#455-77

INSTITUTE FOR RESEARCH ON POVERTY DISCUSSION PAPERS

PITFALLS IN THE RESOLUTION OF IQ INHERITANCE

Arthur S. Goldberger

UNIVERSITY OF WISCONSIN ~ MADISON

Pitfalls in the Resolution of IQ Inheritance

Arthur S. Goldberger

November 1977

This paper was presented at the Honolulu Conference on Genetic Epidemiology, October 17-18, 1977. It will be published in N.E. Morton and C.S. Yung, eds., Genetic Epidemiology (New York: Academic Press, 1978).

PITFALLS IN THE RESOLUTION OF IQ INHERITANCE

Arthur S. Goldberger

Department of Economics
University of Wisconsin
Madison, Wisconsin

I. INTRODUCTION

As a social scientist with no training in genetics, I am grateful for the opportunity to discuss the resolution of IQ inheritance at this conference. I am filled not only with gratitude but also with trepidation. For Morton & Rao (1977) say that

> The literature on inheritance of intelligence has suffered from ... domination of the field by psychologists and sociologists with primary interests and competence outside genetics,

and Rao, Morton, & Yee (1976, p. 241) have written that

> There can be no dialogue between genetics and the social sciences unless ... the latter accepts quantitative models and goodness of fit tests.

But the interests of science dictate that neither my gratitude nor my trepidation should stand in the way of my being rude on this occasion.

In a longer paper (Goldberger, 1977) I have undertaken a detailed analysis of both the Birmingham and Honolulu schools' efforts at fitting models to observed kinship correlations for IQ.

The Birmingham school is represented by the articles of Jinks & Eaves (1974) and of Eaves (1975), who modify the classical model of R. A. Fisher. Fisher specified phenotypic assortative mating, permitted non-additive gene effects, ruled out gene-environment correlation, and also ruled out environmental resemblance among persons living together. As a consequence of this last specification, Fisher's model would be rejected out of hand by any set of IQ kinship correlations which includes adoptive families. So the Birmingham school modifies Fisher's model by introducing a common environmental component which is shared by siblings, and by parents and children, living together, whether those kin be biological or adoptive.

But the Birmingham model is logically untenable because the consequences of the shared environment are not fully taken into account. It is essentially impossible to construct a causal model which will produce the Birmingham formulas for kinship correlations, if only because the conjunction of phenotypic assortative mating and environmental transmission from parents to children will generate gene-environment correlation: Goldberger (1977, pp. 36-37, A1-A15). Few social scientists would try to publish a set of derived equations, like the Birmingham formulary, without first specifying a causal system from which those equations could be derived. Fewer still would succeed.

I now turn to the Honolulu school.

II. THE HONOLULU VENTURE

The Honolulu school is represented by the series of articles in the *American Journal of Human Genetics* -- Morton (1974), Rao, Morton, & Yee (1974, 1976) -- and by the recent papers of Morton & Rao (1977) and Rao & Morton (1977). They build upon the remarkable work of Sewall Wright, and proceed from an explicit, logically tenable, and internally consistent, causal model, the heart of which is captured in the path diagram on p. 229 of Rao, Morton, & Yee (1976). Assortative mating is on the basis of common environment and genotype (rather than on the basis of phenotype). Parents transmit environments as well as genes to their children (who also share additional common environment). Hence environmental resemblance among relatives, and gene-environment correlation, are provided for. Non-additive gene effects, on the other hand, are ruled out. To assist in the resolution of the determinants of IQ, the model also incorporates a second phenotype, an index of family environment (e.g. socioeconomic status). A sharp distinction is permitted between the determination of IQ in childhood and the determination of IQ in adulthood.

The model has 10 free parameters, which I find convenient to specify as follows:

$\underline{c}$ = path from common environment to child's phenotype,
$\underline{h}$ = path from genotype to child's phenotype,
$\underline{p}$ = path from common environment to adult's phenotype,
$\underline{q}$ = path from genotype to adult's phenotype,

f = path from parent's common environment to child's common environment,
x = path from parent's phenotype to child's common environment,
u = correlation between environments of spouses,
m = correlation between genotypes of spouses,
s = correlation of one spouse's genotype with the other spouse's common environment,
i = path from common environment to index.

Several derived parameters also appear:

$$a = (s(f + px) + qx(1 + m))/(1 - f - px)$$

= correlation between an individual's genotype and his common environment,

$$\theta = (1 - 2\,cha)^{-\frac{1}{2}}$$

= phenotypic standard deviation for adopted children.
My notation follows that of Rao, Morton, & Yee (1976) except that I use p and q in place of their cy and hz.

Various versions of this model have been applied to IQ data by the Honolulu group. Their venture into the IQ debate began modestly enough. Morton (1972) told us that

> recent controversy about ethnic differences in behavior is based on two fallacies: first, that a reliable estimate of heritability can be obtained when the environment is not random; secondly, that heritability is relevant to educational strategy.

Morton (1974, pp. 320-321) told us that

> each type of relationship introduces another equation and another assumption, generally in the direction of overestimating heritability,

and that

> While heritability of IQ in man has usually been calculated as greater than .5, ... it would not be possible to argue strongly against a smaller value.

He went on to say (p. 327) that

> one would be quite unjustified in claiming that heritability is relevant to educational strategy.

Within two years, however, Rao, Morton, & Yee (1976, p. 238) had estimated IQ heritability to be .67 ± .07 for children and .21 ± .10 for adults. And this striking difference had led them to conceive the possibility that

> adult education of parents could ... have greater effects on academic performance than preschool education of their children.

Nowadays Rao, Morton, & Yee (1977) are telling us that

> the biological and cultural factors involved in the inheritance of IQ are resolved,

and Morton & Rao (1977, p. 38) close off their discussion of "Quantitative inheritance in man" with this announcement:

> The "nature-nurture" controversy was partly an ideological confusion of individuals and populations, partly a methodological problem in distinguishing cultural and biological causes of family resemblance. As far as that problem has been formulated, it has been solved.

What happened between 1974 and 1976? A rude person might describe the transition, from skepticism to true belief, as a comedy of errors.

III. THE HONOLULU MODELS

The data set analyzed by Rao, Morton, & Yee (1976) was drawn from the compilation in Jencks (1972) and from Burks (1928), and is displayed below. For each kinship, r and n denote the observed correlation and the number of paired observations on which it is based.

Table 1. Data Set Analyzed by Rao, Morton, & Yee

	Variables correlated	r	n
1.	IQs of identical twins	.89	50
2.	IQs of separated identical twins	.69	19
3.	IQs of siblings	.52	2001
4.	IQs of adopted-adopted siblings	.23	21
5.	IQs of adopted-natural siblings	.26	94
6.	IQ of adopted child and his index	.25	186
7.	IQ of child and his index	.44	101
8.	IQ of parent and child's index	.69	205
9.	IQs of adoptive parent and child	.23	1181
10.	IQs of parent and child	.48	1250
11.	IQs of spouses	.50	887

To this data set they fit five variants of their basic model. The most general of those, which I refer to as H1, sets $m = s = 0$, so that spouses' IQs correlate only because the spouses had similar common environments in their youth. From the elaborate formulary in Rao, Morton, & Yee, we can set out the equations of this model as in the table below, in which the various kinships are labelled by acronyms.

Table 2. Equations of Model H1

1.	MZTXY	$h^2 + c^2 + 2cha$
2.	MZAXY	$\theta^2 h^2$
3.	SSTXY	$\frac{1}{2}h^2 + c^2 + 2cha$
4.	FSTXY	$\theta^2 c^2$
5.	FSPXY	$\theta(c^2 + cha)$
6.	SSAXIX	θic
7.	SSTXIX	$i(c + ha)$
8.	OPTXIY	$i\,[f(p(1 + u) + qa) + x(1 + p^2u)]$
9.	OFPXY	$\theta c[f(p(1 + u) + qa) + x(1 + p^2u)]$
10.	OPTXY	$c\,[f(p(1 + u) + qa) + x(1 + p^2u)] + \frac{1}{2}h(pa + q)$
11.	FMTXY	p^2u

With $m = s = 0$, H1 has 8 free parameters, and the formula for the derived parameter a reduces to

$$a = qx/(1 - f - px).$$

The four other variants of the model, which I refer to as H2 - H5, involve additional restrictions beyond $m = s = 0$, namely:

H2: $x = 0$; H3: $x = 0,\ q = h$; H4: $f = 0$;

H5: $x = 0,\ f = 0$.

The estimation procedure is essentially as follows. Let $\rho_j = \rho_j(\theta)$ denote the expected correlation for the j-th kinship, where θ denotes the set of K free parameters; and let r_j denote the observed correlation for the j-th kinship. The corresponding z-transforms are

$$\zeta_j = \tfrac{1}{2}\log((1 + \rho_j)/(1 - \rho_j)) = \zeta_j(\theta),$$

$$z_j = \tfrac{1}{2}\log((1 + r_j)/(1 - r_j)).$$

For a data set with $j = 1, \ldots, N$ kinships, choose θ to minimize the weighted least squares criterion

$$\chi^2 = \Sigma_{j=1}^{N} n_j(z_j - \zeta_j(\theta))^2.$$

The value of the criterion when minimized is referred to a chi-square distribution with degrees of freedom equal to $N - K$. In fact, the Honolulu group works with "bias-corrected" z-transforms: Rao, Morton, & Yee (1974, pp. 331-332); Rao, MacLean, Morton, & Yee (1975, pp. 519-520); Rao, Morton, Elston, & Yee (1977, p. 150). But I ignore this refinement, which seems to have only negligible impact on the results.

The parameter estimates reported by Rao, Morton, & Yee (1976) for the models H1 - H5 are here displayed in Table 3 along with some auxiliary statistics.

Table 3. Parameter Estimates by Rao, Morton, & Yee

	H1	H2	H3	H4	H5
c	.306	.423	.496	.266	.424
h	.819	.835	.757	.789	.835
p	.711	.916	1.074	.714	.918
q	.459	.558	.757	.369	1.159
f	.274	.406	.284	0	0
x	.243	0	0	.577	0
u	.985	.595	.434	.980	.595
i	.858	.752	.642	.812	.752
χ^2	2.71	3.88	9.38	3.60	81.32
d.f.	3	4	5	4	5
a	.201	0	0	.363	0
θ	1.055	1	1	1.086	1

They emphasize the good fits of the models to the data, saying (p. 239) that

> There is remarkable agreement between the observations and a simple model of biological and cultural inheritance ($\chi_3^2 = 2.71$).

For the H1 model, they translate their estimates into decompositions of phenotypic variances, as summarized in Table 4, and emphasize the contrast between the estimates of p, q on the one hand and c, h on the other hand, saying (p. 242),

> 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.

Table 4. Estimated Variance Components for Model H1

Source	Adult IQ		Child IQ	
Genotype	$\underline{q}^2$ =	.211	$\underline{h}^2$ =	.670
Common environment	$\underline{p}^2$ =	.506	$\underline{c}^2$ =	.094
Covariance	2 $\underline{pqa}$ =	.132	2 $\underline{cha}$ =	.101
Residual		.151		.135
Total		1.000		1.000

IV. THE HONOLULU ARITHMETIC

If one takes the H1 parameter estimates from Table 3, inserts them into the equations of Table 2 to get predicted correlations, and compares those predicted correlations with the observed correlations in Table 1, one finds a chi-square value substantially larger than that reported by Rao, Morton, & Yee. Similarly for models H2 - H5. When I did that exercise in June, I was using a pocket calculator, and hence recording some intermediate results. I happened to note that the estimated value of the quantity $\underline{i}(\underline{p} + \underline{qa})$ remained constant over the five models, and that its constant value was precisely the observed value of the 8-th correlation. That led me to conjecture that the authors had accidentally used $\underline{i}(\underline{p} + \underline{qa})$ as the equation for the 8-th correlation in setting out their models for estimation. My conjecture proved to be correct, leading me to conclude that they had fitted five non-models. Consequently their numerical results, interpretations thereof, and policy recommendations could be disregarded.

Wondering what would happen if the error was corrected and the models refitted, I set out to program the models myself. As I was transcribing the 11 equations, I happened to note that the same long expression in square brackets appeared in the 8-th, 9-th, and 10-th equations. I gave it a single symbol to economize on writing. As I was doing so, it dawned on me that I was writing the H1 model in terms of only 7 free parameters, rather than the 8 free parameters used in the Honolulu formulation. The parsimonious reformulation of the H1 model is displayed in Table 5, with some symbols defined at the bottom of the table.

Table 5. Reformulated Equations of Model H1

1.	MZTXY	$h^2 + c^2 + 2\,cha$
2.	MZAXY	$\theta^2 h^2$
3.	SSTXY	$\tfrac{1}{2}h^2 + c^2 + 2\,cha$
4.	FSTXY	$\theta^2 c^2$
5.	FSPXY	$\theta(c^2 + cha)$
6.	SSAXIX	θic
7.	SSTXIX	$i(c + ha)$
8.	OPTXIY	it
9.	OFPXY	θct
10.	OPTXY	$ct + \tfrac{1}{2}hv$
11.	FMTXY	w

$a = qx/(1 - f - px)$

$\theta = (1 - 2\,cha)^{-\frac{1}{2}}$

$t = f(p(1 + u) + qa) + x(1 + p^2u)$

$v = pa + q$

$w = p^2u$

Being a social scientist, I realized that the ability to rewrite a given model in terms of fewer free parameters has an immediate consequence: the original set of parameters is indeterminate, nonestimable, or in social science jargon, underidentified. For H1, it is a well-defined problem to choose a set of values for the 7 parameters c, h, a, i, t, v, w to best fit the 11 observations, and the least-squares principle will produce a unique solution. But it is not a well-defined problem to choose a set of values for the 8 Honolulu parameters c, h, p, q, f, x, u, i to best fit the

11 observations, and the least-squares procedure will not produce a unique solution, there being an infinity of distinct solutions which fit equally well.

It is easy to verify from Table 5 that, of the 8 Honolulu parameters in H1, only c, h, i are determinate: p, q, f, x, u cannot be extracted from a, t, v, w. The paths leading into adult IQ are confounded with the paths of environmental transmission from parents to children. Similar considerations apply to models H2 - H5: in no case is the Honolulu parameter set fully determinate in terms of the present data set. In particular, p is never determinate so that the contrast between the decomposition of variances at the adult and child level cannot be sustained. On this count alone, preschool educators may relax; their jobs are not in jeopardy.

What happens when the reparameterized H1 model is fitted? The results, kindly provided to me by the Honolulu group in July, are as follows, in terms of my parameterization:

$$c = .286, \quad h = .823, \quad a = .228, \quad i = .903,$$

$$t = .762, \quad v = .646, \quad w = .501,$$

with a chi-square value of 3.15 on 4 degrees of freedom. Readers can trace out the combinations of values of p, q, f, x, u, which are compatible with these estimates.

There is another problem with the Honolulu modeling of the present data set, which I had pointed out to them several months earlier. In the Newman, Freeman, & Holzinger (1937) study of separated identical twins, which is the sole source of their MZAXY observation, most of the twins had been tested as adults. The logic of the Honolulu causal model indicates that the appropriate equation for such pairs is not θ^2h^2, but rather θ^2q^2. The Honolulu group realized, as I had not, that making this change would remove the indeterminancy in the H1 model. For, the MZAXY correlation would now isolate q, and permit the remaining unknowns, p, f, x, u to be extracted from a, t, v, w. In September, Rao, Morton, & Yee (1977) reported on the fitting of this new model, which I refer to H1*.

Their parameter estimates for H1* are:

$$c = .290, \quad h = .843, \quad p = .707 \quad q = .566$$

$$f = .290, \quad u = 1.000, \quad x = .179, \quad i = .969,$$

with a chi-square of 6.45 on 3 degrees of freedom. In terms of our formulation, these estimates correspond to $a = .174$, $t = .707$, $v = .688$, $w = .499$. With this set of estimates for H1* in hand, Rao, Morton, & Yee (1977) decide that

> the biological and cultural factors involved in
> the inheritance of IQ are resolved,

while recognizing that p and u are closely correlated and thus poorly resolved.

Several features of their latest analysis of the 11-observation data set are worth noting:

(1) The burden of resolving the determinants of adulthood IQ rests squarely on the slim shoulders of the MZA observation, based on a sample of size 19. Rao, Morton, & Yee (1976, p. 236) had suggested that "twin research might profitably be left to twins."

(2) The fit has deteriorated so that there is no longer "remarkable agreement between the observations and a simple model". Indeed the chi-square is significant at the 10% level.

(3) With $u = 1$, the common environments of spouses are perfectly correlated: by the time that they walk down the aisle together, the typical bride and groom have shared as much IQ-relevant environmental experience as identical twins who have been raised together since birth.

(4) The estimates are wrong.

To verify the last point, it suffices to recognize that the only difference between the H1* model and the corrected-H1 model lies in the 2d equation where q replaces h. Take the corrected H1 estimates provided by the Honolulu group in July, set $q = h = .823$, and solve for p, f, x, u from a, t, v, w. Inserted into the H1* formulary, these will produce the same predicted correlations for all kinships as they did when used in the corrected-H1 formulary. Hence they will produce the same chi-square, namely 3.15. This being less than 6.45 establishes that the latest Honolulu estimates are wrong.

Upon refitting the H1* model myself, I find that the best-fitting parameter values are

$c = .285$, $h = .835$, $p = -.782$, $q = .789$

$f = -.159$, $u = .817$, $x = .375$, $i = .906$.

These produce a chi-square of 2.61 with 3 degrees of freedom. The fit is excellent, but the signs of p and f are quite implausible.

It is hard to share the Honolulu group's confidence in their resolution of the biological and cultural factors involved in the inheritance of IQ.

V. THE HONOLULU DATA

Throughout the comedy of errors, the estimates of c and h have remained relatively constant. It is tempting to conclude that the Honolulu venture has at least succeeded in resolving the determinants of *childhood* IQ. That too would be a mistake.

The structure of the Honolulu models suggests that the estimates of the childhood parameters are heavily dependent on just four observations, the IQ correlations for: identical twins raised together (MZTXY), biological siblings raised together (SSTXY), pairs of adopted children raised together (FSTXY), and pairs of children raised together, one being adopted and the other biological (FSPXY). My conjecture is that that is indeed the case, the remaining equations and observations being irrelevant window-dressing as far as the childhood components of variance are concerned.

For convenience let us confine attention to the cases where $a = 0$ as in H2, H3, H5. The equations for the four key kinships are displayed below along with the observed values used by Rao, Morton, & Yee (1976):

$$\begin{aligned} \rho_1 &= h^2 + c^2, & r_1 &= .89 \\ \rho_3 &= \tfrac{1}{2}h^2 + c^2, & r_3 &= .52 \\ \rho_4 &= c^2, & r_4 &= .23 \\ \rho_5 &= c^2, & r_5 &= .26 \end{aligned}$$

With this specification, each of the following contrasts provides an estimate of $\frac{1}{2}h^2$: $r_1 - r_3$, $r_3 - r_4$, $r_4 - r_5$. With this data set, doubling each contrast in turn gives these estimates of h^2: $2(.89 - .52) = .74$, $2(.52 - .23) = .58$, and $2(.52 - .26) = .52$. When account is taken of their respective sample sizes, these three estimates average out to just about .67, the full model's estimate of h^2. My conjecture is that the same mechanism will essentially operate regardless of the specification of the remainder of the model.

If so, and if our main concern is with the determinants of childhood IQ, it is essential to have a look at the specification, and at the observed values, employed in those contrasts.

First, consider the contrast between $\underline{r}_1$ and $\underline{r}_3$, that is between MZT and SST. For this contrast to estimate $\frac{1}{2}\underline{h}^2$ requires that identical twins (who are of the same age and sex) have no more environmental similarity than ordinary siblings (who may differ in age and sex). Evidence against that specification can be found in the very source material on which the Honolulu group drew, namely Jencks (1972, pp. 286, 287, 289). To get their figure for $\underline{r}_3$, namely .52, Rao, Morton, & Yee (1974) combined one study of same-sex fraternal twins ($\underline{r}$ = .63), with the mean of seven studies of ordinary siblings ($\underline{r}$ = .52). Those underlying figures suggest that identity of age and sex increases environmental similarity. Even the classical twin method applied here would compare the MZT figure, .89, with the DZT figure, .63, producing an $\underline{h}^2$ estimate of .52, in place of .74. And that reduction in $\underline{h}^2$, be it noted, would occur without any allowance for the possibility that MZTs share more environmental experience than same-sex DZTs. Nor is the MZT figure a fact of nature: Nichols (1970), as reported by Loehlin, Lindzey, & Spuhler (1975, p. 109), found $\underline{r}$ = .62 in his sample of 36 MZT pairs, so that with a judicious selection of samples, one could contrast the MZT figure of .62 with the DZT figure of .63, and arrive at an estimate of $\underline{h}^2 = -.02$.

Second, consider the contrast between $\underline{r}_3$ and $\underline{r}_4$, that is between SST and FST. To get their figure for $\underline{r}_4$, namely .23, the Honolulu group picked one adoptive study and discarded three others given in Jencks (1972, p. 291). Rao, Morton, & Yee (1974, p. 353) did so because the four studies together would be statistically heterogeneous, and would average up to $\underline{r}_4$ = .42, a value which is higher than their $\underline{r}_5$ = .26, such an ordering between FST and FSP being an anomaly in terms of their general model. With $\underline{r}_4$ = .42, be it noted, the contrast between $\underline{r}_3$ and $\underline{r}_4$ would have estimated $\underline{h}^2$ to be .20, in place of .58. Nor are these figures facts of nature. Scarr & Weinberg (1977, Table 6) report

$$\underline{r}_3 = .42, \quad \underline{r}_4 = .39, \quad \underline{r}_5 = .30$$

in some 100 families who have adopted black and interracial children. The Texas Adoption Project, according to Scarr (1977, p. 66), has

$$\underline{r}_3 = .37, \quad \underline{r}_4 = .22, \quad \underline{r}_5 = .30,$$

as the preliminary results in a sample of some 300 adoptive families. Readers are invited to construct their own estimates of $\underline{h}^2$ from these more recent data sets.

Rao, Morton, & Yee (1976, p. 234) told us that a critical assumption of their analysis is that "foster parents are random". This assumption is demonstrably false. Every adoption study shows that adoptive parents are well above population averages on IQ, education, income, occupational status, indeed on virtually every measurable variable which might be construed as an index of the environment conducive to intellectual development of children. For Burks's (1928) study, which the Honolulu group used, this point was documented at length by Goldberger (1976a,b).

Rao, Morton, & Yee (1974, p. 357) had told us that

> there are enough problems in human biometrical genetics without introducing biased selection, which is sufficiently protean to invalidate any path analysis, for which we regard random sampling as an essential condition.

If they believe that, they should abandon their venture, or at least stop using adoptive family data on IQ, for they will not find adoptive studies in which the general population has been randomly sampled.

Social scientists are familiar with the proposition that selection on one trait has consequences for the means, variances, and correlations of that trait, and of all other traits with which it is correlated. In the present context, there is every reason to believe that environmental variation is limited across adoptive families. If so, the empirical correlations of adopted children with their parents, and with their siblings, will be attenuated. Thus the Honolulu models are misspecified, in precisely the direction which leads to overestimation of heritability.

Social scientists are also acquainted with the proposition that to obtain unbiased estimates of population parameters from nonrepresentative samples, it is necessary to model the selection process itself. In the present context, that task is not an easy one. A start on it might be made by referring to Karl Pearson (1903). Translated into modern notation, Pearson's analysis tells us that when explicit selection on a single variable in a multinormal distribution takes place, reducing its variance by the ratio $\underline{b}$, then the variance-covariance matrix of the distribution changes from Σ to $\Sigma^* = \Sigma - \underline{b}\,\underline{d}\,\underline{d}'$, where $\underline{d}$ is the column of Σ corresponding to the explicit selection variable. For explicit selection on several variables, reference might be made to Lawley (1943).

VI. THE HONOLULU APPROACH

There is unlikely to be a serious dialogue between the Honolulu school and social scientists until the former begins to deal seriously with the nonrepresentativeness and heterogeneity which characterize the underlying sources of kinship data on IQ. In the interim I will take the opportunity to point out some other aspects of the Honolulu approach that came up as, over the past year, I worked through their articles and papers.

A. Data Sources

To construct their data set, Rao, Morton, & Yee (1974, pp. 352 - 354; 1976, p. 236) made no independent search of the literature but rather relied on Jencks's (1972) compilation. Consequently, they reproduce Jencks's errors: reversing the sample sizes for the two FMT correlations of Burks, incorrectly adjusting Willoughby's FMT correlation, discarding Outhit's FMT correlation because of a misunderstanding, -- and Jencks's arbitrary guesses at sample sizes. They also introduce some fresh errors -- treating the mean of seven observed sibling correlations as if it were a single observed figure in testing for heterogeneity in the SST category.

Relying on Jencks's compilation, Rao, Morton, & Yee have constructed a data set which draws only on studies conducted before 1940.

B. Statistical Methods

A methodological theme that runs through the Honolulu articles is that specification errors lead to poor fits. Rao, Morton, & Yee (1974, pp. 336-337, 356) write

> Failure of either assumption tends to give spuriously high estimates of heritability, an error that may in principle be detected by a goodness-of-fit test against other pairs of relatives ... In sufficiently large samples such discrepancies should be detected by significant deviations from our model.

Rao, Morton, & Yee (1976, pp. 230, 234) write

> A test of goodness of fit should reveal such discrepancies in a well-designed study ... The critical assumptions ... are best tested by residual χ^2 in an overdetermined system.

Morton & Rao (1977, p. 11) write

> If the general hypothesis is acceptable by a goodness-of-fit test ... there is little reason to distrust distributional or causal assumptions which are subsumed by the general hypothesis.

The grounds for their optimism are not apparent. A general principle is that specification errors lead to biased estimates but not necessarily to bad fits. Reliance on overdeterminancy is perilous when all, or most, of the misspecifications run in the same direction. (An analogous situation is familiar to many social scientists: In regression analysis it is assumed that disturbances are uncorrelated with the explanatory variables. Violation of that assumption, as in simultaneous-equation (= reciprocal-causation) models, makes least-squares estimates biased. But the estimates themselves contain no hint of the misspecification, because the calculated residuals are by construction uncorrelated with the explanatory variables). Another general principle is that hypothesis tests should not be accepted until their power against relevant alternatives has been established. I have seen no evidence that the Honolulu school has investigated the power of their tests for path analysis of continuous traits.

Morton & Rao (1977, p. 9) write

> In any case emphasis should be on goodness-of-fit tests in a rich body of data rather than on justification *a priori* of any hypothesis about family resemblance,

and Rao & Morton (1977) write

> However appealing a model may be, it is a simplification of nature and may always be stigmatized as an oversimplification ... To such criticism there is only one answer: a statistical test of goodness-of-fit, which in samples of adequate size and structure can rule out an inadmissible model.

It is instructive to see how these guidelines are employed in practice.

Rao & Morton (1977) fit an 8-parameter variant of their model to an expanded IQ data set, consisting of 16 observations. A chi-square of 39.94 results, which on 8 degrees of freedom, is highly significant. Rather than rejecting the model, they adopt a new criterion of goodness-of-fit. An F-ratio is invented in which the model chi-square is deflated by a heterogeneity chi-square, the latter measuring the variation, across the original studies, of the correlations which were pooled into the 16 observations. An F-ratio of .03 results, and so they adopt that model as "the parsimonious model for American I.Q.". Noting that the model seriously misfits MZA, FST, and FSP, they announce:

> These three observed correlations are highly elevated due to assortative placement, and it is a small wonder that these correlations are under-predicted by our model. Hence these discrepancies do not constitute evidence against our model.

They go on to say that

> We have shown that genetic analysis of I.Q. data is simple, determinate, and consistent over data sets.

Rao, Morton, Elston, & Yee (1977) suggest that working with *z*-transforms produces estimates and test statistics with desirable small-sample properties. These claims should be ignored. As far as I know, it has not been established, in the multivariate case, that the *z*-transforms are, in small samples, multinormally distributed. Furthermore, the equations of the models are nonlinear in the parameters, so that only asymptotic theory will be available. (Many social scientists are familiar with an analogous situation. In conventional regression analysis with normally distributed disturbances, least-squares estimates of the regression coefficients are minimum variance unbiased. But if the regression function is nonlinear in the parameters, the least-squares estimates have that property only asymptotically, their small-sample distributions being unknown).

C. Nuclear Families

Rao, Morton, & Yee (1976, pp. 238, 242) remark that

> The main defect [of their analysis of the 11 IQ observations] 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 ... Further resolution is more likely to come from nuclear families than from the rare relationships that were favored by classical human genetics.

The grounds for such optimism are not apparent. Consider Table 6, which sets out for nuclear families the equations of one version of the Honolulu model. In this version, which corresponds to H2, $m = s = x = 0$, so that $a = 0$ and $\theta = 1$.

There are 7 free paramters, which I take to be c, h, p, q, t, u, i; here $t = fp\ (1 + u)$. There are 10 correlations with distinct equations; Rao, Morton, & Yee (1976, p. 236) erred in counting 13.

Table 6. A Honolulu Model for Nuclear Families

	Variables correlated	Equation
1.	IQs of parents	p^2u
2.	IQ of parent with his index	ip
3.	IQ of parent with spouse's index	ipu
4.	IQs of parent and child	$ct + \frac{1}{2}hq$
5.	IQ of parent with child's index	it
6.	Indexes of parents	i^2u
7.	IQ of child with parent's index	ict/p
8.	Indexes of parent and child	i^2t/p
9.	IQ of child with his index	ic
10.	IQs of siblings	$c^2 + \frac{1}{2}h^2$

This model of 10 equations in 7 parameters makes 3 predictions about the observations; those are in fact the restrictions whose validity is examined by the chi-square test of goodness of fit. From the table, it is easy to see that those 3 restrictions are

$$r_1r_6 = r_3^2, \qquad r_2r_6r_7 = r_3r_8r_9, \qquad r_5r_6 = r_3r_8 .$$

Observe that the parent-child IQ correlation, r_4, and the sibling IQ correlation, r_{10}, do not enter those restrictions. The model makes no predictions about, and is compatible with any observations on, r_4 and r_{10}. Looking at the same point in another way, we see that those two correlations contain two parameters, h and q, which appear nowhere else in the model. As a consequence, the estimates of c, p, t, u, i will be independent of r_4 and r_{10}; and the estimates of h and q will adjust to perfectly fit r_4 and r_{10}, given the estimates of the other five parameters. Indeed the estimate of h will be independent of r_4.

This sort of preliminary algebraic analysis of the Honolulu system sheds light on the informational content of nuclear families with respect to heritability in children and adults. From this perspective, the Honolulu model looks more like a theory of the determination of environmental indexes than like a theory of the determination of IQ.

D. Environmental Indices

A distinctive feature of the Honolulu approach to modeling kinship correlations is the reliance on indices of common environment. Rao, Morton, & Yee (1974, p. 331) say that

> indeterminancy was resolved by combining path analysis with the concept of an index and a theory of hypothesis testing.

Rao, Morton, & Yee (1976, p. 236) say that

> Systematic use of a family environmental index gives a large number of correlations with different expectations.

Morton & Rao (1977, p. 4) write

> The concept of an index, defined by regression of phenotype on relevant environmental factors, is extremely flexible and powerful, often making indeterminate data yield a unique solution and greatly reducing standard errors of determined parameters .... Multiple relationships and indices are complementary, and combination of indices with a variety of relationships is the ideal design for separating genetic and cultural inheritance, leaving many residual degrees of freedom for testing the model.

For their IQ models the Honolulu group has indexed common environment by a single measure of the cultural level of the home (Rao, Morton, & Yee, 1976), and by father's occupation scaled into a measure of socioeconomic status (Rao & Morton, 1977). Rao & Morton (1977) tell us that

> Recent advances in analysis of family resemblance depend critically on indices ... The index of an individual may be created by regressing the phenotype on relevant observed variables which are not themselves products of the genotype ... Indices could be improved by including parental occupation as well as occupation, combining these and other socioeconomic indicators by regression with child's phenotype as the dependent variable. Like Burks's culture index this is likely to correlate highly with parental phenotype, but in a way that is

> accounted for by the estimate of <u>i</u> ... and does not disturb the rest of the analysis.

While social scientists may be happy to learn of the geneticists' interest in socioeconomic variables, they will be puzzled by the role assigned to them. In the Honolulu models for IQ, socioeconomic variables are taken to be mere fallible measures of the unobserved common environment which actually determines IQ. Only one causal path enters into the index (that from common environment), and no causal path emanates from it. Suppose that some of the socioeconomic variables (parental education for example) have a direct effect on the intellectual development of children. Or consider the fact that since socioeconomic status is itself a phenotype, it may well have a genetic component correlated with the genetic component of intelligence. If so, the Honolulu IQ models will be clearly misspecified.

ACKNOWLEDGEMENTS

I am indebted to D. C. Rao and Newton Morton for their care in responding to my many inquiries about their work, to Carol Treanor and S. Mostafa Baladi for computational assistance, and especially to Leon Kamin who provided me with his unpublished analyses of the American IQ samples. My research has been facilitated also by the opportunity to draw on the expertise of Luca Cavalli-Sforza, Dudley Duncan, Marc Feldman, Walter Nance, and Sandra Scarr. Financial support was provided by the Graduate School Research Committee at the University of Wisconsin, the Center for Advanced Study in the Behavioral Sciences, and the National Science Foundation. But none of those persons and institutions should be held responsible for the contents of this paper.

REFERENCES

Burks, B. S. 1928. The relative influence of nature and nurture upon mental development: a comparative study of foster parent - foster child resemblance and true parent - true child resemblance. Twenty-Seventh Yearbook of the National Society for the Study of Education, Part I. Public School Publishing, Bloomington: 219-316.

Eaves, L. J. 1975. Testing models for variation in intelligence. Heredity, 34: 132-136.

Goldberger, A. S. 1976(a). Mysteries of the meritocracy. In The IQ Controversy: Critical Readings (N. J. Block & G. Dworkin, editors). Pantheon, New York: 265-279.

Goldberger, A. S. 1976(b). Jensen on Burks. *Educational Psychologist*, *12*: 64-78.

Goldberger, A. S. 1977. Models and methods in the IQ debate, Part I. Workshop Paper 7710, Social Systems Research Institute, University of Wisconsin, Madison.

Jencks, C. 1972. *Inequality: A Reassessment of the Effect of Family and Schooling in America*. Basic Books, New York.

Jinks, J. L. & Eaves, L. J. 1974. IQ and inequality. *Nature*, *248:* 287-289.

Lawley, D. 1943. A note on Karl Pearson's selection formulae. *Proceedings of the Royal Society of Edinburgh*, *Section A*, *62*: 28-30.

Loehlin, J. C., Lindzey, G., & Spuhler, J. 1975. *Race Differences in Intelligence*. W. H. Freeman, San Francisco.

Morton, N. E. 1972. Human behavioral genetics. In *Genetics*, *Environment*, *and Behavior* (I. Ehrman, G. S. Omenn, & E. Caspari, editors). Academic Press, New York: 247-271.

Morton, N. E. 1974. Analysis of family resemblance. I. Introduction. *American Journal of Human Genetics*, *26*: 318-330.

Morton, N. E. & Rao, D. C. 1977. Quantitative inheritance in man. Population Genetics Laboratory, University of Hawaii, Honolulu.

Newman, H. H., Freeman, F. N., & Holzinger, K. J. 1937. *Twins: A Study of Heredity and Environment*. University of Chicago Press, Chicago.

Nichols, P. L. 1970. The effects of heredity and environment on intelligence test performance in 4 and 7 year white and Negro sibling pairs. Doctoral dissertation, University of Minnesota, Minneapolis.

Pearson, K. 1903. Mathematical contributions to the theory of evolution - XI. On the influence of natural selection on the variability and correlation of organs. *Philosophical Transactions of the Royal Society of London*, *200*: 1-66.

Rao, D. C., Maclean, C. J., Morton, N. E., & Yee, S. 1975. Analysis of family resemblance. V. Height and weight in Northeastern Brazil. *American Journal of Human Genetics*, *27*: 509-520.

Rao, D. C. & Morton, N. E. 1977. IQ as a paradigm in genetic epidemiology. Population Genetics Laboratory, University of Hawaii, Honolulu.

Rao, D. C., Morton, N. E., Elston, R. C., & Yee, S. 1977. Causal analysis of academic performance. *Behavior Genetics*, *7*: 147-159.

Rao, D. C., Morton, N. E., & Yee, S. 1974. Analysis of family resemblance. II. A linear model for familial correlation. *American Journal of Human Genetics*, *26*: 331-359.

Rao, D. C., Morton, N. E., & Yee, S. 1976. Resolution of cultural and biological inheritance by path analysis. American Journal of Human Genetics, 28: 228-242.

Rao, D. C., Morton, N. E., & Yee, S. 1977. Resolution of cultural and biological inheritance by path analysis: Corrigenda and reply to Dr. Goldberger. Population Genetics Laboratory, University of Hawaii, Honolulu.

Scarr, S. 1977. Genetic effects on human behavior: recent family studies. Yale University, New Haven.

Scarr, S. & Weinberg, R. A. 1977. Intellectual similarities within families of both adopted and biological children. University of Minnesota, Minneapolis.