

What if the Hereditarian Hypothesis Is True?

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Abstract

Rushton and Jensen (this issue) make a strong empirical case 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 also examines whether their data warrant the implications they draw for public policy, and whether critics are correct that sometimes it is better to tell untruths than truths.

What if the Hereditarian Hypothesis Is True?

On December 7, 2002, at the annual meeting of the International Society for Intelligence Research (see Holden, 2003, in *Science*), Professor Rushton gave a talk summarizing the data reported in the paper at hand: “Thirty Years of Research on Black-White Differences in Cognitive Ability” (Rushton & Jensen, this issue). None of the persons present questioned the credibility of any of the data he presented.

Why did attendees not question those data, for example, that brain size is correlated with both intelligence and race? Because they _____.

- a. did not know enough about the data to comment on it
- b. want a scientific pretext for claiming that Blacks are genetically less able
- c. were embarrassed and just wanted Rushton to finish and leave the podium
- d. thought it was old news of already well-established validity

Rushton interpreted the data as supporting his and Jensen’s hypothesis that there is a genetic component to the average Black-White difference in cognitive ability. One member of the audience suggested that the data could be interpreted differently and therefore that the hypothesis remains “not proved.” No one else disputed Rushton’s interpretation.

Why did the attendees not take more issue with Rushton’s interpretation? Because they _____.

- a. are closet racists
- b. are pseudoscientists with elitist, anti-Black political agendas
- c. are mean-spirited and don’t care if they hurt the downtrodden
- d. find the partly-genetic hypothesis increasingly plausible as more data accumulate

I cannot speak for all the attendees, but I suspect that a remark to me by one of them captured the assessment of many: “But we knew all that. I expected him to report some new data.” This is a crowd of die-hard empiricists, far more likely over dinner to discuss the nuances of data analysis than public

policy. And I have never heard a bigoted word from any of them, some of whom I have known for decades. Answers a-c are definitely wrong.

But why should we think that answer d is correct? What, in fact, is required to make Rushton and Jensen's hereditarian case plausible? And even if it were confirmed, what policy recommendations would follow, if any?

1. Does there exist a prerequisite evidentiary base for g itself?

At a minimum, a plausible case would require proof of three facts about *g* itself. First, individual differences in general mental ability (*g*) are real and can be measured well. Second, they are highly heritable by adulthood. Third, average *group* differences are real and can be measured well (without bias). The first two facts had all been confirmed for Western populations (though not widely accepted) before Jensen first broached the hereditarian hypothesis three decades ago, and the third fact about group differences was confirmed (though not widely accepted) about a decade later. Various blue-ribbon national committees have since put their stamp of credibility on all three (e.g., Hartigan & Wigdor, 1989; Neisser et al., 1996; Wigdor & Garner, 1982; Wigdor & Green, 1991). The authors largely take this evidentiary base for granted in order to focus on the fourth potential fact, the still hotly contested hypothesis that group differences in *g* are partly genetic.

2. Have the authors considered the most pertinent sorts of evidence for testing their "hereditarian" hypothesis that the mean Black-White difference in g is 50% genetic?

Rushton and Jensen review the research that laypeople usually single out as obviously relevant—cross-racial adoptions and studies of racial admixture. However, these sorts of natural experiments have been plagued by various problems (unknown IQ-selectivity of the individuals involved, etc.) that, as the authors note, often render their meaning ambiguous. They suggest testable hypotheses for resolving the ambiguities (e.g., that the early enhanced IQ levels of Black and mixed-race children adopted into enriched environments will have faded away, as hereditarian theory predicts, when they are retested in adolescence). The authors also suggest that genomic studies would provide a better method of

ascertaining racial admixture and would therefore provide better evidence for testing their claim that greater White admixture will yield IQ levels closer to the average for Whites.

Molecular genetic studies are, in fact, often thought to promise more definitive evidence, one way or the other, about whether the races differ in their distribution of alleles affecting intelligence. While there is progress in genomically distinguishing different population groups, as the authors note (see also Wade, 2002), none of the scores or more of genes affecting normal intelligence has yet been definitely identified. (Many single-gene causes of mental retardation have, however, been discovered.) Evidence of this sort regarding the potential heritability of group differences in *g* may therefore be a long while coming, even should molecular geneticists become willing to investigate the question.

A fairly direct way to test the authors' 50%-genetic-50%-environmental hypothesis is to statistically model between-group differences together with within-group individual differences in IQ. Behavior geneticists have recently developed statistical modeling procedures to estimate the genetic component of mean group (e.g., sex) differences, but, for political reasons, those procedures are rarely applied to mean differences between racial-ethnic groups. Existing studies, which the authors cite, support the 50-50 hypothesis, but there are too few yet to stand alone as persuasive evidence for it.

Rushton and Jensen's argument therefore rests primarily on setting up a contest between their 50-50 *hereditarian* theory and its major rival, what they call *culture-only* (zero percent genetic) theory. (Here they are using the word culture in its broad sense to refer to the whole non-biological environment.) That is, they look at the relative success of the two theories in explaining the *totality* of evidence on race and cognitive ability. This is actually a demanding test, because it requires that any theory fit a *complex pattern* of disparate findings. Well-replicated findings that contradict some core element of a theory will disconfirm that portion of it and raise doubts about the remainder as well. A theory gains some support when empirical findings are consistent with its various distinct elements, but it gains far more when it makes novel predictions—ones that competing theories would not make—that are subsequently confirmed. The thicker, broader, and more enmeshed the network of validated threads

in the theory, the more persuasive the whole becomes. Their strategy, then, is to look at the whole fabric of each theory. How extensive and thickly connected is its validated portion? Does it have gaping holes, especially at its core, burned through by round after round of contrary evidence? How much of the fabric consists of disconnected, ad hoc patchwork to cover up such holes and previous failed patches? Has one of the two competing theories become stronger but the other more tattered over the last 30 years? In Lakatos' (1978) terms, is one *progressive* and the other *degenerating*?

The body of relevant research from which Rushton and Jensen draw has grown enormously in the recent decades, forming an ever broader and denser nomological network for *g*. It is this expanded network that allows them to make novel predictions about race differences as well as to test more effectively prior predictions from both the hereditarian and culture-only theories. Much more is now known, for instance, about *g*'s physiological correlates in the brain, its socioeconomic correlates in people's lives, and its *genetic* correlations with these putative causes and consequences. Two fundamental advances have been to validate the non-psychometric reality and cross-cultural generality of the *g* factor itself and to characterize mental tests and tasks in terms of the degree to which they call it forth (to quantify their "g loadings").

3. *How compelling is the authors' evidence?*

Rushton and Jensen summarize 10 different subsets of research to support their hereditarian hypothesis. As they themselves point out, some of these subsets are less compelling than others. Critics could no doubt ferret out real or imagined flaws in all the individual studies, allowing them to reject, in turn, each one of the many the authors cite. However, the scientific method requires not perfection, which is neither necessary nor reasonable, but *replication*, preferably *independent* replication. How do the 10 subsets (Sections 3-12) fare by this criterion? 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 *not* with 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 of the foregoing brain-related racial differences with 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 (their ability to measure *g*), with not only the magnitude of Black-White-Asian mean differences on those subtests (Section 6), but also measures of those subtests' rootedness in biological and genetic processes (e.g., heritability, susceptibility to inbreeding depression; Section 4), which together indicate that the average racial differences in IQ are mostly differences in biological *g*;
- 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-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 the culture-only theory's prediction that group differences in cognitive ability should, in essence, track group differences in cultural practices and socioeconomic advantage (Section 12).

Most of the foregoing facts have been replicated in enough age groups, racial-ethnic groups, countries, and time periods, and by enough different methods and investigators, that they can be considered highly *generalizable*. For instance, the same pattern of Black-White-East Asian differences

in IQ, reaction time, brain size, and (for children) physical development—and these variables' relation to *g*—is found around the world, at different development ages, and with different methods of measurement.

Most of the individual forms of evidence in Sections 5 and 9 are less well replicated, but they gain strength by being convergent. They all (e.g., similar heritabilities, equal developmental input-outcome covariance matrices) point to the same conclusion, namely, that the gene-environment architecture of *g* is the same for Blacks and Whites (Section 5). Cognitive ability is “built” and affects the organism in the same manner in both races, with no evidence of any race-specific processes. There are thus no differences in the production of *individual variation within the two races* that could account for their *mean differences* in IQ. When Black and White children are matched on IQ, the finding that their siblings regress halfway to their respective racial means (IQ 85 for Blacks and IQ 100 for Whites) is particularly solid evidence, because the prediction was novel and the result cannot be explained by culture-only theory (Section 9).

As noted earlier, the most direct individual tests of genetic vs. environmental effects on mental ability—transracial adoption (Section 7), racial admixture (Section 8), and behavior genetic modeling of mean group differences (Section 5)—have been either uncommon or fraught with ambiguity. Such tests 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 authors quantify the outcome of the contest they have set up between the two theories by tallying scores across each of the 10 subsets of data. I might adjust their tallies somewhat, but what most strongly favors their theory, in my mind, is the coherence or *consilience* of the data supporting it. The evidence meshes together; it converges from diverse directions; the interlocking whole is more than the sum of its parts.

In contrast, culture-only theory has accumulated a long series of failed predictions about the presumed IQ-depressing effects of poverty, low social class, and cultural isolation: none of the social and educational programs that the theory predicted would permanently raise low IQs (e.g., school integration, preschool interventions) has done so. (The stated aims for these two social programs have accordingly shifted over the decades from being intellectual to mostly social-moral.) In addition, the Black-White IQ gap did not narrow at all during the Twentieth Century, remaining at 1.1 standard deviations despite considerable narrowing of racial disparities in education, material advantage, civil rights, and political participation.

Moreover, the facts have often turned out to be the *opposite* of what culture-only theory had predicted. To cite a few:

- American Blacks score relatively better on verbal than non-verbal tests, and better on culture-loaded than culture-reduced ones;
- Black-White differences are largest on the most, not the least, heritable tests;
- American Blacks have lower average IQs than Hispanic American and Native American groups that are more socioeconomically deprived;
- East Asians score better than Whites, worldwide, on putatively “White” IQ tests, even when they live in worse socioeconomic circumstances;
- even severely malnourished East Asian children adopted into White homes develop higher IQs than do White children;
- races that lag in mental development are superior in physical development;
- shared family effects on IQ dissipate, not accumulate, with age;
- the IQs of biological siblings reared together become less alike with age, while those reared apart become more alike (both types converging on a correlation of .5).

The culture-only theory has changed much over the decades in response to its failed predictions but become less plausible in the process. An early claim, plausible at the time, was that Black IQs are

underestimated because mental tests are biased against Blacks. Research disconfirmed that claim decades ago, as noted earlier. Culture-only theorists thus emphasize all the more their allegation that any real cognitive deficits among Blacks result from Blacks suffering more than Whites from deleterious, IQ-depressing environmental factors. As Rushton and Jensen describe, however, those environmental factors cannot plausibly be *shared*-family factors of the sort that the culture-only theory has long presumed important (e.g., poverty). The reason is that behavior genetic studies have shown (in studies that include a broad range of family environments in Western nations) that IQ differences *within* a race are not permanently affected by shared aspects of family environments. This does not rule out the possibility that extraordinarily bad environments permanently depress IQ, but only a small subset of children in the United States or other Western countries would experience such extremes. In fact, 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. The most devastating, most permanent damage to cognitive ability is caused by modern, man-made biological insults, such as radiation and chemical poisons, especially when experienced prenatally (Gottfredson, in press). Such insults tend, however, to afflict only relatively small pockets of the world's population.

The fact that shared environments, at least within developed nations today, seem to have no lasting effects on IQ does not invalidate all potential forms of culture-only theory, but it does require that the environmental mechanisms the theory posits to explain the average Black-White IQ difference be fundamentally different than whatever creates differences *within* races. That is, there must be a Factor X that suppresses the IQs of all Blacks but no Whites, and a Factor Y that enhances the IQs of all East Asians but no Whites. These race-specific factors must also (a) affect all individuals within a race equally (because otherwise the races' covariance matrices would differ, which they do not in the studies conducted thus far), (b) affect all ages in just the same way (because the Black-White IQ gap does not change after age three, the age at which IQ tests can first effectively measure *g*), and (c) remain equally

potent through periods of great social change (because the average Black-White difference has not changed since it was first measured almost a century ago). This uniformity and stability in mean racial differences, worldwide as well as within the United States, is not consistent with the variability and instability in the cultural and socioeconomic advantage over time and place by which culture-only theory would explain those mean racial differences in IQ.

Because differences in material resources across a broad range of family circumstances seem to have no demonstrable effect on IQ by late adolescence, culture-only theories have now begun to stress psychological rather than material disadvantage 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. However, there is no evidence that any of them causes either short- or long-term declines in actual cognitive ability, either within or between races; 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 patch over 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*.

We might ask, finally, whether the authors have given us a full and fair accounting of the evidence most pertinent to their hypothesis. Based on my knowledge of intelligence research, Rushton and Jensen have exercised due diligence in reporting the most pertinent evidence from the field and, very importantly, reporting both that which contradicts and supports their thesis. Perhaps others will

know of *directly pertinent* research that I am not aware of. My experience is that the “neglected” research to which critics point is usually either irrelevant or has already been taken into account by the authors.

In summary, the authors have made a strong 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 Jensen’s default hypothesis, that is, that the heritability of within-group and between-group differences in *g* is the same (80%). The case for culture-only theory is so weak by comparison—so “degenerated”—that the burden of proof now shifts to its proponents to convincingly identify and replicate even one substantial, demonstrably *non-genetic* influence on the Black-White mean difference in *g*, let alone more than 50% of it. Or to propose—and test—a novel prediction that would support culture-only theory while contradicting hereditarian theory.

4. *Are the authors’ policy recommendations warranted?*

The authors make no recommendations for specific policy positions, arguing that their evidence implies none in particular. That is true, because 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 eat, and government should maintain the peace and safety. Most Americans 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 *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 college admissions and hiring or, from a Rawlsian perspective (that natural differences are unfair), require that there be large and permanent racial preferences.

The authors do, however, argue for one particular moral principle for which they say there is an “ethical consensus:” namely, we should treat people as individuals and not as members of groups. Some might dispute that there is such a consensus, because the notion of group rights has been gaining ground in the United States. But I think it is fair to say that culture-only theorists have traditionally argued from

an individual-rights perspective too. They emphasize that Blacks have suffered because Whites have discriminated against them, first overtly and now covertly. Blacks would prosper to the same degree as Whites, they argue, but for this violation of individual rights. The theory's adherents may have often advocated racial preferences, but as a temporary expedient for ensuring the individual rights of Black persons in the face of seeming White intransigence. Critics may wish to dispute Rushton and Jensen's preference for individual over group rights, but they should make their own preferences explicit.

A consequence of adhering to individual rights, *when individuals and races (on average) are not equally able*, is that not all individuals and groups will succeed equally in life. The reason is that *g* is a highly general ability with enormous practical value, especially in school and work settings (Gottfredson, 2002a). That is why Rushton and Jensen argue that the public must learn to live constructively with average racial disparities in achievement.

The evidence that Rushton and Jensen summarize does not dictate that we accept racial inequality, but it does show why we have to choose between two deeply held but conflicting goals—racial parity and individual rights—that we once assumed consistent. The size of the mean racial difference is such that attaining racial parity in education and employment would require adopting very different performance standards for members of the two races or, alternatively, lowering standards dramatically for members of both (Gottfredson, 2000). On the other hand, letting the chips fall where they may under individualism yields rather glaring levels of racial imbalance—in legal terms, *disparate impact*. This is a well-known, much-analyzed problem in personnel selection (e.g., Sackett, Schmidt, Ellingson, & Kabin, 2001; Schmitt et al., 1997) and social policy in general (Gottfredson, 1997). While we might fervently wish it away, there is a substantial tradeoff between racial parity, on the one hand, and aggregate productivity and individual rights, on the other.

Although this tradeoff exists whether or not its cause is genetic, how we explain it will influence how we deal with it. And this is precisely what Rushton and Jensen point to as their work's key implication: if racial disparities in *g* and achievement stem partly from genetic differences, then we must

reevaluate racial parity as the standard for fairness and its absence as a measure of White racism. This standard 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, hereditarian theory weakens the moral rationale for racial equalization policies, such as racial preferences, that posit White racism as its sole cause.

Although Rushton and Jensen do not propose any particular policies to address the dilemma, they are clearly questioning the taken-for-granted moral basis for many current ones. They cause us to wonder whether racial parity should be such a preeminent goal, and whether there should be more reasonable limits on its pursuit. 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. For many critics, the hereditarian hypothesis is not just unwarranted, but dangerous.

Thoughtful people may disagree, but the “implication” to which Rushton and Jensen point seems both warranted and obvious. Moreover, as they suggest, *g* theory can predict fairly well just how big 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. 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.

But might we do damage by openly analyzing the dilemmas posed by phenotypic—or especially genotypic—differences between races? Some social scientists think so. The most vocal among them suggest that it would 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. But we can no more presume this, of course, than that environmentalists are Communists because the Communists were environmentalists. One might note, in addition, that regimes with environmentalist ideologies (Stalin and Pol Pot) exterminated more of their citizens than did the Nazis, 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, may be mostly an attempt to shut off reasoned discussion.

These scenarios of horror are obviously implausible, but aren't there real risks associated with widespread acceptance of the hereditarian hypothesis? Rushton and Jensen themselves suggest that it might worsen race relations. Just this sort of side effect has also prompted other social scientists to argue that society would 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, Glazer says:

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. (Glazer, 1994, p. 16).

We must also ask the converse, however: "What harm might the untruth cause?" Should we really presume that denying average racial differences in *g* has only benefits and the truth only costs? Lying about the average 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. 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 *g*-loaded 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 of their groups they are 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 by ability, 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 the “achievement gap” in even the most advantaged and non-racist of school systems (Lee, 2002). How people interpret the performance differences they observe is another matter. Because they have been exhorted for so long to believe that Blacks and Whites do not differ in cognitive ability, many may attribute the differences in achievement to Black deficits in motivation or other character traits.

Enforcing the egalitarian untruth also misdirects energies and resources into failed ventures. Because the untruth insists 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, even escalating mutual accusations of moral culpability (Whites are racist or Blacks are lazy). In an effort to sustain the untruth, wittingly or not, critics of the hereditarian hypothesis routinely tell us that to believe Blacks are lower in cognitive ability for partly genetic reasons is to believe that Blacks are “inferior.” This rhetoric is regrettable, because it primes people to do just that, to view Blacks as morally inferior should the hereditarian hypothesis be confirmed. As advantageous as *g* is in practical affairs, high *g* is only a tool, not a sign of moral superiority, and critics ought not suggest otherwise.

In short, we must entertain the hereditarian hypothesis, but in a constructive manner. This is what Rushton and Jensen seem to mean when they speak of the need to educate the public about the genetic differences among us and to dispel likely misperceptions. A first item on that list would be that an individual’s race is no measure at all of his or her cognitive ability because all races span the full range of *g*.

5. Does the hereditarian hypothesis leave us without hope?

By this, people usually mean hope of achieving racial parity in life achievements. 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 the achievement gaps. In theory, there are at least two general points of intervention involving *g*: altering the effective influences on *g*, and altering its practical consequences. To narrow the gaps, however, any intervention would have to favor Blacks more than Whites.

With regard to the first, 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 the average racial difference. 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. There is already a race underway to find “smart drugs” and gene therapies that could improve

intelligence. Should they be found, we might be able to raise low IQs and enhance normal ones, but the Black-White IQ gap would shrink only if they were more available to Blacks than Whites.

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. *g* theory makes it clear that ability and achievement are distinct phenomena, and that the latter does not invariably mirror the former. For example, behavior genetic research has shown that the moderately strong phenotypic correlation between *g* and academic achievement is almost entirely genetic in origin, but that academic achievement is less heritable and more subject to environmental influence than is *g*. 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. This means that Blacks would have to benefit more than Whites from manipulating these factors in order to shrink the achievement gaps. It should 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 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 half 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. A great personality, persistence, and experience help to

compensate for lower g , but only somewhat, as personnel psychologists have 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 that is most at risk for multiple health and social problems, regardless of their family background and material resources (Gottfredson, 2002b; Murray, 1998). The risks that lower-IQ people face relative to more able individuals have, in addition, been growing as the complexity of work 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. Genetic differences in g clearly impose constraints on individuals and the people who wish to help them, but they hardly prevent us from improving lives in meaningful ways.

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