

## What if the Hereditarian Hypothesis Is True?

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### Abstract

Rushton and Jensen (this issue) review ten bodies of evidence to support their argument that the long-standing, worldwide Black-White average differences in cognitive ability are more plausibly explained by their “hereditarian” (50% genetic causation) theory than by “culture-only” (0% genetic causation) theory. This commentary evaluates the relevance of their evidence, the overall strength of their case, the implications they draw for public policy, and the suggestion by some scholars that the nation is best served by telling benevolent lies about race and intelligence.

### What if the Hereditarian Hypothesis Is True?

Rushton and Jensen (this issue) review the last thirty years of evidence on an important but spurned question: “Is the average Black-White difference in *phenotypic* intelligence partly *genetic* in origin?” Much relevant scientific evidence has accumulated since Jensen first asked the question in 1969 (Jensen, 1969), but openly addressing the question still seems as politically unacceptable today as it was then. Taking the question seriously raises the possibility that the answer might be “yes,” which for some people is unthinkable. People who recoil from the question likewise recoil from those who ask it, often accusing them of wanting to twist evidence toward that answer. It is therefore no surprise that such research and researchers are often evaluated first against moral criteria and only secondarily, if at all, against scientific ones (Gottfredson, in press b). My commentary on the Rushton-Jensen paper will therefore examine its scientific merits, but also the appropriateness of the other criteria typically applied to such work.

#### I. The Hereditarian Hypothesis: What Is It?

The authors’ “hereditarian hypothesis” is that Black-White differences in general intelligence (IQ, or the general mental ability factor, *g*) are “substantially” genetic in origin, which they quantify as 50% genetic and 50% environmental. They specify 50% genetic because they hypothesize, more generally, that “race differences are simply aggregated individual differences” and because researchers commonly summarize within-group IQ heritability as 50% (it rises from about 40% in childhood to 80% in adulthood in Western samples). They do not attempt to conclusively prove a genetic component, but to show that their hypothesis is more plausible than its major rival, the “culture-only hypothesis,” which entails 0% genetic and 100% environmental causation. The 0-100 mix is a legitimate contrast to their 50-50 hypothesis because social scientists have long tended to deny that there is *any* genetic component

whatsoever to race-IQ differences. By genetic difference, the authors mean that different racial groups differ in their frequency of different alleles (versions of the genes) affecting intelligence.

## II. Scientific Foundations of the Hereditarian Hypothesis: How Sound?

The hereditarian hypothesis becomes scientifically plausible, however, only after at least five evidentiary prerequisites have been met: specially, that IQ differences among *same-race* individuals represent (a) real, (b) functionally important, and (c) substantially genetic differences in general intelligence (*g*), and that mean IQ differences *between* the races likewise reflect (d) real and (e) functionally important differences on the same *g* factor. Critics often do, in fact, try to render the hereditarian hypothesis implausible and unwarranted by claiming that one or more of these evidentiary pillars is missing or already discredited (although critics disagree on which ones are sound and which ones hollow).

A century of research strongly supports all five. It has provided a vast, interlocking network of evidence that *g* is the backbone of all broad mental abilities in all age, race, sex, and national groups yet studied; that higher levels of *g* confer practical advantages in many realms of life; that *within*-group variability in phenotypic *g* has strong genetic roots and many physiological correlates in the brain; and that *between*-group differences in *g* are large and pervasive enough to have broad social significance (e.g., see the journal *Intelligence*; syntheses in Brody, 1992; Carroll, 1993; Deary, 2000; Jensen, 1980, 1998; Plomin, DeFries, McClearn, & McGuffin, 2001; Schmidt & Hunter, 1998; Sternberg & Grigorenko, 1997, 2002; and special journal issues by Ceci, 1996; Gottfredson, 1986, 1997a; Gottfredson & Sharf, 1988; Lubinski, 1996, in press; Williams, 2000). There is thus no scientific reason to spurn the obvious next question. In fact, a plurality of intelligence experts surveyed in 1985 (Snyderman & Rothman, 1988, p. 141) reported that, in their opinion, evidence already favors the conclusion that there is some rather than no genetic involvement in the Black-White IQ difference.

This is hardly the picture of intelligence research that the media and many social scientists paint (e.g., Fish, 2002; Jacoby & Glaberman, 1995). Both often suggest that the whole effort to measure mental abilities—the entire field of psychometrics—is fundamentally flawed and morally suspect (e.g., Fischer et al., 1996, reviewed by Gottfredson, 1997c). As Snyderman and Rothman (1987, 1988) showed almost two decades ago, however, media portrayals of accepted wisdom on intelligence tend to be *opposite* the experts' actual conclusions (see also Carroll, 1997; Gottfredson, 1997b; Hartigan & Wigdor, 1989; Neisser et al., 1996; Wigdor & Garner, 1982; Wigdor & Green, 1991). Thus, despite public lore to the contrary, there is already a deep and vast nomological network of evidence that can be called *g* theory. It constitutes the prerequisite evidentiary base for posing the hereditarian hypothesis.

### III. The Authors' Ten Bodies of Evidence: How Pertinent? How Complete?

The most general difference between *g* theory and culture-only theory is that the former sees both individual and group differences in *g* as embedded substantially in biology, while the other theory looks only to culture, at least when it involves race. The two theories make different predictions in circumstances where genetic and cultural variation are somewhat independent. In such circumstances, the hereditarian hypothesis predicts that racial gaps in IQ will trace degree of genetic relatedness *at least* as much as cultural similarity, whereas culture-only theory predicts the gaps to mirror cultural variation. I will illustrate three rounds in this prediction contest with evidence from seven of the authors' ten bodies of data.

#### Contrasting Predictions

First, although both theories predict ubiquitous race differences in phenotypic abilities, *g* theory predicts that the gaps between any two particular races will be similar over time and place regardless of cultural circumstances (unless frequency of interbreeding changes markedly). Culture-only theory predicts that the gaps will expand or contract depending on similarity in cultural environments, regardless of genetic heritage. The considerable uniformity of the IQ gaps between African Blacks,

American Blacks, Whites, and East Asians over time and place (Rushton and Jensen's Section 3) and the parallel ordering of race differences on simple reaction/inspection time tests in the United States and elsewhere (4) are both consistent with *g* theory. The IQ gaps fail to shift in tandem with cultural variation, contrary to what culture-only theory predicts. The occurrence of the same IQ gaps by race in trans-racial adoption (7) and racial admixture (8) studies have been construed as consistent with both theories when involving Blacks, but the above-average mean IQ of even badly malnourished East Asian infants adopted into White-European homes is more consistent with those infants having a genetic than a cultural advantage over their White-European peers.

Second, *g* theory predicts, but culture-only theory does not, that IQ differences will correlate with variation in "hard-wired" aspects of brain structure and function. The former theory, but not the latter, can therefore account for the nexus of correlations between the *g* loadedness of IQ and reaction time tests (their ability to measure *g*), the tests' heritability and susceptibility to inbreeding depression, Black-White-East Asian mean differences in performance on them, and various physiological traits correlated with good performance (brain size, brain evoked potentials, brain pH levels, and brain glucose metabolism; Sections 4 and 6).

Third, the two theories predict different degrees of *change* in individuals' IQs when their socioeducational environments change substantially: *g* theory predicts little or no lasting change but culture-only theory predicts relative responsiveness. Jensen's (1969) thirty-year-old conclusion about the failure of socioeducational interventions to substantially and permanently raise low IQs still stands (Section 12). Natural variation in environments likewise fails to alter the common developmental processes by which abilities are assembled or built in different races. This commonality in cognitive architecture is indicated by cross-race identity of *g* factors, input-output achievement covariance matrices, and the like (5). It contradicts predictions that different cultures create different intelligences.

### Additional Evidence

The authors do not discuss one body of evidence that many social scientists believe undermines their hereditarian hypothesis: a narrowing of Black-White gaps in standardized academic achievement on the National Assessment of Educational Progress (NAEP), which critics generally take to represent a narrowing of the IQ gap as well. That narrowing has been cited since the first decade of the NAEP trends survey, begun in the late 1970s, as portending a closing of the gap (for recent discussions, see various chapters in Jencks & Phillips, 1998; Neisser, 1998). If true, this would seem to refute Rushton and Jensen's claim that the American Black-White IQ gap remains at 1.1 SD, unresponsive to environmental change.

Although culture-only theorists tend to equate IQ and academic achievement, the two are conceptually and empirically distinct phenomena. Multivariate behavior genetic studies have shown that the moderately strong phenotypic correlation between IQ and standardized academic achievement is almost entirely genetic in origin, but that academic achievement is less heritable and more subject to shared environmental influences than is *g* (Plomin et al., 2001, pp. 199-201). Under the hereditarian hypothesis, we therefore would not necessarily expect Black-White gaps in IQ and standardized academic achievement to be identical. We would, however, expect achievement gaps to fall within a specified range determined by the magnitude of the Black-White IQ gap, in combination with the degree of correlation between IQ and academic achievement. On the other hand, if there are race-specific environmental influences on achievement, then actual achievement gaps might be either larger or smaller than predicted from IQ alone. For example, compensatory educational resources for Blacks but not Whites might produce smaller achievement gaps than predicted by their IQ gap, just as teacher discrimination against Black students might produce ones larger than expected. Tables 1 and 2 summarize the pertinent data.

Table 1 provides standardized mean Black-White differences in IQ for all publicly-available, nationally-representative samples I could locate for the 20<sup>th</sup> century. (Several national samples were excluded because the IQ tests in question were psychometrically weak measures of *g*.) The table reveals no narrowing of the Black-White IQ gap over the 20<sup>th</sup> century. The Black-White IQ gaps have remained at  $1.0 \pm .2$  for both children and adults, and they average 1.02 SDs across the 20 samples. (The average rises and rounds to Rushton and Jensen's 1.1 SDs when I recalculate effect sizes using the White SD, as did Rushton and Jensen, rather than the larger SD for all races combined for the 14 samples in which the White SD was available. )

--- Insert Tables 1 and 2 About Here ---

Table 2 provides standardized mean Black-White differences in achievement on all tests of reading, math, and science in the NAEP time series, 1971-1999. (See Gottfredson, 2003b, for details on these data and their interpretation, and for IQ and achievement data for other races and samples.) It shows that the Black-White gaps in NAEP achievement narrowed during the last thirty years, as the critics note, but mostly in reading. Among 13-year-olds, for example, the Black-White gap in reading narrowed from 1.05 SDs in the 1970s to .73 in the 1990s; in math, from 1.08 to .92; and in science, from 1.10 to 1.09. Averaging results across the three subjects and all three ages, the Black-White achievement gap narrowed from 1.07 SD in the 1970s to .89 in the 1990s, a reduction of 17%. The gaps for Hispanics closed 11% on the average—from .84 to .75 SD (data not shown; see Gottfredson, 2003b). Most of the narrowing in both groups took place by the mid 1980s. The minimum and maximum gaps predicted by *g* theory are, respectively, about .82 and 1.20 SD for Blacks and .62 and .90 for Hispanics (Gottfredson, 2003b). Only three of the 27 decade averages for Blacks (.73; all in reading), and none for Hispanics (not shown), strayed much outside the predicted range. Moreover, the achievement gaps are no longer closing, which suggests that group differences in *g* may set limits on how much narrowing of the achievement gaps is feasible. The NAEP data therefore do not contradict the hereditarian hypothesis.

#### IV. The Totality of Available Evidence: How Compelling?

The final round between the two contenders is “Which theory more consistently and coherently explains the totality of evidence?”

##### Replication

First, how well replicated are the individual threads of evidence that Rushton and Jensen review? As they themselves point out, it varies considerably. The following major facts from Sections 3-6 and 10-12 have been replicated many times, and all with independent sources of data. All are consistent with hereditarian theory but *contradict* culture-only theory.

- worldwide Black-White-East Asian differences in IQ (Section 3), reaction time (Section 4), and brain size, with Whites having the intermediate scores (Section 6);
- an *inverse* correlation between the foregoing race differences in brain attributes and Black-White-East Asian differences in body maturation (Section 6);
- small (.2) and moderate (.4) correlations of IQ, respectively, with skull size and *in vivo* brain volume (Section 6);
- a moderately high correlation (usually .6-.7) of different IQ subtests’ g loadings, not only with the magnitude of Black-White-East Asian mean differences on those subtests (Section 6), but also with measures of those subtests’ rootedness in biological and genetic processes (e.g., heritability; Section 4);
- the rising heritability of IQ with age (within races) and the virtual disappearance by adolescence of any shared environmental effects on IQ (e.g., parental income, education, childrearing practices; Section 5);
- worldwide Black-White-East Asian mean differences in a large suite of biological (e.g., twinning, gestation time, sex ratio at birth) and social (e.g., law abidingness, marital stability) variables, with the three races always in the same rank order (Section 10);



- a genetic divergence (quantitative, not qualitative) of world population (i.e., racial) groups during evolution (Section 11);
- and evidence contradicting culture-only theory's prediction that group differences in cognitive ability should, in essence, track group differences in identifiable cultural practices and socioeconomic advantage (Section 12).

The threads of supporting evidence in Sections 5 (race-common mental architecture) and 9 (regression to the mean) tend to be less well replicated. The most direct individual tests of genetic vs. environmental effects on mental ability—trans-racial adoption (7), racial admixture (8), and behavior genetic modeling of mean group differences (5)—have either been uncommon or fraught with ambiguity. They clearly need to be replicated, as the authors suggest. Being the most direct tests of the hereditarian hypothesis, however, they are also the most politically sensitive to conduct and thus the least likely to be replicated. The more anomalous findings either require replication (e.g., training helped narrow Black African-White gaps on the Raven Matrices in some South African samples) or constitute a paradox for *both* theories (the Flynn Effect).

### Consilience

Second, how well do the two theories explain the total *pattern* of evidence? Does either weave a coherent theoretical fabric? By this criterion, *g* theory is “progressive” but culture-only theory “degenerating” (Lakatos, 1978). The *g*-based hereditarian theory connects *g*-related phenomena at the genetic, physiological, psychometric, and socioeconomic levels to form a coherent pattern that yields novel predictions subsequently confirmed. It is *consilient*. In contrast, culture-only theory has become increasingly tattered over time, patched over by disconnected ad hoc speculation.

*g*-based hereditarian theory. Beginning at the psychometric level, *g* theory has successfully predicted not only when Black-White IQ differences will remain the *same* in magnitude but also when they will *differ* markedly. First the predicted uniformity: Black-White differences are essentially the

same in the West (about 1 SD) across decade, age, and country; they are not substantially or permanently changed by interventions intended to do so (the point of Jensen's 1969 article); this uniformity of gaps extends to three-way comparisons among Blacks, Whites, and East Asians, with East Asians outscoring whites; and there is growing evidence for a four-way contrast, with a 1-SD IQ difference—85 vs. 70—always favoring Western Blacks (who average around 20% White admixture) over Black Africans. Turning to differences in gaps for a given race, *g* theory successfully predicts that gaps are successively larger on *more g*-loaded tests and among children in *higher* social classes (where there is more regression to the mean). The gaps thus contract and expand according to shifts in—not culture—but tasks' cognitive demands and individuals' genetic relatedness.

Next, this systematic patterning of Black-White-East Asian differences in performance can be traced downward from complex IQ tests, to quite elementary cognitive tasks, to biological processes. So far, the three-way race pattern for IQ/*g* differences has been replicated with reaction/inspective time and brain size, both of which are highly heritable and correlated with *g*, as well as with a large collection of purely physical attributes (e.g., twinning). *g* is highly heritable within races and also has replicated metabolic, electrical, and structural correlates in the brain, most of them known to be heritable too (these studies are mostly with whites).

Although Rushton and Jensen do not discuss the fact, the nexus of results for *g* also extends outward into the social realm. For instance, the *g* factor (indeed, the entire hierarchical structure of mental abilities; Gottfredson, 2003b) is the same in all races at all ages yet studied, the most *g*-loaded tests predict school and job performance best (within races), and they predict performance equally well for Blacks and Whites in both the United States and South Africa. These findings have been replicated, but in fewer studies, for other racial-ethnic groups. The *g* nexus goes full circle, from the social back to the genetic, because major life outcomes such as level of earnings, occupation, and education are also moderately heritable (respectively, about 40-50%, 50%, and 60-70%), with half to two-thirds of their

heritability being joint with  $g$  (see Gottfredson, 2002, for a review; studies limited so far to European Whites).

Culture-only theory. One might be able to interpret many of the individual threads of evidence differently, but it is not clear how culture-only theory could coherently reinterpret the entire interconnected web of evidence. In fact, culture-only theory is notable for retreating from its previous failed explanations into ever-less plausible ones. For example, an early claim, plausible at the time, was that Blacks' mental abilities are underestimated because mental tests are biased against them. Research disconfirmed that claim decades ago. Although some culture-only theorists have never relinquished that belief, others began to press more vigorously the claim that any confirmed cognitive deficits among Blacks result from Blacks having suffered more than Whites from deleterious, IQ-depressing cultural conditions.

However, no such factors have been identified in genetically-sensitive research. Virtually all social science claims for the influence of parental rearing or socioeconomic resources on IQ rest on studies that hopelessly found genetic and non-genetic influences (see Scarr, 1997, for a discussion; Phillips, Brooks-Gunn, Duncan, Klebanov, & Crane, 1998, for an example). In fact, behavior genetic research suggests that relatively little if any of the Western Black-White difference in mature IQ could be owing to the *shared* family factors that the culture-only theory has long presumed important (e.g., poverty), because IQ differences *within* a race (in studies that include a broad range of family environments in Western nations) are not permanently affected by environments that siblings share. This does not rule out the possibility that extraordinarily bad shared environments permanently depress IQ, but relatively few children of any race in the West experience such extremes. As the studies of malnourished East Asian adoptees suggest, extreme deprivation of the sort that mankind has always had to deal with (e.g., starvation, infectious disease) seldom permanently impairs cognitive ability to any

substantial degree. It is the far less common, man-made biological insults (radiation poisoning, methyl mercury, etc.) that have the most devastating consequences (Gottfredson, 2003a).

The failure of socioeconomic resources and parenting behavior to have the influence long claimed for them led culture-only theorists to begin stressing more subtle, more race-specific psychological factors as the root cause of group differences in cognitive performance: for example, racism-depressed motivation, racial stress, race-based performance anxiety (“stereotype threat”), and low self-esteem. All are generally posited to result in some manner from White racism and to affect Blacks at all socioeconomic levels (College Board, 1999). However, there is no evidence that any of the factors causes either short- or long-term declines in actual cognitive ability; not all of them (e.g., self-esteem) are lower for Blacks; and none can begin to explain the large array of relevant non-psychological facts, including why the races also differ in brain size and speed (in milliseconds) of performing exceedingly simple cognitive tasks such as recognizing which of several buttons on a console has been illuminated (a reaction time task). Because the American Black-White IQ gap has not narrowed in the century since it was first measured, the psychic injury must also be just as deleterious now as it was during that earlier, more hostile era for Blacks, which seems implausible. Thus, while the proposed psychic insults may temporarily mend some rips in the culture-only theory, they would seem to hold even less promise than the failed socioeconomic ones for explaining the longstanding, worldwide pattern of racial IQ differences and their links to the biological correlates of *g*. The newly-popular assertion that races “don’t exist” is a straw man (no one believes that racial groups are biologically distinct entities) that does nothing to nullify the evidence it would have us ignore.

In summary, Rushton and Jensen have made a compelling case that their 50-50 hereditarian hypothesis is more plausible than the culture-only hypothesis. In fact, the evidence is so consistent and so quantitatively uniform that the truth may lie closer to 70-80% genetic, which is the within-race heritability for adults in the West. The case for culture-only theory is so weak by comparison—so

“degenerated” —that the burden of proof now shifts to its proponents to identify and replicate even *one* substantial, demonstrably *non*-genetic influence on the Black-White mean difference in *g*. Any such demonstration must be genetically sensitive, because most “environments” are partly genetic in origin (different genotypes create and evoke different environments for themselves and their children; Plomin et al., 2001; Scarr, 1997).

#### V. The Authors’ Policy Recommendations: Are They Warranted?

The authors make no recommendations for specific policies, correctly arguing that the hereditarian hypothesis implies none in particular. Social policy is meant to satisfy particular goals and moral precepts, such as that all citizens have equal rights before the law, none should starve while others feast, and government should maintain the peace and safety. Most people would hold strongly to these values whether or not nature churns us out from different molds. Science tells us what *is*, sometimes what *could be*, but never what *should be* (what is fair and just). Proof that the Black-White IQ gap is partly genetic could, depending on one’s goals, therefore be used to justify banning all racial preferences in employment and college admissions or, from a Rawlsian perspective (that natural differences are unfair), require large and permanent racial preferences (cf. Rowe, 1997).

As Rushton and Jensen suggest, *g* theory can predict fairly well how large the racial disparities in achievement will be in different settings, depending on their demands for *g* and the IQ distributions of the groups involved. It can also provide the menu of tradeoffs between parity and aggregate levels of performance under different scenarios for selecting individuals into those settings, and also predict the likely pattern of effects and side effects, by race, of different interventions in education and training (e.g., see Gottfredson, 2000; Sackett, Schmitt, Ellingson, & Kabin, 2001; Schmidt, Rogers, Chan, Sheppard, & Jennings, 1997). In short, it can detail the challenge before us, and the likely costs and benefits of opting for different goals or means of achieving them—the “could be’s” that we might choose among.

Currently, racial parity in outcomes is often treated as the ultimate standard for fairness and lack of parity as a measure of White racism. This standard for non-discrimination is, in fact, the legal or political law of the land in many matters today. For instance, disparate impact in hiring is *prima facie* evidence of illegal discrimination in the United States, with employers then needing to prove themselves innocent (Sharf, 1988). By undermining culture-only explanations of racial inequality, Rushton and Jensen's "provisional truth" thereby undermines the moral legitimacy of all rationales for racial equalization that posit White misbehavior as the its cause. This is surely what inflames critics most, that the public might be persuaded by the hereditarian hypothesis to temper or abandon its efforts to eliminate racial differences in success and well-being.

The authors themselves acknowledge that open discussion of genotypic ability differences between the races might harm race relations. Their most vocal critics predict far worse. Widespread acceptance of the hereditarian hypothesis would, they say, put us on the slippery slope to racial oppression or genocide. They do not explain how this would happen, but usually imply that because the Nazis were hereditarians, hereditarians must be Nazis at heart. But we can no more presume this than that IQ-environmentalists are Communists because the Communists were IQ-environmentalists. One might note, in addition, that regimes with environmentalist ideologies (Stalin and Pol Pot) exterminated as many of their citizens as did the Nazis (Courtois, 1999), and virtually all the victim groups of genocide in the Twentieth Century had relatively *high* average levels of achievement (e.g., German Jews, educated Cambodians, Russian Kulaks, Armenians in Turkey, Ibos in Nigeria; Gordon, 1980). The critics' predictions of mass moral madness, like the frequent demonization of scientists who report unwelcome racial differences, seem mostly an attempt to shut off reasoned discussion.

These horrific scenarios are obviously implausible, but might society be better off not knowing the truth? "For this kind of truth, ... what good will come of it?" (Glazer, 1994, p. 16). Summing up his argument against candor with regard to either phenotypic or genotypic differences, Glazer states that:

Our society, our polity, our elites, according to Herrnstein and Murray, live with an untruth: that there is no good reason for this [racial] inequality, and therefore society is at fault and we must try harder. I ask myself whether the untruth is not better for American society than the truth.

But we must also ask ourselves the converse: “What harm might the untruth cause?” Should we really presume that denying the existence of average racial differences in *g* has only benefits and the truth only costs? Lying about the enduring Black-White difference in phenotypic *g* would seem to be both futile and harmful in the long run. It is futile because the truth—and attempts to suppress it—will become increasingly obvious to the average person. Phenotypic differences in cognitive ability have real-world effects that are neither ameliorated nor hidden by claims to the contrary. They manifest themselves relentlessly. Bigger disparities have more obvious effects in day-to-day encounters, especially in the more cognitively demanding arenas of life, such as school and mid- to high-level jobs. Blacks and Whites both span the full range of intelligence, but the more representative they are of their groups when in moderately to highly *g*-loaded settings, the more likely there will be noticeable racial differences in performance. This would especially be the case, for example, in integrated public schools that do not group students by ability level, and in jobs or educational settings where Blacks and Whites have been selected under notably different standards. Even culture-only theorists are becoming unsettled by the stubborn persistence of large achievement gaps in the most advantaged and anti-racist of school systems (Lee, 2002; Ogbu, 2003).

Lying about race differences in achievement is harmful because it foments mutual recrimination. Because the untruth insists that differences cannot be natural, they must be artificial, man-made, manufactured. *Someone* must be at fault. Someone must be *refusing* to do the right thing. It therefore sustains unwarranted, divisive, ever-escalating mutual accusations of moral culpability (Whites are racist or Blacks are lazy).

## VI. Does the Hereditarian Hypothesis Leave Us Without Hope?

Given what we know about  $g$  itself, Black-White genetic differences in  $g$  would render the goal of full parity in either IQ or achievement unrealistic. This does not, however, rule out the possibility of *reducing* the disparities, especially in achievement. Rushton and Jensen offer no suggestions for doing so, perhaps partly because they are resigned to living with racial inequality, but also because it is difficult to know what would materially narrow gaps in IQ and achievement. In theory, there are at least two points of intervention: altering the effective influences on  $g$ , and altering  $g$ 's practical consequences in school and elsewhere.

With regard to changing  $g$  itself, it is still possible that some part of the Black-White difference is caused by extremely bad environments (of the shared family variety) in the most severely disadvantaged segments of the Black population. Such effects, should they exist, would not account for the greater part of the mean Black-White IQ difference, but their remediation would help reduce it. That leaves genetic and non-shared environmental effects. It is not clear why non-shared effects would hurt Blacks more than Whites, because they influence individuals uniquely, one at a time, and not family by family and thus presumably not race by race either. Moreover, non-shared influences may consist primarily of small random effects of a non-genetic biological nature (e.g., illness; Jensen, 1997), and hence be essentially uncontrollable. Many people have seized upon the Flynn Effect as holding the key to narrowing the Black-White IQ gap. However, the cause of this mysterious secular increase in IQ remains unknown. And having done nothing to change the Black-White gap over the last century, it seems unlikely to hold the key for eliminating it. Ironically, genetic effects may turn out to be the most alterable source of race differences in mean IQ/ $g$ . Genes do not magically “stamp in” any particular level of  $g$ , but code for the hormones, neurotransmitters, and other physiological and structural factors that affect our cognitive functioning and our reaction to environments. There is already a race underway to find “smart drugs” and gene therapies that could improve intelligence.



Even should IQ gaps remain intractable, there is no reason to “give up” on anyone or presume that some people “can’t learn.” All people can learn, though at different rates (that being the nub of the problem). Rather, it means we must reconsider the means by which we try to help people and be more realistic about how much impact we will have. Ability and achievement are distinct phenomena, and that the latter is more sensitive to environmental influence, including the shared variety. There thus are environmental factors independent of *g*—ones that we might conceivably harness—that influence performance on even the most *g* loaded of life’s tasks. Such factors might include quality of instruction and social support, for instance. However, the fact that the covariance matrices for academic achievement are the same for both Blacks and Whites, as Rushton and Jensen note for research to date, suggests that there are no *race-specific* factors affecting the relation between *g* and academic achievement. It should also be noted that interventions that help all people to better meet their (different) potentials will often widen achievement gaps because they tend to *increase* the variance in achievement both within and between races (the mentally fast advance further than the slow).

Instead of attempting to equalize the races, it might be better to help lower-IQ individuals of all races. This would meet especially pressing human needs while narrowing some racial gaps (e.g., in developing basic skills, finishing high school, getting and keeping a job, staying healthy). The weaker learning and problem solving abilities of people in the lower part of the IQ distribution make their daily lives much more difficult and hazardous, and stack the odds against them at every step along the path to educational and occupational success (Gottfredson, 1997d). A great personality, persistence, and experience help to compensate for lower *g*, but only somewhat, as personnel psychologists have repeatedly documented in the workplace.

We might therefore target individuals below IQ 80 for special support, intellectual as well as material. This is the cognitive ability level below which federal law prohibits induction into the American military (for lack of trainability) and below which no civilian jobs in the United States

routinely recruit their workers. It includes about 10% of Whites and a third of Blacks in the United States and the segment of both groups most at risk for multiple health and social problems, regardless of family background and material resources (Gottfredson, 2002, in press a; Gottfredson & Deary, in press; Murray, 1998). The risks that lower-IQ people face relative to more able individuals have, in addition, been growing as the complexity of work, health care, and daily life has increased. g theory suggests that their relative risk might be lowered in at least three ways: (a) that education and training be better targeted to their learning needs (instruction is more narrowly focused, non-theoretical, concrete, hands-on, requiring no inferences, repetitive, and personalized), (b) that they be provided more assistance and direct instruction in matters of daily well-being that we expect most people pick up on their own (e.g., learning how best to avoid various kinds of illness and injury), and (c) that health care providers, social service agencies, and other institutions remove some of the unnecessary complexity (e.g., inadequate or overly complex labeling, instructions, and forms) that often impedes full and effective use of services, medical regimens, and preventive care by the less able. Less favorable genes for g impose constraints on individuals and the people who wish to help them, but they certainly do not prevent us from improving lives in crucial ways.

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Table 1

Hereditarian Hypothesis

Standardized Black-White Differences in Mean IQ ( $d_{IQ}$ ) in National Samples, By Age, 1917-1994

Year <sup>a</sup> /Ages	White		Black		$d_{IQ}^b$
	Mean	SD	Mean	SD	
Armed Services Aptitude Batteries					
1917-1918 (WWI) data: Recruits <sup>c</sup>					<b>1.16</b>
1944-45 (WWII) data: AGCT for recruits <sup>d</sup>					<b>1.25</b>
1980 data: AFQT standardization sample (NLSY) <sup>e</sup>					
18-23	522	86.9	401	94.5	<b>1.21</b>
Coleman et al. (1966) Equality of Educational Opportunity Report <sup>f</sup>					
1965 data					
1st grade	53.6		44.4		<b>.92</b>
12th grade	52.1		40.9		<b>1.12</b>
National Longitudinal Study (NLS) of High School Class of 1972 <sup>g</sup>					
1972 data					
12th grade	208	31	169	28	<b>1.16</b>
WISC-R Standardization Sample <sup>h</sup>					
1974					
6-16.5	102.3	14.1	86.4	12.8	<b>1.06</b>
WAIS-R Standardization Sample <sup>i</sup>					
1981					
16-19	100.8	14.1	86.9	14.5	<b>.93</b>
20-34	101.8	15.1	87.0	11.6	<b>.99</b>
35-54	101.4	14.8	86.6	13.2	<b>.99</b>
55-74	101.4	14.6	87.0	13.0	<b>.96</b>
DAS Standardization Sample <sup>j</sup>					
1986 data					
2.6-3.5	103.0	14.1	91.5	13.4	<b>.77</b>
3.6-5.11	103.6	13.7	86.7	13.3	<b>1.13</b>
6-17	102.7	14.3	89.2	14.1	<b>.90</b>
Stanford-Binet IV Standardization Sample <sup>k</sup>					
1986					
2-6	104.7	14.7	91.0	13.2	<b>.86</b>
7-11	102.6	15.6	92.7	13.2	<b>.62</b>
12 to 18-23	103.5	15.8	86.1	15.1	<b>1.09</b>
WISC-III Standardization Sample <sup>l</sup>					
1991					
6-16	103.5		88.6		<b>.99</b>
PPVT-R: Children of National Longitudinal Study of Youth (NLSY) Mothers					
1986-1994 data					
3-4 <sup>m</sup>	52		40		<b>1.20</b>
5-6 <sup>n</sup>	98.9	15.2	81.9	14.6	<b>1.13</b>

<sup>a</sup>Except where otherwise specified, "year" refers to year of publication and not year of data collection. First two, last two, and 12th-grade samples are not fully nationally representative of their age groups.

<sup>b</sup>Effect size is calculated here as the group mean difference (e.g., W-B) divided by the total SD (including all racial-ethnic groups) for the battery in question. I note when *d*s are based on medians rather than means. "Total" SDs are provided in the footnotes.

<sup>c</sup>Source: Loehlin et al. (1975, pp. 143, 408-409). Based on a variety of tests (Army Alpha, etc.) put on a common scale.

<sup>d</sup>Source: Jensen (1998, p. 376).

<sup>e</sup>AFQT=Armed Forces Qualifying Test. Source: Laurence, Eitelberg, & Waters (1982, p. 43). Mean=500, SD=100. Same sample as used in National Longitudinal Survey of Youth (NLSY; e.g., Herrnstein & Murray, 1994). "White" includes all racial-ethnic groups other than blacks and Hispanics.

<sup>f</sup>Coleman et al. (1966, p. 20). Mean=50, SD=10; effect sizes based on medians, not means. These are the mean of verbal and non-verbal scores.

<sup>g</sup>Source: Osborne (1982, p. 260). IQ="ability index," which is sum of NLS tests of vocabulary, reading, comprehension, mathematics, and letter groups (inductive reasoning).

<sup>h</sup>WISC-R=Wechsler Intelligence Scale for Children-Revised. Source: Jensen & Reynolds (1982, p. 425). Mean=100, SD=15.

<sup>i</sup>WAIS-R=Wechsler Adult Intelligence Scale-Revised. Source: Reynolds, Chastain, Kaufman, & McLean (1987, p. 330). Mean=100, SD=15.

<sup>j</sup>DAS=Differential Ability Scales. Source: Lynn (1996, p. 272).

<sup>k</sup>Source: Thorndike, Hagen, & Sattler (1986, pp. 34-36). Mean=100, SD=16.

<sup>l</sup>Source: Sattler (2001, p. 232). Mean=100, SD=15.

<sup>m</sup>PPVT-R=Peabody Picture Vocabulary Test-Revised. Source: Jencks & Phillips (1998, p. 2). Mean=50, SD=10; effect sizes based on differences in medians, not means.

<sup>n</sup>Source: Phillips, Brooks-Dunn, Duncan, Klebanov, & Crane (1998, p. 108). Mean=100, SD=15. Children of young mothers are overrepresented.

Table 2

Standardized Black-White Differences in Mean Achievement ( $d_{ach}$ ) on NAEP Tests of Reading, Math, and Science, Ages 9, 13, and 17 in 1971-1999

Year	$d_{ach}^a$								
	Reading			Math			Science		
	Age: 9	13	17	9	13	17	9	13	17
1971	1.04	1.08	1.15						
1975	.92	1.02	1.19						
1977							1.22	1.10	1.23
1978				.88	1.08	1.07			
1980	.84	.91	1.19						
1982				.84	1.02	.98	1.03	1.04	1.25
1984	.79	.74	.79						
1986				.74	.79	.93	.86	1.03	1.01
1988	.71	.53	.55						
1990	.79	.58	.71	.81	.87	.68	1.02	1.02	1.04
1992	.83	.73	.86	.82	.93	.87	.97	1.16	1.07
1994	.80	.77	.66	.74	.90	.89	.95	1.15	1.08
1996	.74	.82	.69	.75	.92	.89	.88	1.05	1.03
1999	.91	.74	.73	.82	.98	1.02	1.02	1.06	1.18
Mean for decades									
1970s	.98	1.05	1.17	.88	1.08	1.07	1.22	1.10	1.23
1980s	.78	.73	.84	.79	.91	.96	.95	1.03	1.13
1990s	.81	.73	.73	.79	.92	.87	.97	1.09	1.08

<sup>a</sup>Effect sizes calculated with SDs for entire national sample of students that age in that year taking that test. Calculated from data in National Center for Education Statistics (2000).