Social Allocation Models of Intelligence: A Methodological Inquiry

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The authors consider Professor Jensen's hypothesis that inherited factors may be "implicated" in observed racial differences in measured intelligence. They argue that even if one chooses to accept Professor Jensen's estimates of the proportion of variance in intelligence accounted for by heredity, environment, and their interaction, his hypothesis is not substantiated by his own data. They go on to say that the parameter estimates are highly suspect, given the small sample size of the twin studies and the way disparate studies were combined. The authors simulated, on a computer, the process of studying twins and found that the statistical procedures employed in these studies of intelligence yield quite unstable estimates. In particular, the estimate of the interaction effect is quite unreliable, both because of sample size, and because Jensen chose a statistical model which would attribute some interaction to the main variables—heredity and environment. Finally, the authors propose that the studies of intelligence reported by Professor Jensen ignore the reality of feedback loops, initiated by physical differences, and enhanced by processes of social differentiation in our society.

Reaction to the recent article by Arthur R. Jensen[1] has been both harsh and predictable. However, much of it has been political, and therefore convincing or unconvincing, depending upon one's politics. Jensen's position is that it is "... a

[1] Arthur R. Jensen, "How Much Can We Boost IQ and Scholastic Achievement?," *Harvard Educational Review*, XXXIX (Winter, 1969), 1-123.

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not unreasonable hypothesis that genetic factors are strongly implicated in the average Negro-white intelligence difference" (p. 82). We believe his article contains serious technical errors, both of commission and omission, and we therefore present several statistical arguments which substantially weaken the plausibility of Jensen's hypothesis. We argue that even a tentative acceptance of this hypothesis is not justified by a careful analysis of available data.

Three strategies are available for developing a case against Jensen's postulated relation between race and inherited intelligence.

1. Accept Professor Jensen's model, and his estimates of the model parameters, but show that non-genetic disparities would still account for most or all of the 15 point IQ difference between the races. This explanation does not implicate genetic racial differences and yet is consistent with his estimates of the model parameters.
2. Accept Professor Jensen's mathematical model as a satisfactory descriptive construct, but question his parameter estimates as being either unreliable or incorrect.
3. Reject Professor Jensen's model as inappropriate because of its failure to incorporate relationships which clearly exist among variables in the real world as we know it.

In our analysis, we will adopt each of these three strategies, and demonstrate that they all lead to inferences in direct contradiction to Jensen's suggestion that genetic differences are implicated in observed black-white IQ differences.

Four Major Results of Our Analysis:

First, we accept Professor Jensen's estimate of the proportion of IQ variability explained by hereditary versus environmental factors. Using his proportions, with no interaction effects, we show that more than half of the 15 point difference in mean IQ between races is explained by the differential allocation of the races to social conditions. Second, we demonstrate that if the interaction between genetic and environmental effects accounts for only one percent of the total IQ variance, then a 15 point difference between means occurs even if the racial genetic distributions were identical. Since Jensen's data suggest that the proportion of interaction variance is one percent, this analysis is entirely consistent with his data. Third, we present the results of computer simulations which establish the instability of Jensen's estimation procedures, thus throwing doubt on

the reliability of his parameter estimates. We conclude that if the usual statistical criteria for significance are used, Jensen's data are not inconsistent with the presence of a moderate amount of genetic-environmental interaction. Fourth, we show that if moderate amounts of interaction exist, mean IQs of blacks and whites could easily differ by more than 15 points, despite identical genetic distributions in the two races. These are our statistical arguments, all of which assume that Jensen's model and his estimation procedures are conceptually tenable. Later in the paper, however, we question the quality and the consistency of Jensen's data. Finally, we conclude by suggesting that current genetic-environment models of intelligence ignore important social processes and must be revised.

Jensen's Estimates:

Jensen uses a components of variance model to estimate the proportion of total IQ variability attributable to genetic factors, environmental factors, and their interaction.[2] It is crucial to note, however, that Jensen estimated the three components in a very particular way.[3] From studies of monozygotic twins (MZ), he derives an estimate of the genetic proportion as .75. Then, from studies of un-

[2] The original components of variance model of the type Jensen uses was proposed by C. Eisenhart, "The Assumptions Underlying Analysis of Variance," *Biometrics*, 3, 1947, 1-21. This model is now called one of two names by statisticians; a Model II random effects model, or a Graybill Model 5 Components of Variance. An excellent and very readable treatment of this model appears in W. L. Hays, *Statistics for Psychologists* (New York: Holt, Rinehart & Winston). A detailed treatment of the statistical assumptions underlying this model is available also in S. L. Crump, "The Present Status of Variance Components Analysis," *Biometrics*, 7, 1951, 1-16. The intraclass correlation coefficients used to estimate the various proportions of variance differ from the well known product moment correlation coefficients, although Jensen never makes this distinction directly. The intraclass correlation was developed by Fisher, and clearly described in his classic, R. A. Fisher, *The Design of Experiments* (London: Oliver-Boyd, 1935).

[3] Jensen, in his article, reports intraclass correlations taken from MZ twin and foster pair studies. He nowhere indicates that these correlations depend upon and are derived from the special assumptions of a random effects analysis of variance (ANOVA). This Model II ANOVA requires that the main effects have been sampled at random from the underlying dimension, which is the population of genotypes for the MZ studies, and the relevant array of possible environments for the foster pair studies. Further it requires that the 'other' dimension in each case has been allocated at random within, as well as between, pairs. We actually would want to estimate a two-way ANOVA, with both genetics and environment separately replicated but crossed randomly—but no such design is possible. Instead, two one-way ANOVAs are used to estimate the main effects, and the interaction variance that would have been found directly in a two-way study is estimated by subtraction. Jensen essentially uses three parameters as additive components for this model: V_G (genetic variance), V_E (environmental variance), and V_I (interaction variance). The estimates are obtained from the pair studies, and thus based upon single replication within groups. These components sum to the total phenotypic variance in the population, V_P. Further, for the MZ twin studies, the Model II ANOVA produces a direct link between the intraclass correlation coefficient *r*, the heritability coefficient H, and the ratio of V_G to V_P. The link is that $H = r = V_G/V_P$.

related foster children raised together, he estimates the environment proportion as .24. This leaves .01 for the third component, the genetic-environment interaction.

We should point out that an estimate of a percent of variance does *not* translate directly into any explanation of a difference between racial mean IQs. Otherwise, the analysis would end right here, and we could conclude that 75 percent of the 15 point gap is due to genetic factors. Such reasoning is simply incorrect.

Social Allocation Models:

We call our explanation of the racial differences in mean IQ a social allocation model. By social allocation we mean a process whereby members of different racial groups are assigned to environments non-randomly. This model differs from the classic environmentalist position because it grants that individual differences in IQ (although not racial differences) are largely genetic. With respect to the racial differences, the 25 percent non-genetic component accounts for all the observed difference. We differ from the interactionists in believing that the majority of the variation in intelligence can be separated into additive genetic and environmental components. We have found that the presence of a tiny interaction component can explain large differences between group means.

Other critics of Professor Jensen's analysis have already pointed out that given the structure of today's society, blacks and whites are exposed, or 'socially allocated,' to essentially different environments. For example, in his chapter in the excellent book edited by Deutsch, Katz, and Jensen, I. I. Gottesman points out that "In the light of what has been said in the introduction to this book and the literature on the effects of stimulation in early infancy (Caster, 1961; Riesen, 1961; Thompson and Schaefer, 1961), there is every reason to doubt that a typical Negro infant is reared in a typical white infant's natural habitat."[4]

At several points in his analysis, however (pp. 74-78, 83, 87), Jensen seems to accept one or another of the current indexes of socio-economic status as being a comparable measure of the social condition of blacks and whites. Nearly all such indexes are weightings of education (in number of years completed), income, and occupation (on one or another rating of prestige). These are obviously important dimensions within the social life of our nation, and the two races differ

[4] I. I. Gottesman, "Biogenetics of Race and Class," in *Social Class, Race, and Psychological Development,* ed. by M. Deutsch, I. Katz, and A. R. Jensen (New York: Holt, Rinehart & Winston, 1968).

considerably in their placement on these three dimensions.[5] But the crucial point is that our SES measures do not work identically for both races. Black and white families with identical incomes do not have the same economic options open to them—housing discrimination is only the most obvious reason for assuming that a dollar is worth less to a black family than to a white one. Further, equal numbers of years of education do not imply equal educations; nor do they imply equal access to further benefits, such as jobs or income levels.

That black engineers, college presidents, physicians, and teachers all receive less than similarly employed whites is well known; however, even black postal clerks who are high school graduates earn on the average $250 less per year than their white peers.[6] Other racially related inequities, such as in promotion possibilities, nepotistic privileges, community prestige, are all also lost within our conventional indexes. Equal standings on our measures of social status do not imply equal statuses—at least not for men of different races.

We thus cannot draw conclusions from differences in IQ among blacks and whites "of equal SES" simply because equal SES scores still imply more restricted life-chances for blacks. Jensen, in his Table 3 (page 83), gives data on the prevalence of children with IQs below 75 for each race, holding measured SES constant. He is incorrect in assuming that the actual environments are identical.[7]

On the other hand, an SES index does provide a rough suggestion of the existing range of variation in social conditions. Jensen used SES groupings as a measure of *equality* between whites and blacks—we will use them as a measure of the *disparity* between the races. Since SES measures understate that disparity, we are making an inherently conservative estimate of environmental effects.

We promised to use both Jensen's model and his parameter estimates to illustrate our social allocation model; we now present this investigation and show that non-genetic differences can explain the observed differences in mean IQ.

[5] Whether they are also the relevant dimensions upon which to measure environments with regard to their impacts upon IQ is uncertain.

[6] U. S. Census, 1960, *Subject Reports, Occupation by Earnings and Education,* Report PC(2)-7B, U. S. G.P.O., Washington, D. C., pp. 102, 103.

[7] The SES scale does not even behave similarly for the two races in its gross properties—always a strong signal that we are not measuring the same thing in each population. The U. S. Census SES scale was built from three components (education, income, and occupation, as usual); persons were registered as being "consistent" in SES if they had scores on each of the three underlying 100 point scales which all fell within 20 points of each other. The median overall SES for all whites was 55.2, while the median SES for consistent whites was 11.8 points *higher* at 67.0. For blacks, the median SES was 28.5, but the consistent blacks had scores 15.9 points *lower,* at 12.6. Note that this cannot be a floor-and-ceiling effect, since the displacement is in the wrong direction in both cases.

Exploring Racial Differences via Two-way Tables:

We will use the well-known format of the two-way layout to examine socially allocated racial differences in IQ.

Let us design a 12 by 10 table: twelve rows and ten columns. Suppose we let the rows represent some measure of environment, which is divided into twelve types, and the columns represent some measure of genetic endowment, which will be divided into ten types. We then have a table where each of the 120 cells represents a particular genetic-environment combination. Further, we may assign two numbers to each cell; one number corresponding to the mean IQ of all persons falling into that cell, and the second number giving the proportion of people from some population falling into the cell. If we have all these data available to us, then we may compute the mean IQ for any population by simply computing an overall weighted mean from the 120 cell means, where each cell mean is weighted by the proportion of people in the population falling into that cell.

If we are going to use this approach to compare black-white IQ differences, then the first order of business is to develop separate tables for each race. However, we will argue that blacks and whites have no differences in genetic endowments, which requires that the tables for each race have an identical genetic distribution over the ten columns. Therefore, we may begin by assigning a column mean to each of the ten columns.

Our choice of the number of columns to use was fairly arbitrary, but our assignment of column means is not at all arbitrary. We will begin by accepting Jensen's estimate that the genetic component of variance is 75 percent of the total phenotypic IQ variance. This then requires, if we use an IQ total variance of 225 as a reasonable estimate for both populations combined, that the variance over the ten genetic categories be 75 percent of 225, or 168.75. Accepting also Jensen's contention that genetic endowments are approximately normally distributed, we may then divide the population of each race into tenths, so that each of the ten genetic categories contains one tenth of the population. To determine the column means, finally, we simply find the mean value of each tenth of a normal distribution which has an overall mean of 100.00 and a variance of 168.75. These means are given in Table A, which gives not only the column means when the genetic proportion of phenotypic variance is .75, but also the means when this proportion is .70 and .80, in case the reader wishes to try alternative partitions of variance.

The determination of the twelve row means is more complicated, because although we posited that whites and blacks are identically distributed over the

TABLE A

Percentage Distributions by Race, and Mean IQ Scores, for the Genetic Categories. Means are given for the Cases when the Proportions of the Total IQ Variance (225) accounted for by Genetic Effects are .70, .75, and .80.

Genetic Category:		*Percent of Population in Each Category**		*Mean of Each Category if the Proportion of Variance is:*		
		Whites:	*Blacks:*	*.70*	*.75*	*.80*
1.	(low)	10.0	10.0	78.01	77.02	76.49
2.		10.0	10.0	86.88	86.30	86.04
3.		10.0	10.0	91.52	91.09	90.88
4.		10.0	10.0	95.11	94.85	94.82
5.		10.0	10.0	98.41	98.27	98.20
6.		10.0	10.0	101.59	101.73	101.80
7.		10.0	10.0	104.89	105.15	105.18
8.		10.0	10.0	108.48	108.91	109.12
9.		10.0	10.0	113.12	113.70	113.96
10.	(high)	10.0	10.0	121.99	122.98	123.51

* The two races are assumed to have identical genetic distributions, with one tenth of each race falling into each of the ten genetic categories. A larger number of genetic categories (the choice of ten was arbitrary) *increases* the effects reported in this paper, but only slightly.

ten columns (genetic effects), the two races are clearly distributed differently over the twelve rows, representing environment effects.

First, we must decide how to measure relative environments for blacks and whites. Assume that the SES of the families of white and black children under 14 is a rough indicator of the environments in which the children were raised. Since the estimate of the environment's contribution to IQ was derived from studies of white children, the white proportions in each of the 12 SES groupings provided by the U.S. Census will be used to estimate the mean values for these categories.[8] Then we will ask what the effect of the environmental dimension would be upon a group allocated as the black children are. Because SES understates the disparity between blacks and whites, this procedure *underestimates* the impact of environmental deprivation upon black children.

We will use an estimate of the proportion of phenotypic variance in IQ due to environment of 25 percent. (Thus, we are allowing exactly zero interaction between genetic effects and environmental effects.) 25 percent of a total IQ variance

[8] U. S. Census, 1960, *Subject Reports, Socioeconomic Status*, Report PC(2)—56, U. S. G. P. O., Washington, D. C. p. 50.

of 225 yields an environment component variance of 56.25, which means that our overall environment axis will be represented by a normal distribution with overall mean 100.00 and variance 56.25.

Some readers may find it less appealing to assume a normal distribution for environments than for genotypes. Our perception of how the real world works suggests that there are very disabling environments to which far more children are subjected than would occur within a normal distribution—even among the white population, but especially among the blacks. It can also be argued that prejudice afflicts most black children in a way no white child experiences. If either of the above intuitions is correct, our assumption understates the deprivation of the black population, and thus the case against Professor Jensen's position is stronger than we present. Readers who are unpersuaded by our model for environments should be equally unpersuaded by Professor Jensen's proportions of variance and his conclusions about racial differences.

Since blacks and whites are not identically distributed over the twelve environment categories, we will not break this new normal distribution into equal

TABLE B

Percentage Distributions by Race, and Mean IQ Scores, for Environmental (SES) Categories. Means are given for the Cases when the Proportions of the Total IQ Variance (225) accounted for by Environment are .15, .20, and .25.

Environment Category:		*Percent of Population in Each Category**		*Mean of Each Category if the Proportion of Variance is:*		
		Whites:	*Blacks:*	*.15*	*.20*	*.25*
1.	(low)	2.0	15.8	86.11	83.80	81.96
2.		4.4	20.2	89.89	88.29	86.96
3.		3.2	10.0	91.86	90.61	89.46
4.		4.1	10.1	93.09	92.02	91.06
5.		10.9	16.9	94.93	94.09	93.42
6.		14.3	12.0	97.23	96.79	96.39
7.		16.3	7.3	99.56	99.49	99.42
8.		15.6	4.0	101.94	102.25	102.51
9.		7.1	1.3	103.81	104.40	104.62
10.		5.9	.9	105.07	105.86	106.51
11.		9.3	1.1	107.02	108.16	109.02
12.	(high)	6.8	.5	111.26	112.93	114.30

* From p. 50, U. S. Census of Population, 1960, *Subject Reports: Socioeconomic Status.* Final Report PC(2)—56. U. S. Government Printing Office, Washington, D. C.

twelfths. Rather, we will use the proportion of whites in each of the twelve categories to determine what proportion of the environment distribution should be allocated to each category, and we thus find the mean for each of the twelve rows. These means, as well as the proportion of whites in each category, are presented in Table B, which also gives similarly derived means for an environmental variance component of 15 percent and 20 percent, to be used a bit later.

We thus have found twelve row means and ten column means; the additive ANOVA model, with no interaction effects present, then enables us to find a mean value for each of the 120 cells in our original table. These cell means are given in Table 1A.

TABLE 1A

Mean IQ Score for each Combination of the Ten Genetic and Twelve Environmental Categories, when 75 percent of the Total Variance is Genetic, 25 percent is Environmental, and There are no Interaction Effects Present.

Environment (SES) Category (Rows)	*Genetic Category (Columns)*										
	1	*2*	*3*	*4*	*5*	*6*	*7*	*8*	*9*	*10*	*Row Mean*
1	58.98	68.26	73.05	76.81	80.23	83.52	86.90	90.69	95.42	104.71	81.96
2	63.98	73.26	78.05	81.81	85.23	88.52	91.90	95.69	100.42	109.71	86.96
3	66.48	75.76	80.55	84.31	87.73	91.02	94.40	98.19	102.92	112.21	89.46
4	68.08	77.36	82.15	85.91	89.33	92.62	96.00	99.79	104.52	113.81	91.06
5	70.44	79.72	84.51	88.27	91.69	94.98	98.36	102.15	106.88	116.17	93.42
6	73.41	82.69	87.48	91.24	94.66	97.95	101.33	105.12	109.85	119.14	96.39
7	76.44	85.72	90.51	94.27	97.69	100.98	104.36	108.15	112.88	122.17	99.42
8	79.53	88.81	93.60	97.36	100.78	104.07	107.45	111.24	115.97	125.26	102.51
9	81.64	90.42	95.71	99.47	102.89	106.18	109.56	113.35	118.08	127.37	104.62
10	83.53	92.81	97.60	101.36	104.78	108.07	111.45	115.24	119.97	129.26	106.51
11	86.04	95.32	100.11	103.87	107.29	110.58	113.96	117.75	122.48	131.77	109.02
12	91.32	100.60	105.39	109.15	112.57	115.86	119.24	123.03	127.76	137.05	114.30
Column Mean	77.02	86.30	91.09	94.85	98.27	101.73	105.15	108.91	113.70	122.98	

When the cell mean IQ scores in this table are multiplied by the proportions taken from the corresponding cell in Table 1B, and the products are summed, the result is the average IQ for the white population. That average IQ equals 100. When the same procedure is followed using the proportions for the black population, taken from Table 1C, the result is an average IQ for black children of 91.26—in spite of the fact that both races have identical genetic distribution.

TABLE 1B

Percent of the White Population Allocated to Each Combination of the Ten Genetic and Twelve Environmental Categories.

Environment (SES) Category (Rows)	*Genetic Category (Columns)*										
	1	*2*	*3*	*4*	*5*	*6*	*7*	*8*	*9*	*10*	*Row %*
1	.20	.20	.20	.20	.20	.20	.20	.20	.20	.20	2.0
2	.44	.44	.44	.44	.44	.44	.44	.44	.44	.44	4.4
3	.32	.32	.32	.32	.32	.32	.32	.32	.32	.32	3.2
4	.41	.41	.41	.41	.41	.41	.41	.41	.41	.41	4.1
5	1.09	1.09	1.09	1.09	1.09	1.09	1.09	1.09	1.09	1.09	10.9
6	1.43	1.43	1.43	1.43	1.43	1.43	1.43	1.43	1.43	1.43	14.3
7	1.63	1.63	1.63	1.63	1.63	1.63	1.63	1.63	1.63	1.63	16.3
8	1.56	1.56	1.56	1.56	1.56	1.56	1.56	1.56	1.56	1.56	15.6
9	.71	.71	.71	.71	.71	.71	.71	.71	.71	.71	7.1
10	.59	.59	.59	.59	.59	.59	.59	.59	.59	.59	5.9
11	.93	.93	.93	.93	.93	.93	.93	.93	.93	.93	9.3
12	.68	.68	.68	.68	.68	.68	.68	.68	.68	.68	6.8
Column %	10.0	10.0	10.0	10.0	10.0	10.0	10.0	10.0	10.0	10.0	100.0

The within-cell entries which appear in the table are those assumed by the twin studies. In fact, as long as the row and column constraints are met, any other set of cell entries would result in the same overall mean IQ.

Now, finally, we are ready to compare the overall black and white mean IQs. Table 1B shows how whites are allocated to each of the 120 cells. Notice that this allocation table has ten percent of all whites falling into each of the ten genetic categories. Combining the table of cell means with the white allocations, we find that white children under 14 have an average IQ of 100.00, as Jensen reports.[9]

Turning to the table of black allocations (Table 1C), we find that the row totals are different from those for whites. These black row totals correspond to the proportion of blacks falling into the same twelve environment categories as defined by the U.S. Census Bureau. As we all could have guessed, the blacks cluster more towards the low socio-economic categories than do the whites. But the blacks are also assigned with no interaction or covariance, and ten percent of all black children under 14 are assigned to each of the ten genetic categories.

[9] Of course, the overall mean IQ could have been computed from only the row marginals, with each row mean weighted by the proportion of whites assigned to that row. But this is only the case when the table of cell means has no interaction effects present.

TABLE 1C

Percent of the Black Population Allocated to Each Combination of the Ten Genetic and Twelve Environmental Categories.

Environment (SES) Category (Rows)	*Genetic Category (Columns)*										
	1	*2*	*3*	*4*	*5*	*6*	*7*	*8*	*9*	*10*	*Row %*
1	1.58	1.58	1.58	1.58	1.58	1.58	1.58	1.58	1.58	1.58	15.8
2	2.02	2.02	2.02	2.02	2.02	2.02	2.02	2.02	2.02	2.02	20.2
3	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	10.0
4	1.01	1.01	1.01	1.01	1.01	1.01	1.01	1.01	1.01	1.01	10.1
5	1.69	1.69	1.69	1.69	1.69	1.69	1.69	1.69	1.69	1.69	16.9
6	1.20	1.20	1.20	1.20	1.20	1.20	1.20	1.20	1.20	1.20	12.0
7	.73	.73	.73	.73	.73	.73	.73	.73	.73	.73	7.3
8	.40	.40	.40	.40	.40	.40	.40	.40	.40	.40	4.0
9	.13	.13	.13	.13	.13	.13	.13	.13	.13	.13	1.3
10	.09	.09	.09	.09	.09	.09	.09	.09	.09	.09	.9
11	.11	.11	.11	.11	.11	.11	.11	.11	.11	.11	1.1
12	.05	.05	.05	.05	.05	.05	.05	.05	.05	.05	.5
Column %	10.0	10.0	10.0	10.0	10.0	10.0	10.0	10.0	10.0	10.0	100.0

The within-cell entries which appear in the table are those assumed by the twin studies. In fact, as long as the row and column constraints are met, any other set of cell entries would result in the same overall mean IQ.

Combining the cell mean table with the black allocation table, the overall black mean IQ computes to 91.26. Thus, we see that if Jensen's essential breakdown of .75 and .25 for the G and E proportions of effects on IQ is used, and if U.S. Census data on the differential social allocation of the two races to socio-economic categories are used, *more than half of the mean difference in IQs between races is explained, although the races have identical genetic distributions.*

Let us pause for a moment and see what lowered the mean IQ of the black children. Within each racial group separately, most of the variation among children is fundamentally genetic: this follows from the way in which the marginal means were computed, but can be most easily observed by noticing that more than half of both groups falls into 4 of the 12 rows in their tables. In other words, most of the children have environments quite similar to others of their race. But the black children all cluster toward the lower SES levels while the white children cluster

toward the middle. Even though three quarters of the variance within each race is genetic, the variance between the races is wholly environmental.

We therefore suggest that whites have a distribution over environment types which has a relatively small variance, and blacks similarly have an environmental distribution with small variance. However, the variance *between* the white and black allocations in large. Thus, with only non-genetic differences of 25 percent to separate the races, an expected difference in mean IQs of nearly 9 points is found.

Introducing Interaction:

Our analysis thus far has taken the most favorable case for Professor Jensen's point of view, by using the proportion of .75 for genetic effects, and attributing

TABLE 2A

Interaction Effects on Each Combination of the Ten Genetic and Twelve Environmental Categories, when 1 percent of the Total Variance in IQ is due to Interaction.

Environment (SES) Category (Rows)	*Genetic Category (Columns)*										
	1	*2*	*3*	*4*	*5*	*6*	*7*	*8*	*9*	*10*	*Row Sum*
1	—7.34	—4.24	1.97	1.51	1.30	1.45	2.18	1.45	.99	.72	0.0
2	2.26	—1.64	—9.43	—2.89	1.90	2.05	2.78	2.05	1.59	1.32	0.0
3	.59	.61	.65	—1.64	—2.01	.22	.70	.22	.26	.40	0.0
4	.59	.61	.65	.36	—2.01	—1.78	.70	.22	.26	.40	0.0
5	2.36	2.46	2.67	2.21	2.00	—1.85	—11.12	—1.85	1.69	1.42	0.0
6	.59	.61	.65	.36	—.01	.22	.70	—1.78	—1.74	.40	0.0
7	.16	.26	.47	.01	—.20	—.05	.68	—.05	—.51	—.78	0.0
8	.16	.26	.47	.01	—.20	—.05	.68	—.05	—.51	—.78	0.0
9	.16	.26	.47	.01	—.20	—.05	.68	—.05	—.51	—.78	0.0
10	.16	.26	.47	.01	—.20	—.05	.68	—.05	—.51	—.78	0.0
11	.16	.26	.47	.01	—.20	—.05	.68	—.05	—.51	—.78	0.0
12	.16	.26	.47	.01	—.20	—.05	.68	—.05	—.51	—.78	0.0
Column Sum	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	

The interaction values shown in this table are arbitrary, in that there are an infinite number of such tables, each displaying about 1 percent of the total variance in IQ.

This table was selected to illustrate the consequences of relatively small amounts of interaction, and has no direct empirical basis. Rows and columns may not add to zero because of rounding. Table 3A was similarly constructed, but has an interaction proportion of 10 percent.

the entire remaining .25 to environment effects. Jensen's actual breakdown of proportions was .75 and .24 for genetic and environment effects respectively, with the .01 remainder allowed for interaction. We now examine the possible influence of this tiny interaction component on the overall racial mean IQs.

A .01 interaction effect permits us to introduce an interaction variance of 2.25 into our two-way layout, while dropping the environment variance to 24 percent of 225, or 54.0. Thus, we may modify the cell mean IQs in each of the 120 cells, so long as we satisfy our variance constraint. Our modifications for this case appear in Table 2A. This table of interaction effects gives the particular effects we chose to inject; there are an infinite number of possible interaction effect tables which would satisfy the variance constraints. We note, however, that our particular table satisfies Jensen's parameter estimates, which was our primary concern.

Table 2B gives the adjusted cell mean IQ values, after the interaction effects were included. Finally, Table 2C of black allocations illustrates how our social

TABLE 2B

Mean IQ Score for Each Combination of the Ten Genetic and Twelve Environmental Categories, when 75 percent of the Total Variance is Genetic, 24 percent is Environmental, and 1 percent is due to the Presence of Interaction Effects.

Environment (SES) Category (Rows)	*Genetic Category (Columns)*									
	1	*2*	*3*	*4*	*5*	*6*	*7*	*8*	*9*	*10*
1	51.64	64.02	75.02	78.32	81.53	84.97	89.08	92.14	96.41	105.43
2	66.24	71.62	68.62	78.92	87.13	90.57	94.68	97.74	102.01	111.03
3	67.07	76.37	81.20	82.67	85.72	91.24	95.10	98.41	103.18	112.61
4	68.07	77.97	82.80	86.27	87.32	90.84	96.70	100.01	104.78	114.21
5	72.80	82.18	87.18	90.48	93.69	93.13	87.24	100.30	108.57	117.59
6	74.00	83.30	88.13	91.60	94.65	98.17	102.03	103.34	108.11	119.54
7	76.60	85.98	90.98	94.28	97.49	100.93	105.04	108.10	112.37	121.39
8	79.69	89.07	94.07	97.37	100.58	104.02	108.13	111.19	115.46	124.48
9	81.80	91.18	96.18	99.48	102.69	106.13	110.24	113.30	117.57	126.59
10	83.69	93.07	98.07	101.37	104.58	108.02	112.13	115.19	119.46	128.48
11	86.20	95.58	100.58	103.88	107.09	110.53	114.64	117.70	121.97	130.99
12	91.48	100.86	105.86	109.16	112.37	115.81	119.92	122.98	127.25	136.27

When the cell means in this table are weighted by the proportions shown for the black population (in Table 2C) the average IQ is reduced to 86.81. Weighted by the white distribution from Table 1B, the result remains 100.0. Since the whites and blacks are shown as genetically identical, the disparity is due only to the generally disadvantageous and specifically malicious allocation of environments to the black children.

TABLE 2C

Malicious Allocation of Blacks to Genetic-Environmental Combinations which have Negative Interaction Effects Present. The Black Population has the Same Genetic Distribution as the White Population.

Environment (SES) Category (Rows)	*Genetic Category (Columns)*										
	1	*2*	*3*	*4*	*5*	*6*	*7*	*8*	*9*	*10*	*Row %*
1	8.60	4.72	.18	.18	.18	.18	.18	.18	.18	1.12	15.8
2	.18	4.00	8.57	5.64	.18	.18	.18	.18	.18	.88	20.2
3	.17	.17	.17	3.13	5.16	.17	.17	.17	.17	.54	10.0
4	.17	.17	.17	.17	3.50	5.15	.17	.17	.17	.27	10.1
5	.18	.18	.18	.18	.18	3.66	8.61	2.86	.18	.68	16.9
6	.17	.17	.17	.17	.17	.17	.17	5.87	4.45	.47	12.0
7	.16	.16	.16	.16	.16	.16	.16	.16	4.08	1.96	7.3
8	.13	.13	.13	.13	.13	.13	.13	.13	.13	2.80	4.0
9	.08	.08	.08	.08	.08	.08	.08	.08	.08	.59	1.3
10	.06	.06	.06	.06	.06	.06	.06	.06	.06	.34	.9
11	.07	.07	.07	.07	.07	.07	.07	.07	.07	.46	1.1
12	.04	.04	.04	.04	.04	.04	.04	.04	.04	.14	.5
Column %	10.0	10.0	10.0	10.0	10.0	10.0	10.0	10.0	10.0	10.0	100.0

allocation model works. The proportion of blacks in each of the twelve rows is unchanged from before; however, within rows, the blacks are more heavily weighted in those columns which also have negative interaction effects. Thus, examining the black allocation table, we find that our social allocation of blacks has been 'malicious,' in that the standing of any black in his genetic category is likely to be higher than his standing on environment.

If we weight the cell IQ means in Table 2B by the black allocations to genetic-environment combinations, we find that although the white mean IQ is unchanged at 100.00, the black mean has now dropped to 86.81. Once again, we recall that we have both kept the genetic distributions of blacks and whites identical and used the exact parameter estimates obtained by Jensen. Thus, we see that the observed difference in IQ between the races is essentially entirely accounted for by our social allocation approach, with no genetic differences being present.

As a final analysis of this type, we investigated what would happen to the mean black IQ if we kept the genetic component at .75, while reallocating the remainder so that the environment variance component was .15, and the interaction

proportion .10. The results of this analysis appear in Table 3. Keeping the allocation of blacks to cells identical to the allocation of Table 2, the black mean drops to 82.59. We may therefore conclude that with an interaction component of variance somewhere between .01 and .10, the black mean IQ may be expected to be approximately 85, even though blacks are distributed identically with whites over the genetic categories.

Summing Up:

Our models show how a large difference between black and white mean IQs may be explained not by the hypothesis of genetic differences between races, but rather by the non-genetic differences in allocation of blacks and whites to different environments, and environment-genetic combinations. In terms of our 12 by 10 tables, Jensen has argued that the difference in mean IQs may be attributable to a differential assignment of whites and blacks to the *columns* of the table, with blacks clustered relatively more to the leftward columns than are whites. We believe, rather, that the differential allocation occurs on the *rows,* with blacks more frequently assigned to the upper rows than are whites. By combining our argument with the malicious allocation effects on blacks of interaction, the IQ differences are readily explainable without any resort whatever to the hypothesis of genetic differences between races.

The Parameter Estimates:

So far we have assumed that Professor Jensen's model and his parameter estimates are approximately correct. We now turn our attention to examining the validity of Jensen's particular estimates of P_G (the proportion of variance in measured IQ due to genetic factors), P_E (the proportion of variance due to environment), and P_I (the proportion of variance from interaction).

It is important to question the exact values of these parameters because in a society which allocates whites and blacks with similar genetic endowments differentially to environments, a small amount of interaction, P_I, can go a long way toward allocating people with certain genetic endowments to a favorable environment or an unfavorable environment for those particular endowments. And if a society chooses to take advantage of the genetic-environment interaction by allocating persons of one race in a positive way, while allocating persons of another race in a negative way, then that society can easily build in differences in measured intelligence where there were none at birth. Note we are not arguing

that no individual differences in intelligence exist. We agree with Jensen that these differences are present. Where we disagree with Jensen is with his specific argument suggesting the presence of *racial* differences in genetic endowments. It is here that our interaction argument becomes important, and because of its importance, we digress for a moment to consider its implications.

Implications of Interaction:

We pointed out at the beginning of this article that Jensen's components of variance approach to estimating intelligence uses a Model II ANOVA. Statistical interaction, as imbedded in the Model II ANOVA, relates to a gain or loss in intelligence due to a particular combination of E and G placements. This gain or loss is not predictable from both E and G considered separately in an additive sense. The Model II ANOVA, however, assumes that the cell values, weighted by the proportion of the population in each cell, sum to zero across both variables. Actually, the Model II not only assumes the weighted terms sum to zero; *it insures that they do by attributing any residual to the main effects of environment and*

TABLE 3A

Interaction Effects on Each Combination of the Ten Genetic and Twelve Environmental Categories, when 10 percent of the Total Variance in IQ is due to Interaction.

Environment (SES) Category (Rows)	*Genetic Category (Columns)*										
	1	*2*	*3*	*4*	*5*	*6*	*7*	*8*	*9*	*10*	
1	—23.66	—11.03	4.34	4.34	4.34	4.34	4.34	4.34	4.34	4.34	0.0
2	4.46	—3.96	—19.90	—1.72	4.10	3.91	4.46	3.91	2.82	1.92	0.0
3	3.16	2.74	2.80	—8.02	—11.20	2.61	3.16	2.61	1.52	.62	0.0
4	3.16	2.74	2.80	1.98	—7.20	—11.39	3.16	2.61	1.52	.62	0.0
5	5.16	4.74	4.80	3.98	4.80	—3.39	—22.84	—3.39	3.52	2.62	0.0
6	3.16	2.74	2.80	1.98	2.80	2.61	3.16	—11.39	—8.48	.62	0.0
7	.76	.34	.40	—.42	.40	.21	.76	.21	—.88	—1.78	0.0
8	.76	.34	.40	—.42	.40	.21	.76	.21	—.88	—1.78	0.0
9	.76	.34	.40	—.42	.40	.21	.76	.21	—.88	—1.78	0.0
10	.76	.34	.40	—.42	.40	.21	.76	.21	—.88	—1.78	0.0
11	.76	.34	.40	—.42	.40	.21	.76	.21	—.88	—1.78	0.0
12	.76	.34	.40	—.42	.40	.21	.76	.21	—.88	—1.78	0.0
Column Sum	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	

TABLE 3B

Mean IQ Score for Each Combination of the Ten Genetic and Twelve Environmental Categories, when 75 percent of the Total Variance is Genetic, 15 percent is Environmental, and 10 percent is due to the Presence of Interaction Effects.

Environment (SES) Category (Rows)	*Genetic Category (Columns)*									
	1	*2*	*3*	*4*	*5*	*6*	*7*	*8*	*9*	*10*
1	39.46	61.37	81.53	85.29	88.71	92.00	95.38	99.17	103.90	113.18
2	71.37	72.23	61.08	83.02	92.26	95.36	99.29	102.53	106.17	114.36
3	72.03	80.89	85.74	78.68	78.92	96.02	99.95	103.19	106.83	115.22
4	73.27	82.13	86.98	89.92	84.16	83.26	101.19	104.43	108.07	116.46
5	77.10	85.96	90.81	93.75	97.99	93.09	77.02	100.26	111.90	120.29
6	77.40	86.26	91.11	94.05	98.29	101.39	105.32	94.56	102.20	120.59
7	77.33	86.19	91.04	93.96	96.22	101.32	105.25	108.49	112.13	120.52
8	79.71	88.57	93.42	96.36	100.60	103.70	107.63	110.87	114.51	122.90
9	81.58	90.44	95.29	98.23	102.47	105.57	109.50	112.74	116.38	124.77
10	82.85	91.21	96.56	99.50	103.74	106.84	110.77	114.01	117.65	126.04
11	84.79	93.65	98.50	101.44	105.68	108.78	112.71	115.95	119.59	127.98
12	89.04	97.90	102.75	105.69	109.93	113.03	116.96	120.20	123.84	132.23

Weighted by the proportional distribution of the black population given in Table 2C, this table results in a black average IQ of 82.59. The white population remains unaffected, with a mean of 100.0. Therefore a difference between the mean IQs of the two races of more than 15 points has occurred, yet the genetic potential of both races remains exactly equal.

genetic endowment. This means that, given a world in which a specific combination of genotype and environment always produced exactly the same measured IQ, we could, merely by changing the number of persons assigned to the specific genetic environment combinations, set the proportion of variance 'due to interaction' to various values. The statistical concept of interaction *enforced* by the Model II ANOVA, then always produces the *lowest possible* estimate of the percent of variance due to interaction. Indeed, in a world in which all the interaction terms were used, by selective assignment, to benefit one race, and to deprive another, *Model II ANOVAs carried out within the two groups separately would show no interaction variance in either case, but would translate the true interaction component into (in part) a displacement of the two groups on the genetic axis relative to one another.*

Thus, the importance of interaction is twofold: one, interaction components may in fact exist and if they do they must be correctly estimated or any findings

are worth little; two, the existence of interaction holds out the possibility of reallocating environments among persons so that all benefit and none are harmed. That is, interaction, in the theoretical sense, is an eminently 'political' variable. If no interaction exists, then that environment which is best for one child is also best for every other child. On the other hand, if interaction terms really appear in the effect table for genetics and environment, then some environments will be better for some children, and other environments will be best for other children. Given limits on the number of environments of one kind which we can produce, the presence of interaction allows us to benefit all children by reallocating the environments we do have.

Unreliability of P_I:

Based on the discussion above, we believe it is clear that the precision of the estimate of the interaction variance is crucial, if inferences concerning racial differences in genetic endowments are to be drawn. We now examine Jensen's estimation procedure.

The basic data from which Professor Jensen concluded that $P_I = .01$ appear in his Table 2 (p. 49). He notes that this value is obtained from four studies of monozygotic twins which yield a median r for IQ of .75, and five studies of unrelated children reared together, which give a median r for IQ of .24. From his components of variance model, the proportion of variance in IQ explained by the interaction component is $1.00 - .75 - .24 = .01$. The crucial point to note here is that the interaction proportion was not estimated directly from any data; rather, it was estimated from a linear combination of environment and genetic effects.

Now it is well known that the variance of an estimate which is a linear combination of two variables, is the sum of the variances for the two variables. (This assumes independently collected data for the estimates of P_E and P_G, which we will assume.) And since we have argued at some length that the value of the interaction component is critical, we decided to investigate the variance of Jensen's estimate quite closely.

In order to do this, we gave birth to approximately 540,000 twins, in a computer simulation of what Jensen's analysis would be like if done a great many times. Specifically, we wished to study the variance of the estimate of intraclass correlation coefficients of the type used by Jensen, when the coefficients are the median correlations taken from several studies. We thus generated sets of five twin studies, which came from a population with a specified P_E and P_G. Each of

the individual twin studies was based on somewhere between 30 and 60 twins, drawn at random from a uniform distribution. For each panel of five studies, the median r of the five r's was found. For each P_E and P_G combination, 200 such panels were generated. The variance of each set of 200 median r's was then computed, and the results are given in Table 4.

TABLE 4

Standard Errors of Estimates of Median Intraclass Correlation Coefficient r, *from Simulating 200 Panels of 5 Studies, each Study Containing 30-60 Twins.*

	r_G	r_E	r_I
coefficient	.80	.20	0
standard error	.0310	.0760	.0818
coefficient	.80	.10	.10
standard error	.0310	.0822	.0895
coefficient	.70	.20	.10
standard error	.0490	.0760	.0913
coefficient	.70	.10	.20
standard error	.0490	.0822	.0942
coefficient	.60	.20	.20
standard error	.0507	.0760	.0921

The remarkable property which these median estimates of r's display is their instability. For example, consider the situation where the true proportions of variance for G, E, and I are .70, .20, and .10 respectively. Then using simulated twin samples which tend to even be a bit larger than those used by Jensen, we see that the standard error of estimate of the interaction coefficient is 0.0913, or nearly as large as the coefficient itself. It is thus *not unlikely* that if the "true" interaction component was .10, Jensen would have found its value to be approximately zero, given the data available to him.

Suppose we even take a more extreme example in Jensen's favor, as his strongest argument appears to be that the heritability component is high, and approximately .75. If we let the components of variance for G, E, and I be .80, .10, and .10 respectively, then the standard error of estimate for the interaction component is still very high at 0.0895. Once again, given his sample size, it is not unlikely that Jensen would find a negligible interaction component when in fact the true component was 0.10. Yet, we have discussed earlier the great importance of

even a small interaction component in accounting for racial differences in observed IQ.

Finally, if we compare the standard errors of the median estimates given in Table 4 with those which would have been attendant to a *single large twin study, we see that the standard errors from the median estimates are approximately equivalent to having a single large study of 100 twins.* Thus, the estimates of the model parameters which Jensen uses as the underpinning for his entire analysis have approximately the same reliability as if he had selected one sample of 100 twins. That Professor Jensen would draw such far-reaching implications from a sample of this size, and in the complete absence of any black twin studies data at all, is remarkable.[10]

Quality of the Data:

Not only is the statistical reliability of Jensen's parameter estimates fairly low, but the quality of the twin studies data may be questioned as well. Jensen himself is well aware of the problems which attend testing young children from less advantaged backgrounds.[11]

When I worked in a psychological clinic, I had to give individual intelligence tests to a variety of children, a good many of whom came from an impoverished background. Usually I felt these children were really brighter than their IQ would indicate. They often appeared inhibited in their responsiveness in the testing situation on their first visit to my

[10] Our simulations averaged 225 pairs of twins per replication of 5 studies, and produced estimates with an average variance that would have been found in a single large study with 145 pairs of twins. The efficiency of the median is thus about 145/225, or 64.4 percent. Professor Jensen, for three MZ studies, reports 114 pairs of twins, and if we assume the fourth study had 40 twins, the effective number of pairs is about 100. (64.4 percent of 154 is 100.) The proper procedure for pooling intraclass correlations, drawn from the same population is, of course, by the use of Fisher's Z transformation, and not by taking medians. For the reader who wonders how seemingly minor technical details could modify the broad outlines of Professor Jensen's original arguments, we can only point out again that a few percent of variance can be responsible for large differences between group means. Yet we are dealing with statistical procedures which, even when perfectly applied, are subject to large sampling errors. To ignore such technical details is to ignore the crux of the argument.

[11] The last sentence in Jensen's quote is perhaps worth a note. If we wanted to measure the purely genetic component of IQ and to ignore or avoid the environmental component, then we would certainly not view the 8 to 10 point gain as having "much of anything to do with changes of ability." Clearly, if we are looking only for genetic factors, then all the environmental component is measurement error. But when we are attempting to resolve the apportionment controversy, we must, as Jensen said, rely solely on an operational definition which equates ability to the IQ score. If the score changed, ability changed. Jensen's inclinations in this matter are surprising.

office, and when this was the case I usually had them come in on two to four different days for half hour sessions with me in a "play therapy" room, in which we did nothing more than get better acquainted by playing ball, using finger paints, drawing on the blackboard, making things out of clay, and so forth. As soon as the child seemed to be completely at home in this setting, I would retest him on a parallel form of the Stanford-Binet. A boost in IQ of 8 to 10 points or so was the rule; it rarely failed, but neither was the gain very often much above this. So I am inclined to doubt that IQ gains up to this amount in young disadvantaged children have much of anything to do with changes in ability. (p. 100)

In other words, Jensen feels that certain prior environments 'inhibit responsiveness' and thereby lower IQ scores rather uniformly, by about 8 to 10 points—which is more than half the difference between the white and black mean IQs found by Coleman. Because of our national prejudice against the black person, we would expect that the black child would learn quite early in life that whenever he was in a setting in which 'evaluation' was likely to occur, then the experience was probably going to prove unpleasant. He may thus display inhibited responsiveness, possibly of a kind not so easily overcome by fingerpainting.[12]

Internal Consistency of Parameter Estimates:[13]

1. Monozygotic Twin Studies:

The monozygotic twin studies summarized by Professor Jensen estimate the genetic component of phenotypic variance only if both members of the pairs are identical genetically and otherwise assigned to environments at random. Besides their genetic endowment there is, after all, one other thing identical twins have in common: they presumably spent the first nine months of life in identical

[12] There may be some confusion between environmental effects and unreliability of IQ scores. Think of a good wall thermometer; the small variations in its reading from occasion to occasion are not unreliability—they are the changes in the room temperature, and it was for the purpose of detecting them that we put the thermometer in the room. To assume that any fluctuation in measured ability subsequent to conception must be due to unreliability in the measurements is to fall into the concept of fixed intelligence which Hunt so completely demolished. Of course, such environmentally derived gains or losses in IQ are not maintained unless the environmental factors which contributed to them also persist.

[13] The reader will need to have one point firmly in mind throughout the following discussion. All three kinds of pair studies—MZ twins reared apart, MZ twins reared together, and foster children reared together—estimate two proportions. Each directly estimates the proportion of phenotypic variance due to whatever factors both members of a pair have in common. Unity minus that number estimates the proportion of variance due to whatever factors are not identical for both members of a pair. The procedure is completely blind; it does not specifically estimate genetic factors or environmental factors. Whatever affects both children in each pair identically, appears in the direct estimate. Whatever has been randomly assigned to both children in a pair, appears in the indirect estimate.

environments. Jensen argues at length that the prenatal environment may carry a large part of the total environmental impact upon IQs:

> Thus, much of the average difference between MZ twins, whether reared together or reared apart, seems to be due to prenatal environment factors. The real importance of these findings, of course, lies in their implications for the possible role of prenatal environment in the development of all children. It is not unlikely that there are individual maternal differences in the adequacy of the prenatal environment. (p. 68)

And, he later adds:

> There is no doubt about the fact of the greater prevalence in poverty areas of conditions unfavorable to optimal pregnancy and safe delivery. (p. 69)

But those differences are included in the 75 percent of the phenotypic variation assigned to genotypic allotments, merely because the MZ twins have them in common. Further, the foster pair studies would also *underestimate* the environmental variance for the same reason. The foster children did not have common prenatal environments.

Jensen further indicates that:

> In pairs of identical twins, the twin with the lower birth weight usually has the lower IQ (by 5 to 7 points on the average) at school age. This is true in both white and Negro twins. The birth-weight differences are reflected in all 11 subsets of the Wechsler Intelligence Scale for Children and are slightly greater on the Performance than on the Verbal Tests (Willerman and Churchill, 1967). The investigators interpret these findings as suggesting that nutrient supplies may be inadequate for proper body and brain development in twin pregnancies, and that the unequal sharing of nutrients and space stunts one twin more than its mate. (p. 68)

Since this is a *difference* between the twins, it will show up in the part of the total variance not accounted for in the genetic intraclass correlation. It has been added to the $(1.00 - .75 = .25)$ environmental column. But the great majority of the natural population are singletons, not twins, and they would not be subjected to an environment which included competition from their womb-mates. Therefore, we may be misestimating the total variance in the natural population when we make use of the MZ twin studies, and also *overestimating* the effects of environment.

We do not know how to resolve this potential conflict in estimation; the MZ twin studies might have underestimated or overestimated P_E. Our point is merely that the effects may or may not cancel, and thus the estimate of P_E may or may not be biased.

2. The Foster-Pair Discrepancy:

Jensen points out:

> Another interesting comparison is between MZ twins reared together (r = .87) and reared apart (r = .75). If 1.00 − .75 = .25 (from the MZ twins reared apart) estimates the total environmental variance, then 1.00 − .87 = .13 (from MZ twins reared together) is an estimate of the environmental variance *within families* in which children are reared together. Thus, the difference between .25 − .13 = .12 is an estimate of the environmental variance *between families.* (p. 51)

That would certainly be the case. Except that the estimate of the proportion of total variance due to environmental differences, which is derived from studies of unrelated foster pairs raised together, is also an estimate of only the *between family* differences in environmental conditions. That estimate, from Jensen's Table 2, was .24, not .12. In fact, if we add the several estimates up:

Genetic factors:	
(from MZ twins reared apart)	.75
Within family differences	
(from MZ twins reared together)	.13
Between family differences	
(from unrelated foster pairs raised together)	.24
	1.12

We have just accounted for 112 percent of the total variance, which is not possible. Thus, there is some question as to how to appropriately adjust these three components so that they add correctly.

3. Covariance Between Heredity and Environment:

Jensen states that:

> Children with better than average genetic endowment for intelligence have a greater than chance likelihood of having parents of better than average intelligence who are capable of providing environmental advantages that foster intellectual development. (p. 38)

In other words, Jensen implies that if the covariance term between heredity and environment exists, then it should be positive.

Suppose that in accordance with Jensen's suggestion, we assume that there is a positive covariance term in the 'real world' sampled by the studies of MZ twins reared together, but that this term also appeared in both the MZ twins reared apart and in the foster pair studies. While this would be experimental error, one

might suspect that neither twins nor foster children are assigned to substitute families at random. This approach might help us to "repartition" the components of variance so that they add to 1.00. In other words, suppose we assume the covariance term was 'counted twice;' it was included in the estimate of .75 attributed to genetic factors by the MZ reared apart studies, and then included again in the .24 attributed to between families environmental conditions by the foster pair studies. Let's see what happens:

P_G is the proportion of variance due to genetic factors
P_B is the proportion of variance due to between family environmental differences
P_W is the proportion of variance due to within family differences
P_C is the proportion of variance due to the covariance of genetic and between-family differences

We know that all four proportions must add to 1.00 by definition. We also have data from three types of studies (and we are assuming that the total variance was the same in all three sets of studies), which gives us three additional equations:

By definition:

$$P_G + P_B + P_C + P_W = 1.00$$

From the MZ twins reared together studies:

$$P_G + P_B + P_C = .87$$

From the MZ twins reared apart studies:

$$P_G + P_C = .75$$

From the foster pair studies:

$$P_B + P_C = .24$$

We can then solve for the following estimates:

$$P_G = .63$$
$$P_B = .12$$
$$P_W = .13$$
$$P_C = .12$$

The heritability has been reduced to .63 and the environmental component increased to .37. Although we do not wish to argue that these revised proportions are in reality correct, this again points out that Professor Jensen's analysis contains an inconsistency which must be resolved.

4. Combining the Unreliability and Foster Pair Discrepancy Arguments:

If we combine the results of our twin study simulations from Table 4 with the inconsistency argument presented above, we see that there really need be no discrepancy at all, and that the four components may not sum to 1.00 simply because of sampling errors. Our simulations have shown that estimates of the median intraclass correlation from five fairly small studies are quite erratic when the true proportion of variance due to the factor equated within pairs is small. Thus, perhaps the true proportion of variance between families is in reality only .12, and the median value of .24 came about due to sampling errors. (In fact, given Jensen's sample sizes, the standard deviation of the distribution of medians is about .08 when the true proportion of variance is .12. Thus, using a normal approximation, the observed median value of .24 is *not* significantly different from a hypothesized value of .12 at the .05 level of significance, one-sided or two-sided.) However, with that kind of error lurking in the wings, the stage machinery of a Model II ANOVA may not be the best way to dramatize the features of our world's environments which have raised or afflicted our children's intellectual capacities.

Summing Up:

In concluding this section, then, there are serious questions which need answering before Jensen's parameter estimates can be accepted. We have shown how a small amount of interaction between genetic endowment and environment can easily explain how two races with identical genetic endowments can have large differences in mean IQs. We have further argued that even assuming the twin studies data were of excellent quality, the data are not at all statistically inconsistent with the existence of something like a .10 interaction component of variance, and that this magnitude of interaction could account for mean differences of more than one standard deviation in black-white IQs without any genetic differences between races (see Table 3). Finally, the inconsistencies of the data in their additive behavior, together with the complete lack of any data at all on black twins, in our judgment wrecks the credibility of even a tentative assertion of genetic differences in intelligence between races.

Statistical Assumptions and Model Considerations:

As the study of race and intelligence is invariably shrouded by both political and statistical debate, we wish to take the liberty of offering a few observations on the inherent problems of measurement and model selection.

All scientists know that one of the most crucial stages in the research process is the model selection. For example, Jensen has selected the Model II ANOVA to estimate his parameters, and therefore implicitly agrees to be bound by its assumptions, limitations, and descriptive ability of the real world. After all, incorrect assumptions will permit meaningless or misleading conclusions to be drawn from extensive, perfect information. Thus, the selection of a model has the limited virtue of making obvious its dependence upon the worth of the direct measurements it requires.

If this comment suggests that we believe that scientists cannot really resolve controversies such as this until direct identification of genotypes and environments is possible, that is correct. Jensen says:

> Determining the heritability of a characteristic does not at all depend upon a knowledge of its physical, biochemical, or physiological basis or of the precise mechanisms through which the characteristic is modified by environment. Knowledge of these factors is, of course, important in its own right, but we need not have such knowledge to establish the genetic basis of the characteristic. (p. 44)

We believe Jensen is incorrect: determining the heritability of a characteristic in a single population not only depends upon a knowledge of its physical, biochemical, and physiological basis but also on an equally exact knowledge of its environmental contributors. Where we have reason to believe that we can identify and isolate the causes of a characteristic experimentally, we can control for or randomize various combinations of factors and thus can employ certain statistical techniques with some immunity from gross error. Without such *a priori* knowledge, we would be driven to consider land-ownership during the middle ages to be a sex-linked dominant trait in first-born males with a coefficient of heritability near unity. Our knowledge that the currently proposed genetic mechanisms cannot single out the first-born prevents us from drawing that conclusion, and substantial documentation about the social system at that time corroborates our decision.

However, when someone asks whether the difference in observed mean IQs between white and black populations is attributable to genetic differentiation, a fundamentally different problem is being posed. It bears directly on the choice of a components of variance model to investigate racial differences.

Any components of variance model presupposes that the sources of variation can be distinguished and crossed in the experimental situation—*not only can be, but were*. Placing a covariance term in a model handles the possibility that two

sources of variation are linked in the real world—but only conceptually. Every study Professor Jensen reports has neglected the most obvious linkage between genetic and environmental factors: that the environment is composed of sentient beings who note genetic differences and act to differentiate them further.

A genetic characteristic revealed by external measurement can function both as a sign and as a signal. For example, when we detect the blueness of an eye we have the sign of the presence of a specific genotype. The blueness of an eye may also be the signal for a set of social processes to occur (love, or so we are told); but that entails no difficulty in deriving estimates of heritability coefficients, because we have quite definite information to the effect that environmental conditions cannot change eye color. But if the environment can also affect the measured characteristic, then the possibility of a feedback loop is real: that is, the environment reads the sign of the genetic variation as a signal for initiating social processes which enhance the variation; the enhanced variation in the characteristic is then again taken as the signal for another round of differentiation, and so forth. Further, we have no reason to believe that the enhancement of individual differences would be either as strong or as accurate for one social group as for another.

We certainly know that human environments do read genetic signs as social signals, and not just for romantic love: a darkened skin is more than the genetic sign of the transmissable presence of melanine—it has become the signal for an array of social processes in our society whose scope is currently unknown and whose nature is perhaps best explicated by others. The essential point, however, is that monozygotic twins reared apart have not been stripped of the signs of genetic differences in IQ that could signal the occurrences of other social events. Jensen, in dismissing Rosenthal's experiment on teacher expectations on statistical grounds, failed to note that it is one of the very few instances in the literature when a researcher deliberately tried to mislead the signal-seeking component of a social system about a genetic sign. Our ability to account for a difference in population means is wholly dependent upon the success of such separations as the one Rosenthal attempted.

Serious future research into questions of race, environment, and genetic endowment must take into account this environmental feedback process, both in the choice of a model, and in the design of surveys or experiments to collect data. Otherwise, many of the crucial processes which affect IQ will be ignored, and research results may be little more than projections of the assumptions of their authors.