gence must be assumed to be extremely population specific, which is probably the cause for the enormous range of heritabilities reported (e.g., .20 to .87 according to Atkinson, Atkinson, Smith, & Bem, 1993).

But there is not only the question of generalizability. There is the more fundamental question about the use of heritability coefficients altogether. Two implications of the "substantial" heritability of intelligence have repeatedly been postulated: limited changeability of cognitive abilities (e.g., Jensen, 1969) and demand for eugenic measures (e.g., Herrnstein & Murray, 1994). The report (Neisser et al., 1996) correctly pointed out that "heritability does not imply immutability" (p. 86), but considering the intuitive plausibility of the arguments for both immutability and eugenics, it failed to explicate the somewhat counterintuitive nature of heritability in quantitative genetics in a way that prevents readers from falling into the traps of intuition.

With the environmentally caused variability in the denominator of the definition of h^2 (see last equation), an empirically determined heritability is dependent on environmental variability (e.g., differences in school quality, intellectual background of families) in the sense that the larger the environmental variability, the smaller the heritability, and vice versa. We may, for example, imagine an egalitarian society in which children grow up under essentially identical conditions, which would mean an accordingly very high degree of heritability. It is clear that this very high heritability does not have anything to do with the possibility of changing the intelligence of a member of that society by some kind of intervention simply because it is due to the lack of environmental variability. So if a heritability coefficient is estimated for some population, its size depends on the environmental variation in the sample as it is and not as it possibly could be. It cannot tell anything about the possible effects of interventions because these, if they did take place, would result in a different (i.e., smaller) heritability. Let me stress that, counter to intuition, the size of such effects can in no way be predicted from the heritability coefficient as estimated because no knowledge exists about the relation between the size of the environmental effects as operating in the "ordinary population" and the size of the effect of the interventions.

All of this being so, there can also be no meaning in eugenic measures with respect to intelligence because, contrary to the impression left by heritability estimates, the relative strength of genetic effects and the effects of possible environmental measures are unknown.

The vast differences between populations in environmental variability and the resulting extreme relativity of heritability estimates for intelligence in my view make them of little use for predictions of any kind.

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Direct Evidence for a Genetic Basis for Black–White Differences in IQ

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The APA Task Force on Intelligence (Neisser et al., February 1996) gave a generally fair summary of the state of knowledge until the final section on Black–White differences. At that point, the article deteriorated. The Task Force dismissed the thesis that the difference may have some genetic basis with the following statement: "There is not much direct evidence on this point, but what little there is fails to support the genetic hypothesis" (p. 95). This conclusion cannot be accepted. Two items of direct evidence are particularly persuasive.

First, it has been argued for some time that the adoption of Black babies by White parents would provide crucial evidence on the genetic hypothesis. As Rose, Kamin, and Lewontin (1984) stated, "The only way to answer the question of genetic differences in IQ between groups would be to study adoption across racial and class boundaries" (p. 127). The required experiment was carried out by Weinberg, Scarr, and Waldman (1992), and the results were that Black babies adopted by White parents registered no IQ gains. This conclusion was argued in detail by me (Lynn, 1994). Weinberg et al. attempted to dispute it, but Scarr (1995) conceded that my interpretation of the study was correct. Scarr wrote that "those adoptees with two African American birth parents had IQs that were not notably higher than the IQ scores of black youngsters reared in black families" (p. 7). The experiment showed that matching Blacks and Whites for family environment did nothing to reduce the IQ difference and, hence, provided direct evidence for a genetic basis for the difference.

A second source of direct evidence consists of Black–White differences in average brain size, taken in conjunction with the fact that brain size is positively associated with IQ. Seven data sets summarized by Rushton (1995, pp. 127–130) showed a consistent White advantage of approximately 4%. An additional data set analyzed in Lynn (1993) confirmed this conclusion. The positive association between brain size and IQ has been shown in numerous studies reviewed by Jensen and Sinha (1993).

The Task Force (Neisser et al., 1996) did not say what it meant by "direct evidence." It would be useful if the Task Force would specify what evidence it would accept as falling into this category. Pending this clarification, I suggest that it is difficult to think of any evidence more direct than the results of the transracial adoption study and the racial differences in brain size.

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Intelligence and Lead: The "Known" Is Not Known

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Considered overall, the APA Task Force on Intelligence report (Neisser et al., February 1996) is sound and well-balanced. Nevertheless, the short section (p. 89) on lead as a biological variable with a negative effect on intelligence is very misleading. In the most objective and comprehensive review available, Pocock, Smith, and Baghurst (1994) reported a deficit of 1–2 IQ points associated with a typical doubling of blood lead from 10 to 20 μg/dL. This synthesis calculation was based, in part, on meta-analyses. More important than the analyses, however, are Pocock et al.'s succinct comments that reflect inconsistencies in findings, the failure to control fully (or at all) for confounding influences in many studies, and the very real possibility of reverse causality (i.e., the risk that children of lower intelligence are more likely to access and ingest lead). The situation is further clouded in that some of the most widely cited research on the topic is characterized by substandard science (Office of Research Integrity, 1994).

The cited Port Pirie study (Baghurst et al., 1992) is indeed one of the better and larger studies, but startling results showed an adverse effect for girls but not boys (Baghurst et al., 1992; McMichael et al., 1992). This finding, considered in conjunction with a report of an adverse effect for boys but not girls (Pocock, Ashby, & Smith, 1987), is indicative of the uncertainties that permeate this issue. It has been suggested that the inconsistencies are due to characteristics of the populations sampled, that is, that socioeconomic status, ethnicity, gender, or other characteristics render some children more vulnerable than others. However, findings of effect for specific groups (i.e., interaction effects) have not held up under replication (Ernhart, 1995). Given the small effect sizes obtained among studies reporting adverse findings, the cautions made by Pocock et al. (1994) merit considerable attention. If there is an effect of lead exposure at low levels, that effect is small indeed.

The issue is not merely academic (Shell, 1995). Policymakers in governmental agencies have been strongly influenced by advocates who have a stake in declaring that lead poisoning is the number one environmental disease of children, and who, in so doing, divert resources from an attack on the socially toxic environment (Garbarino, 1995) to which all too many children are exposed. The costs are high in terms of unnecessary regulations, expensive abatement programs, and exorbitant litigation, as well as unwarranted anxiety on the part of parents.

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Methodological Errors in the Prediction of Ability

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Neisser et al. (February 1996) failed to challenge repeated methodological errors in the prediction of intellectual ability. To the extent that they did, they tended to sustain, however unintentionally, the hereditarian position. I focus on two such errors, one related to monozygotic twins and the other to the absence of information on the actual Scholastic Aptitude Test (SAT) score distributions in group studies.

McGue, Bouchard, Iacono, and Lykken (1993) reported IQ correlations ranging from .68 to .78 for monozygotic twins reared apart, an imputation of high "heritability" (h^2). Statements of this nature ultimately confound genotypical and phenotypical considerations. Monozygotic twins have more than a shared heredity; they also look the same! Ordinarily, the genotypical substrate of each twin's makeup is not part of the onlooker's psychological field. What is seen in one environmental context is a person who has a certain appearance, and what is seen in the other environment, where the other twin resides, is a person with the identical appearance. Suppose that aspects of appearance per se are efficient stimuli for environmental reaction and that the developing twin's interaction with the reactors elicits responses that may encourage or depress the manifestations of intelligence. The proper control for this could be to match a monozygotic twin not only with the identical sibling but also with unrelated persons whose appearance is judged to be similar to that of the twins. The degree of matching could rest on such criteria as observer ratings, measurements, skin surface variations, and so forth. As an ancillary investigation, one could find adoptive homes where two adoptees tend to match each other in appearance. If this is not a pertinent variable, then one would expect the correlations between the children in such families to approximate zero, as is the case in the generality