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Resolution of Cultural and Biological Inheritance by Path Analysis

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Two schools have developed genetic analysis of quantitative data. One radically simplifies environmental effects to provide an estimate of dominance deviations [1]; the other realistically elaborates environmental effects, thereby risking indeterminancy even when the estimate of dominance deviations is sacrificed [2]. We have argued [3] that the second approach is preferable, since environment common to relatives is likely to be a more important source of variation than is dominance. We concentrated on resemblance of sibs, half-sibs, and twins, remarking that [3]: "Our treatment of parent-offspring pairs has been simplified, since most studies of familial correlation deal with children. If our scheme is not adequate, environment of the parent as a child may be introduced, connected to the environment he provides his children by a direct path and a path through his adult phenotype. Relations between adult siblings and between indices of common environments in successive generations would be useful in determining the values of these paths." In the present paper, the model is extended to cover correlations between mates, in-laws, uncle-niece, first-cousin, and other pairs. Analysis of two examples and discussion of the strategy for resolution of cultural and biological inheritance are also presented.

THE MODEL

Racial parameters [3] and dominance deviations [4] are neglected. Family environment in childhood is estimated by an index, usually a measure of social class, and is assumed the same for all individuals reared together by the same parents. Five of the path coefficients previously defined (c, h, i, m, z) are retained. Two new parameters (s, u) are introduced for components of the marital correlation, and three others (f, x, y) for causes of family environment (table 1). There is assumed to be no influence of sex on these parameters, otherwise we must distinguish f, f' for the father and mother. Even where a maternal effect was suspected (i.e, for I.Q.), it was not demonstrated [5]. All relations are taken to be linear, with equal variances for relatives who were not adopted. Neither assumption is severe, since gene-environment interactions have not been found to contribute to family resemblance [6], and the validity of the data is questionable when standardization for age and sex fails to impose homoscedasticity.

MARITAL AND PARENT-OFFSPRING CORRELATIONS

We suppose that parental phenotypes and the indices of their childhood environments are determined prior to marriage (i.e., cohabitation does not increase simi-

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TABLE 1

PATH COEFFICIENTS OF THE GENERAL MODEL

Symbol	Definition
Previously defined:	
c	Effect of family environment on child's phenotype
h	Effect of genotype on child's phenotype (square root of "heritability")
i	Effect of family environment on index (a measure of reliability of the index)
m	Correlation between parental genotypes
hz	Effect of genotype on adult's phenotype
New parameters:	
Marital:	
s	Correlation between (childhood) family environment of an adult and the spouse's genotype
u	Correlation between (childhood) family environments of spouses
Causal:	
f	Effect of parent's (childhood) family environment on environment of children
x	Effect of parent's (adult) phenotype on environment of children
cy	Effect of (childhood) family environment on adult's phenotype
Derived:	
a	Correlation between individual's genotype and (childhood) family environment $= [hzx(1+m)+s(f+cyx)]/(1-f-cyx)$

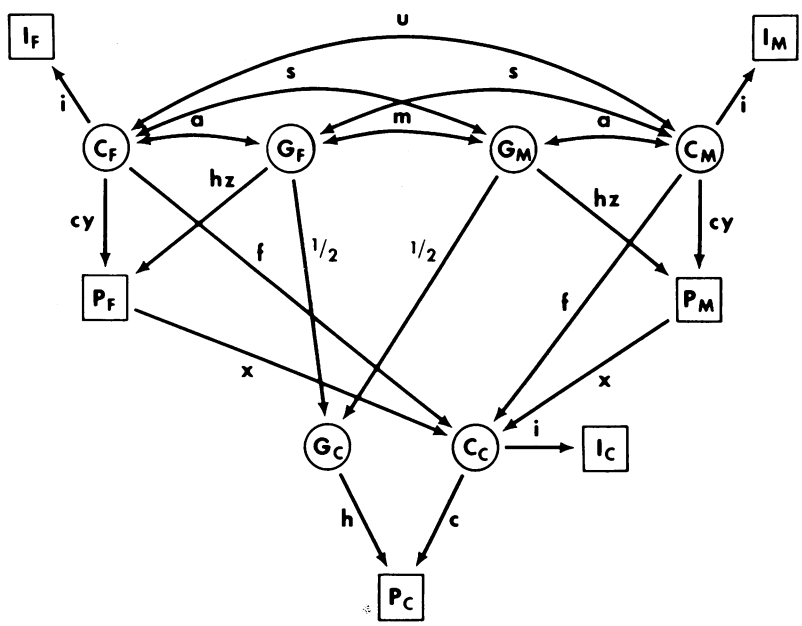

FIG. 1.—Marital and parent-offspring path diagrams. The subscripts *F, M, C* denote father, mother, and child, respectively. *G* is genotype, *P* is phenotype, and *C* is common environment with index *I*.

larity of mates). This assumption is often overlooked and may well be wrong, making the marital correlation anomalously high and useless for path analysis. A test of goodness of fit should reveal such discrepancies in a well-designed study.

Let P_F, I_F denote the adult phenotype and index, respectively for the father, and P_M, I_M for the mother. C_F, C_M relate to the family environments of the parents as children. Following a recent convention [7], we designated unobserved causes by circles and measured effects by squares. From the path diagram in figure 1, the

TABLE 2

EXPECTED MARITAL AND PARENT-OFFSPRING CORRELATIONS

Relation	Variables	Correlation
FMT:		
father-mother		
at marriage	(P_F,P_M)	$(hz)^2m + 2hzscy + (cy)^2u$
	(P_F,I_F), (P_M,I_M)	$i(cy + hza)$
	(P_F,I_M), (P_M,I_F)	$i(cyu + hzs)$
	(I_F,I_M)	i^2u
OPT:		
offspring-parent		
living together	(P_F,P_C), (P_M,P_C)	$h^2z(1 + m)/2 + cxm(hz)^2 + hzsc(f + cyx) + hzacf + c^2uf + cyh(a + s)/2 + hzx\,c^2ys + c^2yu(f + cyx) + cx$
	(P_F,I_F), (P_M,I_M)	Same as for FMT
	(P_C,I_C)	$i[c + h^2zx(1 + m) + h(a + s)(f + cyx)]$
	(P_F,I_C), (P_M,I_C)	$i[x + cyf + hzaf + cyshzx + cyu(f + cyx) + hz(mhzx + sf + scyx)]$
	(P_C,I_F), (P_C,I_M)	$i[h(a + s)/2 + c(f + cyx)(1 + u) + cxhz(a + s)]$
	(I_F,I_C), (I_M,I_C)	$i^2[(1 + u)(f + cyx) + hzx(a + s)]$
OPA:		
offspring-parent		
living apart	(P_F,P_C),(P_M,P_C)	$[h^2z(1 + m) + cyh(a + s)]\theta/2$
	(P_F,I_F), (P_M,I_M)	Same as for OPT
	(P_C,I_C)	$ci\theta$
	(P_C,I_F), (P_C,I_M)	$hi(a + s)\theta/2$
OFP:		
offspring-foster parent ..	(P_F,P_C), (P_M,P_C)	$c[x + mx(hz)^2 + hzscyx + f(cy + hza) + (f + cyx)(hzs + ucy)]\theta$
	(P_F,I_F), (P_M,I_M)	Same as for OPT
	(P_F,I_C), (P_M,I_C)	Same as for OPT
	(P_C,I_C)	Same as for OPA
	(P_C,I_F), (P_C,I_M)	$ci[hzx(a + s) + (1 + u)(f + cyx)]\theta$
	(I_F,I_C), (I_M,I_C)	Same as for OPT

NOTE.—Subscripts *F*, *M*, *C* denote father, mother, and child, respectively. The correlation between genotype and childhood common environment is $a = [hzx(1 + m) + s(f + cyx)]/(1 - f - cyx)$. The ratio of the phenotypic standard deviations for children reared by their own parents and randomly adopted is $\theta = 1/\sqrt{1 - 2\ hac}$.

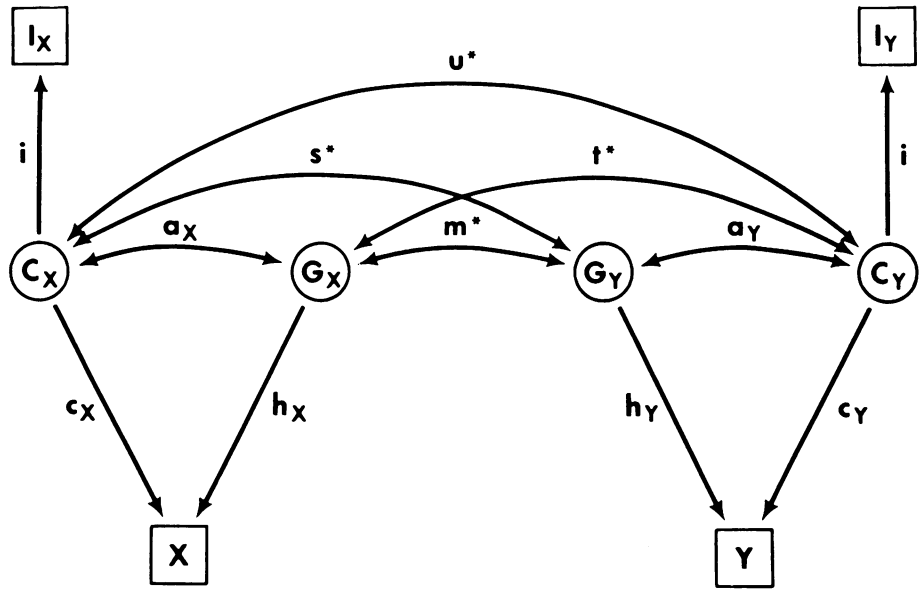

FIG. 2.—Simplified path diagram for any two individuals (except parent-offspring pairs) with phenotypes X,Y.

correlation between child's genotype (G_O) and family environment (C_O) is $(a + s)(f + cyx) + hzx(1 + m) \equiv a$, assuming constancy over generations. We can thus eliminate a as a parameter by setting $a = [hzx(1 + m) + s(f + cyx)]/(1 - f - cyx)$. Expected correlations for marital (FMT) and parent-offspring (OPT) pairs can be derived from figure 1, which may also be used, if modified, for foster parents and adopted children, relations denoted OPA and OFP in table 2.

In the form presented here the causal paths between generations are logically more complete and satisfying than as originally presented [3]. Environment of the child is determined jointly by parental phenotypes and their childhood environ-

TABLE 3

EXPECTED CORRELATIONS FROM FIGURE 2 FOR RELATIONSHIPS LISTED IN TABLE 4

Variables	Correlation
X,Y	$(h_x h_y m^* + c_x c_y u^* + c_x s^* h_y + h_x t^* c_y)\omega\lambda$
X,I_X	$i(c_x + h_x a_x)\omega$
Y,I_Y	$i(c_y + h_y a_y)\lambda$
X,I_Y	$i(h_x t^* + c_x u^*)\omega$
Y,I_X	$i(h_y s^* + c_y u^*)\lambda$
I_X,I_Y	$i^2 u^*$ if $u^* \neq 1$ (1, otherwise)

NOTE.—ω = Ratio of phenotypic standard deviation of X if reared by own parents to actual standard deviation of X; λ = ratio of phenotypic standard deviation of Y if reared by own parents to actual standard deviation of Y; for natural-adopted pairs, X is natural; for adult-child pairs, X is adult.

TABLE 4

CORRELATIONS

Relation	Description	h_x	h_y	c_x	c_y	a_x	a_y	m^*	s^*	t^*	u^*	ω	λ
MZT	MZ twins reared together by parents	h	h	c	c	a	a	1	a	a	1	1	1
MZP	MZ twins reared apart, one by true parents	h	h	c	c	a	0	1	a	0	0	1	θ
MZA	MZ twins reared apart by foster parents	h	h	c	c	0	0	1	0	0	0	θ	θ
MZF	MZ twins reared together by foster parents	h	h	c	c	0	0	1	0	0	1	θ	θ
SST	Full sibs reared together by parents	h	h	c	c	a	a	m_1	a	a	1	1	1
SSP	Full sibs reared apart, one by true parents	h	h	c	c	a	0	m_1	a	0	0	1	θ
SSA	Full sibs reared apart by foster parents	h	h	c	c	0	0	m_1	0	0	0	θ	θ
HST	Half sibs reared together by parents	h	h	c	c	a	a_1	m_2	a_1	a	1	1	1
HSP	Half sibs reared apart, one by true parents	h	h	c	c	a	0	m_2	a_1	0	0	1	θ
HSA	Half sibs reared apart by foster parents	h	h	c	c	0	0	m_2	0	0	0	θ	θ
HSS	Half sibs reared apart by own parents	h	h	c	c	a	a	m_2	a_1	a_1	u_1	1	1
FST	Foster sibs reared together	h	h	c	c	0	0	0	0	0	1	θ	θ
FSP	Foster sibs reared by parents of one	h	h	c	c	a	0	0	0	a	1	1	θ
OET	Offspring-excluded parent	hz	h	cy	c	a	a_1	m	s	a	u_2	1	1
AST	Adult full sibs	hz	hz	cy	cy	a	a	m_1	a	a	1	1	1
AMT	Adult MZ twins	hz	hz	cy	cy	a	a	1	a	a	1	1	1
AHT	Adult half sibs (like HST)	hz	hz	cy	cy	a	a_1	m_2	a_1	a	1	1	1
LIT	Like in-laws (ego's spouse's sib's spouse)	hz	hz	cy	cy	a	a	m_3	s_1	s_1	u_3	1	1
UIT	Unlike in-laws (ego's spouse's sib)	hz	hz	cy	cy	a	a	m_3	s	s_1	u	1	1
UNT	Cognate uncle-niece	hz	h	cy	c	a	a	m_4	s_2	t_1	u_2	1	1
ANT	Affinal uncle-niece	hz	h	cy	c	a	a	m_3	s_1	t_2	u_4	1	1
FCT	First cousins	h	h	c	c	a	a	m_5	s_3	s_3	u_5	1	1

TABLE 4 (*Continued*)

CORRELATIONS

Relation	Description	h_x	h_y	c_x	c_y	a_x	a_y	m^*	s^*	t^*	u^*	ω	λ
ACT	Adult first cousins	hz	hz	cy	cy	a	a	m_5	s_3	s_3	u_5	1	1
TUT	MZ twin and child of cotwin	hz	h	cy	c	a	a	m_1	s_2	a	u_2	1	1
TAT	MZ twin's child and cotwin's spouse	hz	h	cy	c	a	a	m	s	t_3	u_6	1	1
TCT	Children of MZ twins	h	h	c	c	a	a	m_2	s_4	s_4	u_7	1	1
ATT	Adult children of MZ twins	hz	hz	cy	cy	a	a	m_2	s_4	s_4	u_7	1	1

NOTE.—$a_1 = [(a + 3s)(f + cyx) + hzx\ (1 + 3m)]/2$; $m_1 = (1 + m)/2$, $m_2 = (1 + 3m)/4$, $m_3 = m(1 + m)/2$, $m_4 = (1 + m)^2/4$, $m_5 = (1 + m)(1 + 3m)/8$; $s_1 = s(1 + m)/2$, $s_2 = (a + s)/2$, $s_3 = [hzx\ (1 + m)(1 + 3m) + 2\ (f + cyx)(a + 2s + sm)]/4$, $s_4 = [hzx\ (1 + 3m) + (a + 3s)(f + cyx)]/2$; $t_1 = [hzx\ (1 + m)^2 + (f + cyx)(2a + s + sm)]/2$, $t_2 = hzxm\ (1 + m) + (f + cyx)\ s\ (3 + m)/2$, $t_3 = 2\ [hzxm + s\ (f + cyx)]$; $u_1 = x^2 + 3m\ (hzx)^2 + 3u\ (f + cyx)^2 + 2hzxfa + fcyx + (f + cyx)(f + 6hzxs)$, $u_2 = hzx\ (a + s) + (1 + u)(f + cyx)$, $u_3 = u\ (1 + m)/2$, $u_4 = hzxs\ (1 + m) + u\ (f + cyx)\ (3 + m)/2$, $u_5 = [(1 + m)(1 + 3m)(hzx)^2 + (f + cyx)^2\ (2 + 5u + um) + 4hzx\ (f + cyx)\ (a + 2s + sm)]/2$, $u_6 = 2\ [hzxs + u\ (f + cyx)]$, $u_7 = (1 + 3u)(f + cyx)^2 + (1 + 3m)(hzx)^2 + 2hzx\ (a + 3s)(f + cyx)$; $\theta = 1\ \sqrt{1 - 2\ hac}$.

ments, while parental variables are not influenced by their children. The critical assumptions are: (1) maternal and paternal effects are equal; (2) foster parents are random; and (3) true parents exercise no environmental effect (prenatal or postnatal) on an adopted child. These assumptions are best tested by residual χ^2 in an overdetermined system (i.e., with the number of correlations with different expectations exceeding the number of parameters to be estimated).

OTHER NUCLEAR RELATIONS

Path diagrams for pairs other than parent-child are illustrated in figure 2. Generic symbols like s^* are evaluated for each relationship by extending the diagram as in figure 1 for both P_x and P_y and assigning appropriate correlations between the two sets of parental variables (tables 3 and 4). The first 13 cases (table 4) were treated by Rao et al. [3] under a less general model, while offspring-excluded parent pairs (OET) were introduced by Rao et al. [4] for parentage exclusions. The remaining 13 cases are new to path analysis: AST, AMT, and AHT are exactly like the corresponding pairs of children reared together (SST, MZT, and HST), except that the individuals are observed as mature adults, which has the effect of replacing c by cy and h by hz. The other 10 new cases will be considered in later sections.

Half-sibs present special problems. It is assumed that multiple spouses are as similar to each other in genotype and childhood environment as to their common partner, and that the environments of half-sibs reared together are as similar as those of full sibs. For HST one child (X) is reared by his true parents, while the other (Y) is reared by the common parent and a step-parent. For HSP and HSA the environments are assumed to be uncorrelated, and for adopted children the foster parents are not related to the true parents. HSA is best realized by an unmarried woman whose children by different fathers are placed for adoption at birth. HSS is strictly possible only with polygamy but may be closely approximated by sequential monogamy. The correlation between family environments was previously a nuisance parameter, but it is derived here from the general model.

For OET [4] the excluded parent is assumed to cohabit with the family as social parent from the child's birth or before. The exclusion may either be acknowledged on interview or preferably revealed by genetic evidence.

MORE REMOTE RELATIVES

The assumption for half-sibs that multiple spouses are as similar to each other as to their common partner may be extended to spouses of relatives with kinship ϕ, whose other correlations are therefore assumed to be $s\phi$, $m\phi$, and $u\phi$. A testable corollary of this assumption is that an individual is no more or less similar in genotype and childhood environment to a spouse's sib than to the latter's spouse. The correlational paths between two relatives with kinship ϕ and their spouses are shown in figure 3, which can be completed by adding all the causal paths to descendants. Expected correlations for a given pair of relatives can then be derived

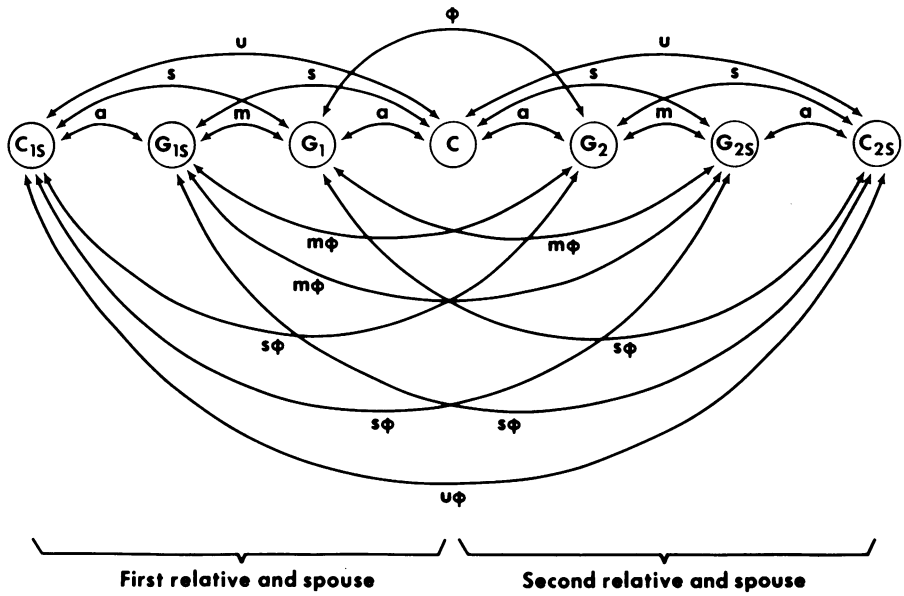

FIG. 3.—The 21 correlational paths for two individuals (with kinship ϕ) reared together and their spouses. G_1 and G_2 are genotypes of the two individuals whose common environment is C. G_{1S}, C_{1S} refer to the spouse of G_1, and G_{2S}, C_{2S} refer to the spouse of G_2.

by specifying ϕ. For example, for uncle-niece, first-cousin, and in-law pairs, the two relatives whose kinship is ϕ are full sibs, hence $\phi = (1 + m)/2$. These relations can be treated as in figure 2, by deriving the quantities s^*, t^*, u^*, etc., from the completed figure 3. This yields the same genetic correlations for uncle-niece and first-cousin pairs as phenotypic assortative mating [1], except for omission of dominance deviations and replacement of $1 + 2m + m^2$ by $1 + 3m$ for first cousins. Although our treatment of assortative mating is more general, allowing for cultural as well as biological inheritance, it does not underestimate the genetic component. Six relationships in table 4 arise from in-law, uncle-niece, and first cousin pairs.

CHILDREN OF MZ TWINS

Nance and Corey [8] advocate the use of relatives of MZ twins for testing maternal effects and estimating heritability under a model which omits genotype-environment covariance. In the absence of maternal effects the main differences from nuclear families are the greater effort required to collect data, and the assumption that inferences about inheritance are best made from multiple births. Nance and Corey proposed an approximate theory for variance components, but more reliable estimates can be obtained from maximum likelihood analysis of z-transformed correlations (see below). Spouses of MZ twins are assumed to be as similar in genotype and childhood environment as spouses of a single individual,

and the environments of their children are assumed as similar as in half siblings. The last four relationships in table 4 arise from children of MZ twins. These correlations are derived from figure 3 by setting $\phi = 1$.

STATISTICAL ANALYSIS AND SAMPLING DESIGN

Rao et al. [3] developed maximum likelihood analysis of z-transformed correlations, assumed independent. Later this assumption was relaxed [7], but use of a large number of relationships in studies of family resemblance makes independence a reasonable approximation. These references should be consulted for details of the method. In brief, correlations are normalized to a close approximation by z transforms [9]. The corresponding logarithmic likelihood is then maximized by Newton-Raphson or Fletcher-Powell iteration, yielding estimates, tests of hypotheses, and standard errors. Estimates may be converted into components of variation and their large-sample standard errors. Inferences based on standard errors, however, are less reliable than those based on the logarithmic likelihood. When we say that an estimate is or is not significantly different from some expectation, we always refer to the likelihood ratio test, not to standard errors. These principles, applied through the computer program NUVAR,* are illustrated in the next two sections.

Systematic use of a family environmental index gives a large number of correlations with different expections. When attention is limited to nuclear families (FMT, OPT, and SST) there are 13 such equations and only 10 parameters to be estimated in the general case. Therefore other relationships, rarer or more difficult to collect, should serve primarily as a test of consistency for conclusions from nuclear families. Adult sibs (AST), which are usually overlooked, are especially valuable. Parental exclusions (OET) help to determine the effect of family environment. Half-sibs and uncle-niece pairs, both cognate and affinal, determine validity of the model outside nuclear families. With such a wealth of common relationships it is amazing that twins have attracted so much attention, given the special features of their placentation and upbringing. We suspect that many investigators were attracted to twins because the analysis seemed simple. Perhaps it is time to suggest that, for its contribution to biometrical genetics, twin research might profitably be left to twins.

INHERITANCE OF I.Q.

Rao et al. [3] summarized American data on I.Q. for 10 relationships, to which we may now add the marital correlation of .5 based on 887 pairs [10]. Analysis by the methods used here shows that the genetic correlation of mates (m) is not significantly different from 0, and that all of the marital correlation may be due to preference for a spouse from the same environment (table 5). The effect of parental childhood environment or the effect of parental adult phenotype on the child's environment may be null, but not both simultaneously. Previously we wrote [3]:

* Copies and descriptions of the program, which follows the prototype COMVAR [3], are available from the authors.

TABLE 5

ANALYSIS OF I.Q. OF TRUE AND FOSTER CHILDREN (TABLE 11 OF [3])

Hypothesis	df	χ^2	h	c	y	z	m	u	s	f	x	i	a
Present analysis:													
$m=s=0$	3	2.71	.819 ± .041	.306 ± .073	2.324 ± .547	.561 ± .137	0 ...	.985 ± .480	0 ...	.274 ± .185	.243 ± .222	.858 ± .165	.201 ± .152 ...
$m=s=x=0$	4	3.88	.835 ± .036	.423 ± .044	2.165 ± .383	.668 ± .115	0 ...	.595 ± .246	0 ...	.406 ± .072	0 ...	.752 ± .149	0 ...
$m=s=x=0$, $z=1$	5	9.38	.757 ± .034	.496 ± .032	2.165 ± .382	1 ...	0 ...	.434 ± .165	0 ...	.284 ± .032	0 ...	.642 ± .116	0 ...
$m=s=f=0$	4	3.60	.789 ± .033	.266 ± .054	2.686 ± .716	.468 ± .153	0 ...	.980 ± .466	0 ...	0 ...	.577 ± .135	.812 ± .133	.363 ± .077 ...
$m=s=f=x=0$..	5	81.32	.835 ± .036	.424 ± .044	2.165 ± .382	1.388 ± .135	0 ...	.595 ± .246	0 ...	0 ...	0 ...	.752 ± .149	0 ...
			h	c	k	z	m					i	a
Previous analysis [3]:													
$m=0$			.822 ± .035	.286 ± .044	2.402 ± .691	.402 ± .242	0 ...	... ...	... ...	... ...	... ...	.902 ± .119	.231 ± .138 ...

"Gene-environment correlation may be appreciable ($r = .326 \pm .195$) but is barely significant ($\chi^2_1 = 6.85 - 2.80 = 4.05$). This provides modest support for the hypothesis that class differences in mean I.Q. have a genetic component. Good designs and enormous samples would be necessary (and might not be sufficient) to establish with precision a genetic component in class (or race) differences." With the more general analysis considered here, gene-environment correlation no longer approaches significance ($\chi^2_1 = 3.88 - 2.71 = 1.17$), although its estimate does not change much (note that $x = s = 0$ implies $a = 0$).

The correlation between midparent genotype and family environment of children (r) is derived from figure 1 by adding two causal paths of value $1/\sqrt{2(1 + m)}$ from each parental genotype to the midparent genotype; we get $r = a\sqrt{2/(1 + m)}$, so our estimate of r (for $m = s = 0$) corresponds to $r = .284 \pm .215$, in close agreement with the previous analysis. Adult heritability remains significantly less than heritability in childhood, presumably because the leveling effect of the school system is replaced by varying stimulation in different occupations. The effect of family environment is significantly greater for adults than children. Under the present model, the causal path is from childhood environment to adult I.Q. Since family environment is so important, it is conceivable that adult education of parents could, by diminishing the intergenerational path between family environments, have greater effects on academic performance than preschool education of their children [10].

Apart from the above substantive conclusions, the I.Q. data are remarkable for their low power (tables 6 and 7). Genetic correlation of mates and gene-environment correlation are not significant, but neither are substantial values excluded. Because of the large standard errors even a considerable increase in the quantity of data would not resolve these uncertainties. The main defect is that these data depend on rare relationships and fail to make systematic use of information available in environmental indices and adult sibs in nuclear families. If indices of parents and children are determined, uncertainty about the magnitude of gene-environment correlations (s, u), the genetic correlation of mates (m), and the causal paths which determine family environment can be dispelled.

In recent years disturbing questions have been raised [11] about the British data

TABLE 6

VARIANCE COMPONENTS FOR I.Q. ($m = s = 0$) AS FRACTIONS OF TOTAL PHENOTYPIC VARIANCE

Source	Children	Parents
Genotype*	.670 ± .067	.211 ± .104
Common environment†	.094 ± .045	.506 ± .245
Genotype-environment covariance‡	.101 ± .028	.132 ± .039
Residual	.135 ± .016	.151 ± .131

* h^2 or h^2z^2
† c^2 or c^2y^2
‡ $2hca$ or $2hczya$

TABLE 7

VARIANCE COMPONENTS FOR CHILD'S COMMON ENVIRONMENT ($m = s = 0$) AS FRACTIONS OF TOTAL VARIANCE (fig. 1)

Source	
Parental childhood environments*	$.298 \pm .205$
Parental phenotypes†	$.176 \pm .162$
Parental phenotype-environment covariance‡	$.400 \pm .107$
Residual	$.126 \pm .216$

* $2f^2(1 + u)$
† $2x^2(1 + mh^2z^2 + 2shzcy)$
‡ $4fx[cy(1 + u) + hz(s + a)]$

of Sir Cyril Burt, and the American data which we used have not escaped criticism [12]. Most of these studies are 40 years old and were collected when the times were more favorable to such research. Since sample design and analysis (apart from the brilliant paper of Wright [2]) were primitive, it is inevitable that these studies can be faulted now. However, there is remarkable agreement between the observations and a simple model of biological and cultural inheritance ($\chi^2_3 = 2.71$). Surely gross errors would be erratic in direction and magnitude, and the close agreement of all relations would not be observed. The burden of proof is clearly on critics of these data to show that different results would be obtained with the more rigorous and powerful designs available today.

CULTURAL INHERITANCE

Cavalli-Sforza and Feldman [13] introduced a model in which cultural and biological inheritance were both determined by a single locus. They simulated a number of cases under random mating, with random adoption, the same genetic and environmental effects for parents and children, and the effect of parent's environment on child's environment mediated only through parental phenotype. The inclusion of genotype-environment interaction in their model and the various inconsistencies between the model as described and their simulation results were initially intriguing. A selection of their results, including all discrepant cases, was analyzed under our new model (table 8). Note for example case BCPV4, where randomly adopted twins are correlated although the genotype means are equal.

Discrepancies due to dominance, gene-environment interaction, and unexplained factors are reflected by large values of χ^2, which was calculated on the assumption of 1,000 pairs for each of seven relationships. This is an unrealistically large body of data, especially for identical twins reared apart, and failure of our model might not be detected in a smaller body of data. However, the interesting point in these simulated data is not the power to detect discrepancies, but rather that h^2 as calculated never overestimates broad heritability, whether defined by the H of Cavalli-Sforza and Feldman or by r_{AA} for identical twins reared apart. Even $h^2 + 2hca$, which includes gene-environment covariance, never appreciably overestimates broad

TABLE 8

ANALYSIS OF SIMULATED DATA OF CAVALLI-SFORZA AND FELDMAN ($m = s = u = f = 0$, $y = z = 1$)

Case	Reason for Choice	h	c	x	a	χ^2	df	Biological Inheritance				Cultural Inheritance			
								H	r_{AA}	h^2	$h^2 + 2\,hca$	C	$r_{MZ} - r_{AA}$	c^2	$c^2 + 2\,hca$
B4	dominance, $c = 0$	.641	0	0	0	10.47	6	.429	.429	.411	.411	0	0	0	0
BC3	discrepant	.464	.479	.773	.569	13.68	4	.436	.366	.215	.468	.250	.320	.229	.482
BC4	dominance, $c > 0$	.680	.298	0	0	69.01	4	.474	.460	.462	.462	.065	.080	.089	.089
BCPV1	discrepant	.667	.379	.634	.556	20.36	4	.707	.679	.445	.726	.159	.187	.144	.425
BCPV2	$z \neq 1$	.623	.513	.556	.484	20.97	4	.739	.586	.388	.698	.222	.375	.263	.573
BCPV3	discrepant	.742	.349	.337	.284	18.52	4	.668	.694	.551	.698	.151	.125	.122	.269
BCPV4	$h = 0$	0	.436	.596	0	1.03	5	0	.018	0	0	.206	.187	.190	.190
BCPV8	discrepant	.692	.376	.608	.545	15.31	4	.777	.715	.479	.763	.126	.188	.141	.425
BCPV9	dominance, $a > 0$	.655	.516	.479	.417	49.66	4	.769	.604	.429	.711	.210	.375	.266	.548
BCPV10	discrepant	.756	.337	.371	.321	13.58	4	.722	.723	.572	.736	.126	.125	.114	.278

heritability. We leave it as a problem for those who stress gene-environment interactions to devise a counter example.

Rao and Morton [6] suggested two measures of broad cultural inheritance, denoted by C and $r_{MZ} - r_{AA}$, the difference in correlation of identical twins reared together and apart. The narrow estimate c^2 never appreciably exceeds either measure of broad cultural heritability, although $c^2 + 2hca$ sometimes does. We conjecture that the bounds would be even tighter if the only discrepancies in the Cavalli-Sforza and Feldman data were the explicable ones due to dominance and gene-environment interaction. In any event, analysis of these extreme examples supports our contention that the general model cannot exaggerate genetic factors, even when its assumptions are violated by gene-environment interaction.

One type of interaction could be a source of confusion. Suppose that, as may well be the case, genotypes choose environments which favor their best expression, thereby inducing both a gene-environment covariance (which is included in our model) and a greater heritability than under an environment which proscribed liberty of choice. This is only a special case of the proposition that genetic parameters such as allele frequencies, selection coefficients, and variance components are spatiotemporally limited to the populations from which they were derived [14].

DISCUSSION

The function of path analysis is to explain correlations, not the residual variances which include gene-environment interactions, and unanticipated perturbations are best allowed for by tests of goodness of fit. However, nonrandomness of environmental effects should be expected, not only in man but also for domestic and laboratory animals and those field plot designs of plant geneticists which allocate seed from one plant to the same row [15]. Fisher's seminal paper of 1918, written before his work on experimental design at Rothamsted, used for illustration human data in which randomization could be imagined but not realized. Today the geneticist who fails to differentiate between environment common to children and parents and ascribes any excess of sib correlation over parent-offspring correlation to dominance must defend his integrity and intelligence. There can be no dialogue between genetics and the social sciences unless the former makes adequate allowance for cultural inheritance, and the latter accepts quantitative models and goodness of fit tests. A model which allows for all possible marital correlations between environments and genotypes has no hereditarian or environmentalist bias and hopefully provides a common ground for geneticists and social scientists of different persuasions, if they are able to separate their pretensions as scholars and political philosophers.

SUMMARY

Analysis of family resemblance is developed in terms of three genetic parameters, six parameters for cultural inheritance, and one parameter for an index estimating family environment. With efficient use of nuclear families the model is fully determinate. Other biological and social relationships provide additional degrees of free-

dom for testing goodness of fit. Performance of the model is satisfactory on simulated data with extreme gene-environment interaction.

Applied to a large body of published data on I.Q., neither genetic assortative mating nor gene-environment covariance is significant by a likelihood ratio test, but heritability is less and cultural inheritance is greater for adults than children. Whereas family resemblance of children is largely genetic, for adults it is largely due to their childhood environments, presumably acting on occupational aspirations. Further resolution is more likely to come from nuclear families than from the rare relationships that were favored by classical human genetics.

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