that element  $c_{k,l}$  is the genetic covariance within individuals measured on occasions  $k - 1$  and  $l - 1$ . Element  $c_{1,1}$  is  $h^2$ .

The expected genetic covariances are given by

$$\mathbf{C} = h^2[\rho_g \mathbf{\Pi} \mathbf{\Gamma} \mathbf{U} \mathbf{\Gamma}' \mathbf{\Pi}' + (1 - \rho_g) \mathbf{\Pi} \mathbf{\Gamma} \mathbf{\Gamma}' \mathbf{\Pi}'], \quad (1)$$

where  $\rho_g = g_c^2$  and  $\mathbf{U}$  is an  $m + 1$  square matrix of 1's.  $\mathbf{\Pi}$  and  $\mathbf{\Gamma}$  are lower triangular. A typical element of  $\mathbf{\Pi}$  is

$$\pi_{k,l} = p^{(k-l)}, \quad k \geq l.$$

Similarly, a typical element of  $\mathbf{\Gamma}$  is

$$\gamma_{k,l} = \gamma^{(k-l)}, \quad k \geq l.$$

An element of the lower-triangular product  $\mathbf{III}\Gamma$  is

$$\alpha_{k,l} = \sum_{i=0}^{i=n} \gamma^i p^{n-i}, \quad n = k - l, l \leq k,$$

which simplifies to

$$\alpha_{k,l} = \frac{p^{n+1} - \gamma^{n+1}}{p - \gamma}.$$

We now rewrite Eq. (1) as

$$\mathbf{C} = h^2[\rho_g \mathbf{A} + (1 - \rho_g) \mathbf{B}]. \quad (2)$$

$\mathbf{A}$  and  $\mathbf{B}$  are both symmetric, with elements

$$a_{k,l} = \sum_{i=1}^{i=k} \alpha_{k,i} \sum_{i=1}^{i=l} \alpha_{l,i}$$

and

$$b_{k,l} = \sum_{i=1}^{i=l} \alpha_{k,i} \alpha_{l,i} \quad \text{for } l \leq k.$$

Now

$$\sum_{i=1}^{i=k} \alpha_{k,l} = \left( \frac{1}{p - \gamma} \right) \left[ \frac{p(1 - p^k)}{1 - p} - \frac{\gamma(1 - \gamma^k)}{1 - \gamma} \right],$$

so

$$a_{k,l} = \left( \frac{1}{p - \gamma} \right)^2 \left[ \frac{p(1 - p^k)}{1 - p} - \frac{\gamma(1 - \gamma^k)}{1 - \gamma} \right] \left[ \frac{p(1 - p^l)}{1 - p} - \frac{\gamma(1 - \gamma^l)}{1 - \gamma} \right]. \quad (3)$$

Similarly,

$$b_{k,l} = \left( \frac{1}{p - \gamma} \right)^2 \sum_{i=1}^{i=l} (p^{k-i+1} - \gamma^{k-i+1}) (p^{l-i+1} - \gamma^{l-i+1}),$$

which becomes

$$\left( \frac{1}{p - \gamma} \right)^2 \left[ p^{2+k-l} \frac{1 - p^{2l}}{1 - p^2} - (p^{1+k-l} \gamma + p \gamma^{1+k-l}) \frac{1 - p^l \gamma^l}{1 - p \gamma} + \gamma^{2+k-l} \frac{1 - \gamma^{2l}}{1 - \gamma^2} \right]. \quad (4)$$

The genetic component of the phenotypic covariance between measurements made at time  $m$  and those made at time  $n \geq m$  is obtained by substituting for  $c_{n+1,m+1}$  in Eq. (2) the expectations for the corresponding elements of **A** and **B** from Eqs. (3) and (4), yielding

$$c_{k,l} = h^2[\rho_g a_{k,l} + (1 - \rho_g) b_{k,l}]; \quad k = n + 1, l = m + 1.$$

These expectations may be substituted for the more conventional expected genetic covariances between relatives in applications of maximum-likelihood methods for the estimation of components of variance or path coefficients from longitudinal or cross-sectional family data. It remains to be seen which combinations of parameters can be estimated from particular data sets.

As  $p$  and  $\gamma$  both tend to zero, Eqs. (3) and (4) both tend to unity when  $k = l$ . Furthermore, in these circumstances  $a_{k,l} = 1$  when  $k = l$  because the genetic correlation reflects the effects of pleiotropic loci, whereas  $b_{k,l}$  tends to zero when  $p$  and  $\gamma$  tend to zero because age-specific loci do not contribute to covariance between occasions. When all the genetic effects are pleiotropic (i.e.,  $\rho_g = 1$ ) and  $p = \gamma = 0$ , the model becomes the classical biometrical genetical model in which there are no developmental effects.

As  $k$  and  $l$  both tend to infinity (i.e., at the culmination of development),  $a_{k,l}$  and  $b_{k,l}$  approach equilibrium values for  $|p| < 1$  and  $|\gamma| < 1$ . For the pleiotropic component we have the equilibrium genetic variance (i.e., the phenotypic variance in adults), a function of

$$\hat{a} = [(1 - p)(1 - \gamma)]^{-2}.$$

The equilibrium value for the age-specific component is a function of

$$\hat{b} = [(1 - p^2)(1 - \gamma^2)]^{-1},$$

obtained by setting  $k = l = \infty$  in the expectation for  $b_{k,l}$ .

At equilibrium,  $\hat{a}_{k,l}$  is the same for every  $k, l$ . However, the component of covariance due to age-specific genetic effects decays as  $\Delta = |k - l|$  increases. For large  $l$ , Eq. (4) simplifies to

$$\hat{b}_{k,l} = \left(\frac{1}{p - \gamma}\right)^2 \left[ \frac{p^{2+\Delta}}{1 - p^2} - \frac{p^{1+\Delta}\gamma + p\gamma^{1+\Delta}}{1 - p\gamma} + \frac{\gamma^{2+\Delta}}{1 - \gamma^2} \right].$$

The above treatment regards "time" as a discontinuous variable. However, development is a continuous process and many kinship studies involve the sampling of a wide range of heterogeneous ages rather than a longitudinal study at a series of well-defined specific ages. If we allow the interval between occasions,  $\delta t$ , to approach zero, we may modify the expectations of **A** and **B** to allow for "continuous" development. If  $t$  is

the time of measurement, then the number of intervals since  $t = 0$  is  $m = t/\delta t$ . As  $\delta t \rightarrow 0$ , terms of the form  $p^{al}$  approach the limit  $p^a \exp(-\alpha t)$ , where  $\alpha = 1 - p$ . This may be seen by noting that  $l = m + 1$ ,  $m = 0, 1, 2$ , etc., so that  $p^{al} = p^a \times p^{am}$ . The second term in the product may then be replaced by the exponent  $\exp(-\alpha t)$  for  $t \geq 0$ . Similarly,  $\gamma^{bl}$  becomes  $\gamma^b \exp(-\beta t)$ , where  $\beta = 1 - \gamma$ . Substitution of these continuous forms in Eqs. (3) and (4) yields expected genetic covariances of measurements made at times  $t$  and  $u = t + \Delta$ :

$$a_{t,u} = \left( \frac{1}{p - \gamma} \right)^2 \left[ \frac{p(1 - e^{-\alpha t})}{1 - p} - \frac{\gamma(1 - \gamma e^{-\beta t})}{1 - \gamma} \right] \times \left[ \frac{p(1 - e^{-\alpha u})}{1 - p} - \frac{\gamma(1 - \gamma e^{-\beta u})}{1 - \gamma} \right] \quad (5)$$

and

$$b_{t,u} = \left( \frac{1}{p - \gamma} \right)^2 \left[ p^2 e^{-\alpha \Delta} \frac{1 - p^2 e^{-2\alpha t}}{1 - p^2} - p\gamma(e^{-\alpha \Delta} + e^{-\beta \Delta}) \times \frac{1 - p\gamma e^{-(\alpha + \beta)t}}{1 - p\gamma} + \gamma^2 e^{-\beta \Delta} \frac{1 - \gamma^2 e^{-2\beta t}}{1 - \gamma^2} \right]. \quad (6)$$

The constants  $\alpha$ ,  $\beta$ , and  $\Delta$  are defined as above. Variances at time  $t$  are obtained by setting  $\Delta = 0$ . Limiting values for covariances may be obtained by letting  $t \rightarrow \infty$ . Analogous expressions may be derived by environmental components.

### Predicted Patterns of Developmental Change in Family Resemblance

Numerical evaluation of Eqs. (5) and (6) for different combinations of genetic and environmental parameters may be used to explore the expected developmental changes in statistics such as correlations between relatives and heritability estimates as a function of age and differences in age at the time of measurement. We present some examples which illustrate many of the essential predictions of different developmental mechanisms.

The first case is obtained by setting  $p = \gamma = 0$  and  $\rho_g = 1$  for the genetic component but allowing  $\rho_e = 0$  and  $\eta > 0$  for the environmental component. This case corresponds to a developmental model in which the same single set of genes is expressed on each occasion (i.e., there is complete "pleiotropy" over time) but in which the intervening product,  $G'$ , displays no temporal continuity. Developmental change is thus provided by the "remembering" of information acquired from the environment and its effects on the measured phenotype at each stage.

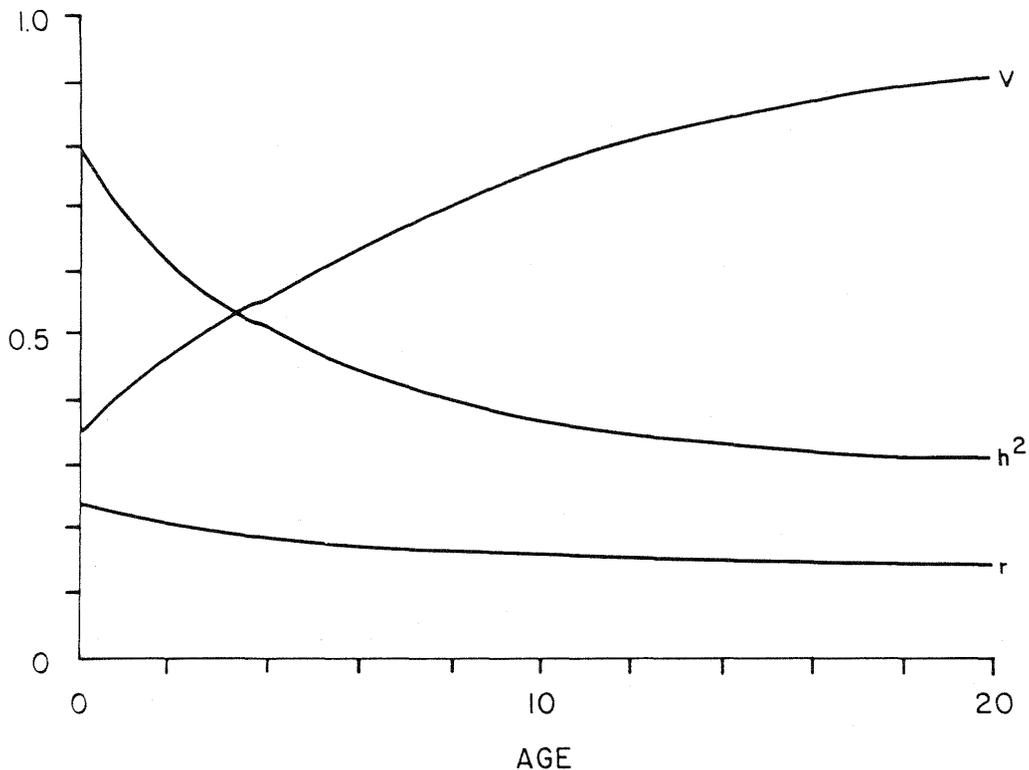
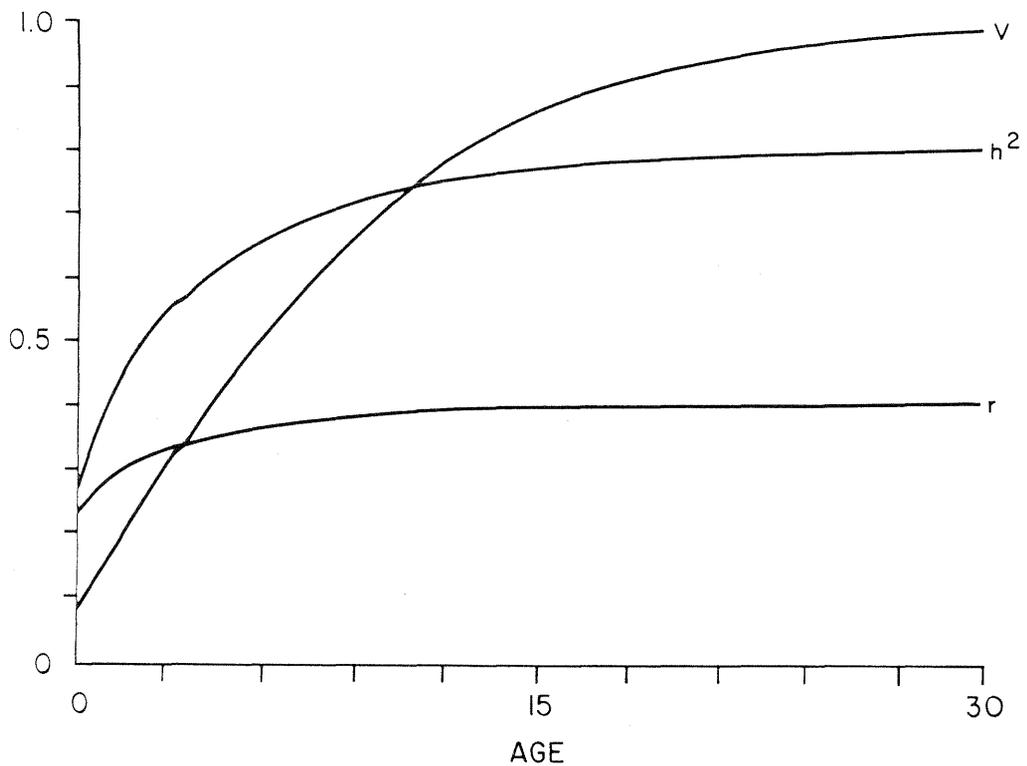


Fig. 2. Temporal changes in variance ( $V$ ), heritability ( $h^2$ ), and parent-offspring correlation ( $r$ ) when information from the environment is stored over time and a single common set of genes operates throughout development. Note: Initial heritability is assumed to be 0.8,  $\eta = 0.95$ .

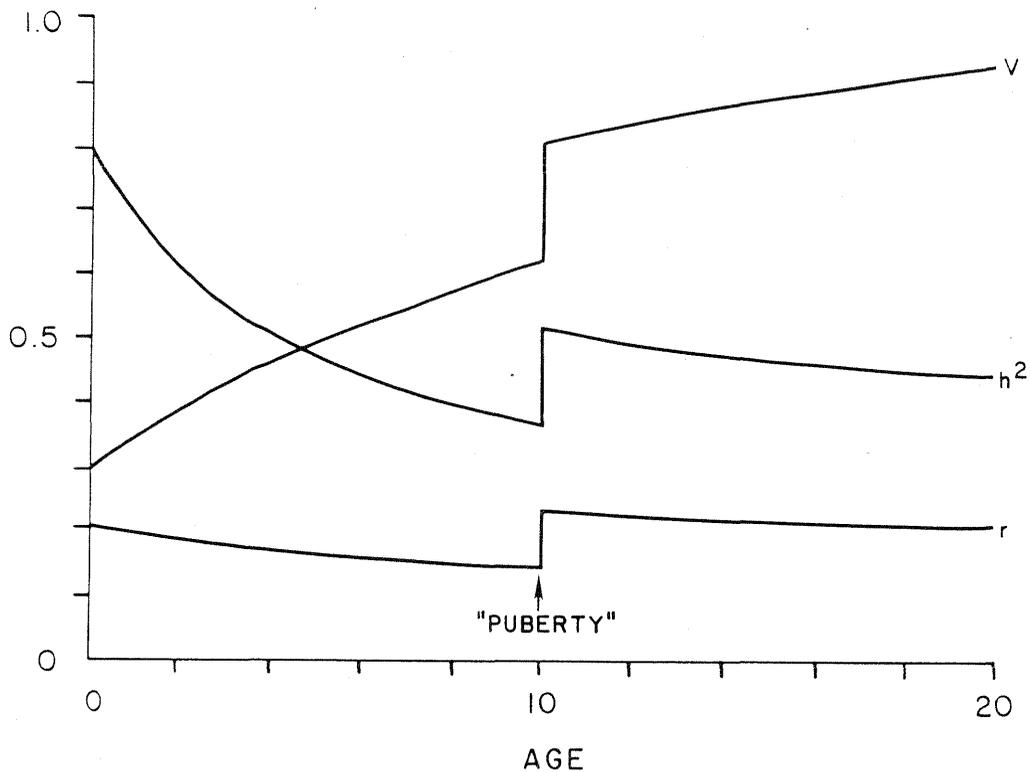
Predicted changes in the total variance, heritability, and parent-offspring correlation with age are shown in Fig. 2. The genetic variance and covariance are constant over time because of the assumptions that  $p = \gamma = 0$  and  $\rho_g = 1$ . However, the progressive accumulation of new information from the environment causes a *decrease* in heritability over time because the additional environmental experiences increase the proportional contribution of environmental factors to individual differences. The parent-offspring correlation approaches its expected asymptotic value of  $\frac{1}{2}\hat{h}^2$ , where  $\hat{h}^2$  is the limiting value of the heritability as  $t \rightarrow \infty$ . However, with the parameter values assumed, the correlation between parents and *young* children actually exceeds  $\frac{1}{2}\hat{h}^2$ . The excess might wrongly be attributed to the effects of cultural inheritance in nuclear family data.

A second important special case is given in Fig. 3. The model assumes that a single common set of genes is expressed throughout development ( $\rho_g = 1$ ) and that environmental effects are occasion specific ( $\rho_e = 0$ ). Developmental continuity is ensured by the direct longitudinal influence



**Fig. 3.** Temporal changes in variance ( $V$ ), heritability ( $h^2$ ), and parent-offspring correlation ( $r$ ) when there is direct continuity of phenotypic effects. Note: Initial heritability is assumed to be 0.25,  $p = 0.85$ ,  $\eta = \gamma = 0$ .

of previous phenotypic differences on the present phenotype ( $p > 0$ ;  $\gamma = \eta = 0$ ). This model represents a more dynamic conception of the roles of genes and environment in development because each occasion requires the same basic inherited skills ( $\rho_g = 1$ ) and exploits the particular environmental information available in each situation ( $\rho_e = 0$ ). However, the result of this joint action of genes and environment on a specific occasion is transmitted forward in time and continues to exercise an effect ( $p > 0$ ) on subsequent situations. As in the previous model, the total variance increases from  $t = 0$  to an asymptotic value. There are obvious differences in trend for the heritability and parent-offspring correlation, however. The heritability now increases because the common genetic effects,  $g_c$ , create correlations between the  $G'$  effects which contribute additional terms to the genetic component of the phenotypic variances and covariances. The genetic contribution to the variance thus increases approximately with the square of the age. However, the environmental components are specific to occasions and their effects on phenotypic variance thus accumulate approximately linearly over time. The result is that even quite small initial genetic effects, if they are expressed consistently



**Fig. 4.** The effects of a second set of genes "switched on" at "puberty" ( $T = 10$ ) on the variance ( $V$ ), heritability ( $h^2$ ), and parent-offspring correlation ( $r$ ) when temporal continuity is environmental. Note: Initial heritability of the first and second gene sets is 0.8,  $p = \gamma = 0$ ,  $\eta = 0.95$ .

throughout development, may have cumulative effects on individual phenotypes that far outweigh the substantial but unsystematic effects of the environment.

This set of parameters predicts a continuous increase in the parent-offspring correlation to its asymptotic value of  $\frac{1}{2}h^2$ . However, the correlation between parents and their juvenile offspring is less than  $\frac{1}{2}h^2$ . Such a finding might be interpreted wrongly as evidence of nonadditive genetic effects.

The model described so far has assumed that gene expression starts at birth and persists according to the same basic rules throughout development until adult life. That is, the model does not allow for major changes in gene expression which accompany critical development stages such as puberty. The basic model, however, is easily modified to reflect the "switching on" of major new genetic systems at a critical age, such as might accompany a major transition in cognitive development. Figures 4 and 5 present predicted changes in selected statistics under the two previous models when a second set of genetic effects is activated at some

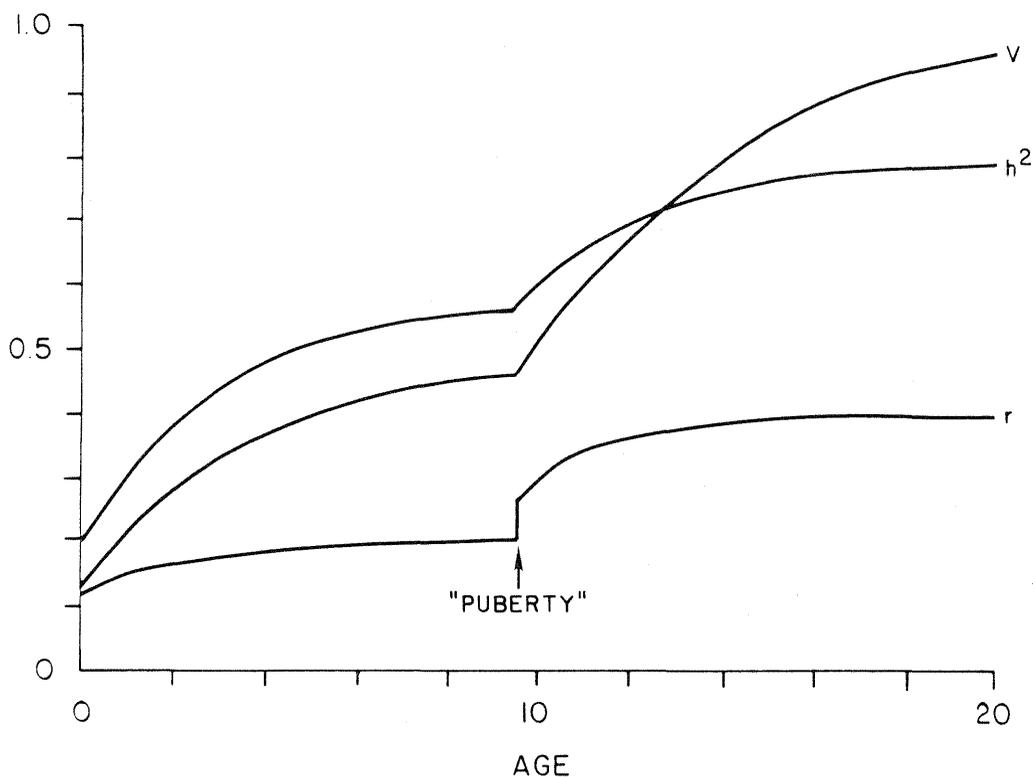


Fig. 5. The effects of a second set of genes "switched on" at "puberty" ( $T = 10$ ) on the variance ( $V$ ), heritability ( $h^2$ ), and parent-offspring correlation ( $r$ ) when temporal continuity is genetic. Note: Initial heritabilities are 0.2 and 0.6, respectively,  $\eta = \gamma = 0$  and  $p = 0.7$ .

critical time corresponding, for example, to puberty. Figure 4 summarizes the expected changes when the environment is the main source of continuity (corresponding to Fig. 2) and Fig. 5 shows the predicted pattern when there is phenotypic continuity over time similar to that presented in Fig. 3 for the case of a single set of genes.

Figure 4 shows that the effects of the second set of genes lead to a sharp increase in the genetic component at the critical stage, which is then eroded as before by the accumulation of environmental effects. The trajectory of the decline is unchanged after the critical stage. Figure 5 reveals that the second set of genes increases the rate of change in heritability with age. Although these figures are artificial in the assumption that individuals do not differ in the age of onset of the second major genetic component, they illustrate trends to be expected under broadly different theories of developmental change. Other figures could be generated to reflect the consequences of major new sources of environmental variation such as might accompany the start of elementary school.

### An Application to Cognitive Development

Few sets of longitudinal data are published in a form that allows us to test both genetic and developmental hypotheses simultaneously. Wil-

son (1983, Tables 1 and 2) presents the correlations between monozygotic (MZ) and dizygotic (DZ) twins for measures of cognitive ability made at several ages from 3 months through 15 years and the within-individual ("phenotypic") cross-temporal correlations. A detailed genetic analysis also requires the cross-temporal correlations across twins which are not published in the paper cited. However, the data still allow us to test and exclude some important hypotheses.

We employed the method of weighted least-squares (WLS) to fit various forms of the developmental model to correlations tabulated by Wilson after  $z$  transformation to improve the approximation to normality. Our analysis is approximate at best for two main reasons. First, we do not have the exact sample sizes contributing to every correlation. For the twin correlations, we used the tabulated degrees of freedom to generate weights. For the phenotypic cross-temporal correlations, only the median sample sizes were available, from which we generated approximate weights. Second, we assumed that the raw correlations are statistically independent, which they certainly are not. For those cases that have been studied in detail (McGue *et al.*, 1984), violation of the assumption of independence does not lead to serious bias in parameter estimates but may lead to errors in their estimated variances and in the goodness of fit test. In this example, we are concerned merely with illustrating some of the principles of the model. A more exact treatment could be adopted when complete data become available.

Even within the very restricted model we have outlined, there is a very large number of possible specific subhypotheses that might be considered. We give (Table I) the results of fitting some forms of the overall model to the correlations tabulated by Wilson. There are 30 twin correlations and 101 of the 105 possible cross-temporal phenotypic correlations. A rough guide to the goodness of fit of a model involving  $p$  free parameters is the weighted residual sum of squares for  $131 - p$  degrees of freedom. This statistic is approximately distributed as chi-square when the model fits. None of the models fits very well. Model 12 gives the best fit of those tabulated, and still has a residual  $\chi^2$  of 360 for 126 df. However, even this simple model is a vast improvement over many of the others and involves very few parameters. The first four models are equivalent to conventional "factor" models for the covariance structure over time similar to those employed by Martin and Eaves (1977) and others in the analysis of multivariate twin data. There is no "developmental" component in these models in the sense that earlier influences do not exercise any long-term effect on later performance. They are included for comparison only. Model 1 assumes that there is a single common factor with identical genetic loadings on every occasion of measurement and occasion-specific

Table I. Some Models for Cognitive Development

Model	Genetic			Environmental					$\chi^2$	df
	$h$	$\rho_g$	$\gamma$	Family			Unique			
				$b$	$\rho_b$	$\beta$	$\rho_e$	$\eta$		
1	0.76	1	—	—	—	—	0	—	4304	130
2	0.92	0	—	—	—	—	0	—	17983	130
3	0.76	0	—	—	—	—	1	—	3824	130
4	0.92	0.67	—	—	—	—	0	—	3736	129
5	0.19	1	0.97 <sup>a</sup>	—	—	—	0	0.97 <sup>a</sup>	2522	129
6	0.07	1	0.97	—	—	—	0	—	1969	129
7	0.35	0.02	0.99	—	—	—	0	—	602	128
8	0.05	1	0.99	0.35	0	0.98	0	—	398	127
9	0.10	1	0.98	0.78	0	0.91 <sup>a</sup>	0	0.91 <sup>a</sup>	592	127
10	0.30	0	0.99	0.05	1	0.98	0	—	516	127
11	0.25	1	0.99	0.55	1	0.20	0	—	934	127
12	0.06	1	0.99	0.39	0	0.99	0	0.66	360	126

<sup>a</sup> Parameters constrained to be equal *ex hypothesi*.

within-family environmental effects. Model 2 assumes that both genetic and environmental effects are occasion specific, i.e., predicts no cross-temporal correlation. Model 3 assumes specific genetic effects but a common factor in the within-family environment. Model 4 that assumes the within-family environment is occasion specific but that genetic effects are both common and specific. Clearly, these models do not exhaust all the conventional multivariate models that might be contrived for these data and make assumptions which are clearly inappropriate for these data, including no family environment (obviously not the case for the early measures), a constant heritability over age (the genetic components increase with age), and a constant phenotypic correlation (the cross-temporal phenotypic correlations decay as a function of increasing age difference). The fact that these models give a very bad fit is thus consistent with a cursory examination of the data.

The important finding, however, is the improvement in fit to the raw correlations that results from including developmental parameters in the model. The remaining models (Table I) all specify some form of cross-temporal continuity which allows for the successive incorporation of new information into the measured phenotype. The models are illustrative rather than exhaustive.

Model 5 is a developmental version of Model 1. It assumes that the same genes operate throughout development ( $\rho_g = 1$ ) but that environmental input is occasion specific in origin ( $\rho_e = 0$ ). This model assumes

that continuity during development is a function of cross-temporal phenotypic effects ( $p$ ) rather than memory for environmental input or persistence of genetic effects alone. In tabulating the results we represent the effects of  $p$  as an equality constraint on  $\gamma$  and  $\eta$ . The improvement in chi-square over that for Model 1 is approximately 1800 units for the addition of the single parameter and suggests that the developmental perspective adds greatly to our understanding of the data. This model is very similar to that represented graphically in Fig. 3, predicting an increase in the heritability with age and a reduction in the cross-temporal phenotypic correlations with increasing age difference. The fit improves still further if we allow for developmental continuity in the genetic effects alone (Model 6). A third major increase in information results from allowing  $\rho_g$  to take its own value (Model 7). The WLS estimate of this parameter is 0.02, which is very close to zero, implying that there is marked age specificity in the onset of genetic effects; that is, new genetic effects are continually being "switched on" and accumulating during development. However, the "continuity" parameter,  $\gamma$ , is very high (0.99), implying that once these genetic effects are established, they are extremely persistent over time.

Model 7 is about as far as we can get with improving the fit without a further radical change in the model. Models 8–12 show some of the effects of allowing for the shared environment in development, in the attempt to explain some of the obvious effects of the shared environment apparent at an early age. We introduce parameters  $\rho_b$ ,  $\beta$ , and  $b$  to represent the analogues in the model for the shared environment to the genetic parameters  $\rho_g$ ,  $\gamma$ , and  $h$ . In each case, the family environment,  $B$ , is assumed to operate throughout development. This assumption is not essential. An alternative developmental model would allow for an initial "pulse" of family environment, possibly due to prenatal and perinatal effects, which gradually decays with age. The model is easily modified to allow for this process by omitting all family environmental effects apart from the initial effect,  $B_0$ , and reevaluating the expected covariances.

Model 8 is best compared with Model 6. The environment within families is assumed to be occasion specific in both models and to show no developmental continuity. The same genes are assumed to be expressed at every stage ( $\rho_g = 1$ ) and their effects are allowed to accumulate over time ( $\gamma > 0$ ). There is assumed to be continuous, occasion-specific input from the family environment which is "remembered" over time ( $\rho_b = 0$ ,  $\beta > 0$ ). This model thus implies that the parents (say) are a continual source of environmental stimulation, providing novel experiences which may be relatively advantageous at some times and relatively disadvantageous at others (hence  $\rho_b = 0$ ), and that the child stores some of these

experiences over time ( $\beta > 0$ ) in a form that influences his or her subsequent test performance. The improvement in fit achieved by including the shared environment is almost 1600 chi-square units for the addition of only two parameters. Although we may doubt the precision of the test statistic, this gain is substantial by any criterion. Model 9 tries to incorporate environmental perturbations within families into the developmental process and assumes that they are "remembered" with the same precision as the effects of the family environment ( $\rho = \eta > 0$ ). Model 9 is actually worse, confirming that environmental differences within families have different developmental implications for the individual from those of the shared environment. If all environmental experiences were equally salient for the individual, regardless of their origin, we might expect them to be "remembered" with comparable precision. We do not make too much of this point in the present analysis because errors of measurement also contribute to our estimates of the within-family environment in family data.

Models 10 and 11 try to assess the consistency of the quality of the family environment over time. If parents provided a consistently "good" or "bad" environment throughout development, we would expect  $\rho_b = 1$ . If the quality of the parental environment changes haphazardly over time, then  $\rho_b = 0$ . Both models imply that the environment within families is occasion specific and not remembered. Model 10 assumes that new genetic effects are switched on continually ( $\rho_g = 0$ ) and Model 11 assumes that the same genes are expressed all the time ( $\rho_g = 1$ ). In both cases we allow the genes to have long-term developmental effects ( $\gamma > 0$ ). Neither model fits as well as Model 8, which assumes that parents fluctuate inconsistently in the relative quality of the environment they provide for their children.

Of all the models we tried, the best unbounded solution is given by Model 12. This model assumes that a single common set of genes is switched on at birth, albeit with a very small initial heritability ( $h = 0.06$ ), with effects that persist very effectively over time ( $\gamma = 0.99$ ), allowing a rapid accumulation of genetic effects during development. The model allows for a substantial, continuous, and age-specific input from the family environment ( $b = 0.39$ ), with effects that also persist over time with high reliability ( $\beta = 0.99$ ). The implication of the model is that the family environment has a persistent effect but that the "quality" of input at a given time relative to the mean varies greatly from time to time. Sometimes one set of parents "gets it right," and then on another occasion another set of parents finds the better stimulus to cognitive development. Good or bad, however, the persistence of these effects over time implies that they are extremely salient to the relative cognitive development of

the individual. Our model for the environment within families suggests that their long-term importance for development is significant but less marked ( $\eta = 0.66$ ). Although individual differences within the family might be important at a given age, their effects do not persist very long relative to those of genes and the family environment. This finding is consistent with Wilson's observations about his own data that the relative cognitive abilities of MZ twins seem to change repeatedly over time, with one twin first being ahead and then the other. For DZ twins, in contrast, their standing in relation to each other remains much more consistent over time, confirming the long-term persistence of genetic effects. One reason for the "transience" of environmental effects within families may simply be the confounding of "salient" environmental effects with those of measurement error. However, one researcher's test-retest errors of measurement are another's transient environment. At this stage, the best we can do is to distinguish those aspects of individuals' experiences that are remembered and influence subsequent performance from those that have no persistent developmental effects and, therefore, might profitably be regarded as measurement error.

## DISCUSSION

The best of the 12 forms of the basic model does not fit the published data very well ( $\chi^2_{126} = 360$ ); nevertheless, it represents a substantial gain over many models that lack a developmental perspective. The last model also fits much better than many alternative forms of the developmental model. The fit might be improved further by allowing for different errors of measurement at different ages and for a second set of genetic effects with later initial expression. More refined analyses, however, require additional statistics, including the cross-twin correlations over time, so that the developmental effects of genes and environment may be distinguished more effectively. Whatever the final outcome, however, the parameters of our model give us greater insight about the way in which genetic and environmental effects unfold and accumulate over time. They may prove sufficient to explain such empirical findings as the relative "spurts and lags" of cognitive development to which Wilson has long drawn our attention (e.g., Wilson, 1972) and provide a mathematical formulation of a developmental process which may predict the patterns of age change in twin correlations described by Fischbein (e.g., 1981).

The model also allows us to explore theoretically various crucial ideas about the course of development and their basis in genetic and environmental factors. Our numerical examples show that different quantitative trends in second-degree statistics derived from twin and family data follow

from different models for development. For example, models in which information derived exclusively from the environment is stored over time lead to a gradual reduction in the contribution of genetic factors to individual differences. In contrast, if the results of gene action at a particular occasion persist over time, the contribution of genetic factors to variation will increase with age. The correlations between relatives will change with age in systematic ways which reflect, inter alia, the relative persistence of genetic and environmental effects over time and the cross-temporal correlation between the primary effects of genes and those of environment. The greatest increase in heritability with time is predicted when the same genes are expressed consistently throughout development and the early effects of the genes on the phenotype have long-term consequences for measurements made at later ages. In striking contrast, "memory" for previous environmental effects will tend to eradicate genetic effects in the long run if the genetic effects do not induce comparable permanent phenotypic changes with lasting effect.

The model may be used as a basis for the analysis of many kinds of data in which the expression of genes and environment accumulate over time. The method of maximum likelihood may be applied to unreduced cross-sectional kinship data when the covariances between individuals in a pedigree are each expected to be different because of heterogeneity in the age structure of the pedigree (Eaves, 1978). If we have longitudinal data on individuals measured at a series of predetermined intervals, then it is possible to formulate and maximize the likelihood of the observed covariance structure in terms of a developmental model.

A common problem in the analysis of behavioral development is the fact that different units are often used at different ages. This fact does not detract from the basic import of our model because many of the broad features of various versions of the model are invariant with respect to changes of scale. The main problem we might anticipate is the differential reliability of tests conducted at different ages. Appropriate changes can be made to the model if estimates of unreliability are available.

In describing the model, we made a number of arbitrary simplifications that can be removed quite easily. We have assumed that gene effects are first expressed at birth or at some arbitrary constant time determined *ex hypothesi*. This assumption can be relaxed and the method of maximum likelihood employed, in theory, to estimate the "age of onset."

Although we have allowed for the effects of the "shared" environment in our analysis of the longitudinal twin data, the present form of the model does not allow for assortative mating or formulate any model for cultural transmission from parent to child that could account for genotype-environment covariance. The consequences of these influences will

depend on the mechanism of social interaction between spouses and their effect on children. Under more realistic models of development, the effects of cultural inheritance may depend on the specific age structure of the family and the timing of crucial social changes. Some models for the impact of the family environment upon development will predict that such social effects are transient and will not persist very long after children leave home. A recent paper by Carey (1985) shows how a developmental model for sibling interactions based on phenotype can predict the covariance structure of sibships of arbitrary size and illustrates the power of a mathematical model that combines the effects of genes and social interaction in a coherent theory for developmental change.

Although our model is extremely simple and employs relatively few parameters, it gives great scope for fundamentally different patterns of temporal change in twin and family data and can elucidate many features of published longitudinal data on cognitive development. Our conclusions are remarkably consistent with those of other investigators (e.g., Plomin and DeFries, 1985). However, the model we present provides an explicit and economical mathematical formulation for ideas that have, hitherto, been expressed only in words. Our model should help investigators to think more precisely about the implications of different theories of development for the pattern of family resemblance so that alternative hypotheses may be compared. The practical value of the model, and the feasibility of extending it to more subtle cases, remains a fertile area of further inquiry.

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