The Davis cases were a result of extreme social isolation precipitated by the fact that the girls were illegitimate and relatives of the mothers kept them in seclusion to keep neighbors and the community from knowing of their existence. The classic case of such isolation was seen in the first seven years in the life of Helen Keller. Because of blindness and deafness acquired early in infancy, she was cut off from the linguistic development necessary to fully realize her innate intelligence. Once she was provided a special teacher, Anne Sullivan Macy, who knew and understood her plight and knew how to communicate with her, she was able to grasp the meaning of language and rapidly developed into the gifted person she was capable of becoming (Lash, 1980).

Although I agree with Neisser et al. (1996) that many critical questions about intelligence are still unanswered, I disagree with them when they stated that "no adequate explanation of the differential between the IQ means of Blacks and Whites is presently available" (p. 97). I think the studies of Yerkes (1921), Klineberg (1935), and Lee (1951) provide a reasonable explanation of that differential. Furthermore, the studies of Davis (1947) and Lash (1980) point out how very critical the opportunities for language development are in the realization of normal intelligence.

On the other hand, the work of Dennis (1942) shows that the subculture of some ethnic groups, like that of the Hopi people, provides for them a distinct advantage on tests like the Goodenough Draw-a-Man Test, which tests for drawing ability. Hopi children are consistently superior to White children on that test because at an early age they learn how to draw and love it. What Dennis (1942) showed so dramatically is that one's subculture often makes a big difference in how people perform on IQ tests.

When it comes to our full understanding of intelligence, there still remain many issues unresolved and so many questions unanswered. The Neisser et al. (1996) contribution to the quest for greater knowledge about intelligence is deeply appreciated. It has helped to shed light on the many misconceptions found in the bestselling book, *The Bell Curve* (Herrnstein & Murray, 1994).

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"The Genetic Hypothesis": It Was Not Tested but It Could Have Been

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I wish to comment on "The genetic hypothesis" (p. 95; for the Black-White difference in psychometric intelligence) in the Neisser et al. (February 1996) article, particularly the reference to two studies that used blood groups to estimate the degree of African ancestry in American Blacks in relation to their IQ scores (they found no relation). I have experience in such admixture estimation (e.g., Reed, 1969, 1973) and, as mentioned in the target article (Reed & Jensen, 1992, 1993), in studying biological factors in intelligence. My 1969 article gave the first estimate of the proportion of White ancestry in American Blacks (Pw) with a standard error, 0.220 ± 0.0093 (using the Duffy blood group gene Fy), and because it was based on large samples (more than 3,000 each of Blacks and Whites), it remains the best single estimate for non-Southern American Blacks.

I contend that, because of their methodology, the two studies cited above—Loehlin, Vandenberg, and Osborne (1973) and Scarr, Pakstis, Katz, and Barker (1977)—did not adequately test the possible association of cognitive ability with Pw. Consequently, their negative results provide no evidence against the genetic hypothesis. I suggest a method that, had it been used with data of the second study and if the genetic hypothesis is true, probably would have confirmed the genetic hypothesis.

The methodologies of these two studies share a basic misconception—that all blood (and serum) groups are useful in estimating Pw. This is plainly false, as I (Reed, 1969) showed. The Pw estimate in this population, using the A and B genes of the ABO blood groups, was 0.200 ± 0.044; the above estimate with Fy* provides (.044)/(.0093) ± 22 times more information than this ABO estimate. If I had estimated Pw using the MN blood groups (both the Loehlin and Scarr groups used them), the standard error would have been even much larger than for ABO and would have been worthless (see below).

The racial informativeness of a gene used to estimate Pw (measured by the reciprocal of the variance of Pw) is a function of its relative frequencies in the two ancestral populations, African and White. A genetic Locus I is perfectly informative (an "ideal locus"); MacLean et al., 1974; Reed, 1973) when it has two codominant alleles (genes; say I+ and I−), with one allele being homozygous (i.e., II or I−I−) in all individuals of one ancestral population and the other allele being homozygous (I+I+) in all individuals of the other ancestral population. Thus, when testing an American Black, every allele at this ideal locus derived from a White ancestor is recognized as such. The Gm serum group locus (testing for nine factors) closely approximates such an ideal locus, but with multiple alleles; three are White alleles and four are sub-Saharan African alleles (Roychoudhury & Nei, 1988). The Fy* allele alone, with a
frequency of about .43 in Whites and about .01 in Africans, is not ideal. When present in an American Black person, we are reasonably sure that it came from a White ancestor, but other White matings could have contributed an Fy b allele (frequency about .57 in Whites and about .01 in Africans) and so would not be recognized (when testing only for Fy a). But contrast this with the situation using the MN blood groups: In both Whites and Africans, the M and N alleles each have frequencies close to .50. This locus provides essentially no information on the ancestry of American Blacks!

The consequences of using all blood and serum groups available, without regard to their great differences in racial informativeness, as the Loehlin (Loehlin et al., 1973) and Scarr (Scarr et al., 1977) teams did, are severe.

**Loehlin Group**

Of the eight blood-group systems used, only Duffy (using Fy a) has some utility in Loehlin et al.'s (1973) small sample of Black persons (42 twins). Assuming that they had the equivalent of 60 unrelated individuals (their sample contains monozygous and dizygous twins), one can calculate that a P a for their sample would have a standard error of about .064 and, therefore, a large 95% confidence range (about .24). Other blood groups would have considerably larger standard errors and confidence intervals and, so, give little or no information. Yet Loehlin et al. performed rank-correlation between blood-group genes (arranged in descending order of the difference between frequencies in Whites and Blacks) and association with cognitive ability. The small sample size and noninformativeness of most blood groups mean that, except for Fy a, they were usually dealing with noise, and their negative result was to be expected.

**Scarr Group**

Scarr et al. (1977) used Black twins from the Philadelphia area, and the number (181) was large enough, using both Fy a and Fy b of the Duffy group, to give a useful estimate of P a. Gm serum groups were determined (testing for four factors) and could also have given a useful estimate of P a. Ten other groups were also tested. Scarr et al. attempted to obtain for each individual a measure of individual ancestry to associate with an estimate of cognitive ability, but this measure is deeply flawed. They used an "odds coefficient," log(A a A a A a A a /B b B b B b ...), in which A was one ancestral population (e.g., African) and B was the other ancestral population, and the subscripts were the loci of the different blood and serum groups. A was the frequency of an individual's phenotype (group) at Locus 1 in Population A, B was the frequency of his or her phenotype in Population B at that locus, and so on. This coefficient was intended to give a rank ordering of individuals according to their degree of ancestry from one population, say A.

Now consider the effect of one uninformative Locus X, for example the MN blood groups, on this coefficient. Because A/B varies essentially at random, and A/B multiplies all the other ratios, the odds coefficient acquires considerable randomness. Add the random effects of other only slightly informative loci, such as the ABO, and the coefficient will necessarily lose much of its potential for ancestry identification. Scarr et al.'s (1977) procedure for dealing with zero phenotype frequencies—replacement by .0001—further distorts the coefficient, particularly for the informative Duffy and Gm groups. This is because, with the usual sample sizes, absence of a phenotype at Locus Y does not mean that its true frequency is not of the order of .01-.001. This procedure would often bias log(A a B b) by about ± 1 to ± 2. With the above problems, it is not surprising that the correlations between the odds coefficient and the measures of cognitive skills were nonsignificant; it would be surprising if they were otherwise. (Incidentally, although Scarr et al. thanked me and others for "consultation on the design and analysis of the study" [p. 86], I did not have any part in the design or analysis.)

**For a More Powerful Test of the Genetic Hypothesis**

MacLean et al. (1974) studied 372 adult Blacks in the Rochester, New York, area for possible correlation of diastolic blood pressure (DBP) with proportion of African ancestry (P a). They used 10 blood and serum groups, including Duffy and Gm, and corrected the DBP readings for gender, age, and obesity. Although they recognized that accurate individual estimates of P a were not possible (Reed, 1973), such estimates were made anyway, and the corrected DBP values were regressed on them. A very significant (p < .001) positive linear regression was found: Increasing DBP accompanied increasing P a. Evidently, the overall information on P a was more than adequate, although individually inaccurate. But the point of this account is that DBP is a surprisingly good surrogate for IQ score: Both are quantitative traits, are moderately heritable (h² for DBP is estimated to be .37 by Cavalli-Sforza & Bodmer, 1971), and have similar relative changes going from 100% African ancestry to 100% White ancestry. Furthermore, the t value (3.4) for their regression is still significant at the .001 level for 120 degrees of freedom. Therefore, if the genetic hypothesis was true, I predict that the MacLean et al. methodology applied to the Scarr et al. (1977) data would show this.

Scarr et al. (1977) knew of the MacLean et al. (1974) study (they referred to it), but they chose to use their own method. After expending so much effort in collecting their data, it is a pity not to analyze them properly.

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