

# Genetic Differences and School Readiness

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The author considers whether differences in genetic endowment may account for racial and ethnic differences in school readiness. While acknowledging an important role for genes in explaining differences *within* races, he nevertheless argues that environment explains most of the gap *between* blacks and whites, leaving little role for genetics.

Based on a wide range of direct and indirect evidence, particularly work by Klaus Eyferth and James Flynn, the author concludes that the black-white gap is not substantially genetic in origin. In studies in 1959 and 1961, Eyferth first pointed to the near-disappearance of the black-white gap among children of black and white servicemen raised by German mothers after World War II. In the author's view, Flynn's exhaustive 1980 analysis of Eyferth's work provides close to definitive evidence that the black disadvantage is not genetic to any important degree.

But even studies showing an important role for genes in explaining within-group differences, he says, do not rule out the possibility of improving the school performance of disadvantaged children through interventions aimed at improving their school readiness. Such interventions, he argues, should stand or fall on their own costs and benefits. And behavioral genetics offers some lessons in designing and evaluating interventions. Because normal differences in pre-school resources or parenting practices in working- and middle-class families have only limited effects on school readiness, interventions can have large effects only if they significantly change the allocation of resources or the nature of parenting practices.

The effects of most interventions on cognitive ability resemble the effect of exercise on physical conditioning: they are profound but short-lived. But if interventions make even small permanent changes in behavior that support improved cognitive ability, they can set off multiplier processes, with improved ability leading to more stimulating environments and still further improvements in ability. The best interventions, argues the author, would saturate a social group and reinforce individual multiplier effects by social multipliers and feedback effects. The aim of preschool programs, for example, should be to get students to continue to seek out the cognitive stimulation the program provides even after it ends.

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In national tests of school readiness, black preschoolers in the United States are not doing as well as white preschoolers. Researchers find black-white gaps not only in achievement and cognitive tests, but also in measures of readiness-related behaviors such as impulse control and ability to pay attention. Could some of these differences in school readiness be the consequence of differences in genetic endowment? In what follows I will review research evidence on this question.<sup>1</sup>

### Evidence on the Role of Genetic Differences

To evaluate the research findings on the role of genetic differences in cognitive ability, I begin by drawing a clear distinction between evidence that genetic endowment explains a large fraction of differences *within* races and evidence that it explains differences *between* races and ethnic groups. There can be little doubt that genetic differences are an important determinant of differences in academic achievement within racial and ethnic groups, though the size of that effect is not known precisely. Depending on the measure of achievement used, the sample studied, and the age of the subjects, estimates of the share of variance explained by genetic differences within racial and ethnic groups range from as low as 20 percent to upward of 75 percent. However, most estimates, particularly those for younger children, seem to cluster in the range of 30 to 40 percent. The fraction of variance explained by genetic differences in a population is termed the *heritability* of the trait for that population.<sup>2</sup>

But the heritability of academic achievement *within* racial or ethnic groups says little about whether genes play a role in explaining differences between racial groups. Suppose one scatters a handful of genetically diverse seed

corn in a field in Iowa and another in the Mojave Desert. Nearly all the variance in size within each group of seedlings could be due to genetic differences between the plants, but the difference between the average for those growing in the Mojave and those growing in Iowa would be almost entirely due to their different environments.

If researchers were able to identify all the genes that cause individual differences in school readiness, understand the mechanism by which they affect readiness and the magnitude of those effects, and assess the relative frequency of those genes in the black and white populations, they would know precisely the extent to which genetic differences explain the black-white gap. But only a few genes that influence cognitive ability or other behaviors relevant to school readiness have been tentatively identified, and nothing is known about their frequency in different populations. Nor are such discoveries imminent. Although genetic effects on several different learning and school-related behavior disorders have been identified and many aspects of personality are known to have a genetic component, genes have their primary effect on school readiness through their effect on cognitive ability.<sup>3</sup> Experts believe that a hundred or more genes are responsible for individual differences in cognitive ability. Many of these genes are likely to have weak and indirect effects that will be difficult to detect. It could be decades before enough genes are identified, and their frequencies estimated, to make it possible to determine what role, if any, they play in explaining group differences.

So it is necessary to turn to less direct ways of answering the question. Much has been written on this topic in the past fifty years. James Flynn's *Race, IQ, and Jensen*, published in 1980, remains the most thoughtful and thor-

## Clearing Up a Confusion

It is difficult to discuss genetic causation of the black-white test score gap. The reason, I believe, is that people confuse genetic causation with intractability. Suppose that the entire black-white gap in school readiness were genetic in origin, but that a shot could be given to black babies at birth to offset completely the effects of the genetic difference. Would anyone care about the genetic component of the racial gap? If it is possible to remedy or ameliorate the black-white difference, the only question is how much it would cost and whether society is willing to pay the price. As this article explains, genetic causation is nearly irrelevant to the question of how malleable a trait is.

Some argue that a genetic cause for black-white differences would lessen the moral imperative for removing them, but as the example of the shot illustrates, this is not the case. It would be hard to argue that the fact that the differences were genetic rather than environmental in origin would make it any less of an imperative for society to be sure that every black child got the shot. Some would say that the fact that the cause is beyond the child's control would make it more important. Jessica L. Cohen and I have made this argument in more detail in "Instinct and Choice: A Framework for Analysis," in *Nature and Nurture: The Complex Interplay of Genetic and Environmental Influences on Human Behavior and Development*, edited by Cynthia Garcia Coll, Elaine L. Bearer, and Richard Lerner (Hillsdale, N.J.: Lawrence Erlbaum and Associates, 2003), pp. 145–70.

ough treatment available.<sup>4</sup> More recently Richard Nisbett wrote a shorter review of this literature.<sup>5</sup> Both Flynn and Nisbett take the view, as do I, that genetic differences probably do not play an important role in explaining differences between the races, but the point remains controversial, and Arthur Jensen provides a recent discussion from a hereditarian perspective.<sup>6</sup> Here I will review the major types of evidence and explain why I think they suggest that environmental differences likely explain most, if not all, of the black-white gap in school readiness. I will concentrate entirely on the evidence on cognitive ability, as it is the most studied trait that influences school readiness, and genetically induced differences in cognitive ability account for the vast majority of genetically induced differences in school readiness within ethnic groups. Almost no studies have been done of racial differences in other traits

that might influence school readiness. And I choose to focus on the black-white gap rather than to consider the role of genetic differences in determining the academic readiness of disadvantaged groups more generally, again, because it is a topic that has been more thoroughly studied.

## Direct Evidence on the Role of Genes: European Ancestry and Cognitive Ability

Blacks in the United States have widely varying degrees of African and European ancestry. If their genetic endowment from their African ancestors is, on average, inferior to that from their European ancestors, then their cognitive ability would be expected to vary directly in proportion to the extent of their European ancestry. Some early attempts to assess this hypothesis linked skin color with test scores and found that lighter-

skinned blacks typically had higher scores. But skin color is not strongly related to degree of European ancestry, while socioeconomic status clearly is. Thus the differences might reflect environmental rather than genetic causes. Nearly all commentators agree that these early studies are not probative.

More recent studies have looked at measures of European ancestry, such as blood groups or reported ancestry, that are not visible. Such studies have found little or no correlation between the measure of ancestry and cognitive ability, though all are subject to methodological criticisms that could explain their failure to find such a link. Thus although these studies do not provide evidence for a role for genes in explaining black-white differences, they do not provide strong evidence against it.

### **Direct Evidence on the Role of Environment: Adoption and Cross-Fostering**

If there is no direct evidence of a role for genes in explaining the black-white gap, perhaps there is direct evidence that environment can or cannot account for the whole difference between blacks and whites. Several studies have shown that environmental differences between blacks and whites can, in a statistical sense, “explain” nearly all of the difference in cognitive ability between black and white children.<sup>7</sup> But because the studies do not completely control for the genetic endowment of either the child or the parents and because many of the variables used to explain the difference are themselves subject to genetic influence, the effect being attributed to environment may in reality be due to genetic differences.

What is needed is a way to see the effect of environment without confusing it with the ef-

fect of genetic endowment. For example, randomly choosing white and black children at birth and assigning them to be fostered in either black or white families would ensure that the children’s environments were not correlated with their genetic potential and would show how much difference environment makes. No existing study replicates the conditions of this experiment exactly, but some come close. The strongest evidence for both the environmentalist and hereditarian perspectives is of this sort.

After the end of World War II both black and white soldiers in the occupying armies in Germany fathered children with white German women. Klaus Eyferth gathered data on a large number of these children, of mainly working-class mothers, and gave the children intelligence tests.<sup>8</sup> He found almost no difference between the children of white fathers and those of black fathers. The finding is remarkable given that the black children faced a somewhat more hostile environment than the white children. Hereditarians have challenged these findings by appealing to the possibility that the black soldiers who fathered these children might have been a particularly elite group. Flynn has researched the plausibility of this explanation and concludes that such selection did not play more than a small role.<sup>9</sup> Thus Eyferth’s study suggests that the black-white gap is largely, and possibly entirely, environmental.

A study similar to Eyferth’s found the cognitive ability of black children raised in an orphanage in England to be slightly higher than that of white children raised there.<sup>10</sup> Again, critics have raised the possibility that the black children were genetically advantaged relative to other blacks, and the whites disadvantaged relative to other whites. And again, Flynn finds it unlikely that this contention explains

much of the disappearance of the black-white gap.<sup>11</sup> This study, too, suggests that the black-white gap is mainly environmental.

If the black-white gap is mainly genetic in origin, children's cognitive ability should not depend on the race of their primary caregiver, comparing those of the same race. Yet two studies comparing the experience of black children raised by black or white mothers suggest that it does.<sup>12</sup> Here too, because the children were not randomly assigned to their caregivers, it is possible that the children raised by black mothers were of lower genetic potential, but it would be hard to make such a selection story explain more than a small fraction of the apparent environmental effect.

Another transracial adoption study provides mixed evidence, but some of the strongest that genes play a role in explaining the black-white gap.<sup>13</sup> A group of children, some with two black parents and some with one white and one black parent, were raised in white middle-class families. When the children's cognitive ability was tested at age seven, the children with two black parents scored 95, higher than the average black child in the state (89) and only slightly below the national average for whites, while the mixed-race children scored 110, which was considerably above it.<sup>14</sup> On the one hand, this finding suggests a huge effect of environment on the cognitive ability of the adopted black and mixed-race children. On the other hand, the higher scores of the mixed-race children suggest that parents' genes may account for some of the difference from the black children, and that the mixed-race children may have had a better inheritance by virtue of having one white parent. Both black and mixed-race children scored worse than the biological children of their adoptive parents

(who scored 116), an expected finding because the adopting parents were an elite group and likely passed on above-average genetic potential to their children. But they also scored considerably below the average of 118 for comparison white children adopted into similar homes.

When the same children were retested ten years later, the results were different.<sup>15</sup> The

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scores of the children with two black parents had dropped to about the average for blacks in the state where they lived before they were adopted (89). The scores of the mixed-race children had dropped too (99), but remained intermediate between those of the children with two black parents and those of the adoptive parents' biological children, which had also declined, to 109. The scores of the white children raised in adoptive homes had dropped the most, falling to 106.

The disappearance of the salutary effect of the adoptive home, however, does not mean that genes determine black-white differences. We can assume that as the children aged and moved out into the world, the effect of the home environment diminished, and both whites and blacks tended to the average for their own population because of either genetic or environmental effects. By showing how the effect of a child's home environment disappears by adolescence, this study sug-

gests that environmental disadvantages experienced by blacks as children cannot explain the deficit in their cognitive ability as adolescents and adults. But environmental disadvantages facing black adolescents and adults could still explain those deficits. The transience of environmental effects on cognitive ability is a theme to which I shall return. The persistence of the advantage of the mixed-race children over the children with two black parents is suggestive of a role for genes. It is not, though, definitive: several other explanations have been offered, including the late adoption of the children with two black parents and parental selection effects unrelated to race.<sup>16</sup>

### Indirect Evidence on the Role of Genetic Differences

Although the direct evidence on the role of environment is not definitive, it mostly suggests that genetic differences are not necessary to explain racial differences. Advocates of the hereditarian position have therefore turned to indirect evidence.<sup>17</sup>

Several authors have argued that estimates of the heritability of cognitive ability put limits on the plausible role of environment.<sup>18</sup> The argument is normally made in a mathematical form, but it boils down to this. First, it is now widely accepted that differences in genetic endowment explain at least 60 percent of the variance in cognitive ability among adults in the white population in the United States.<sup>19</sup> If all the environmental variation among U.S. whites can explain only 40 percent of the variance among whites, how could environmental differences explain the huge gap between blacks and whites? The mathematical argument implies that the average black environment would have to be worse than at least 95 percent of white environments, but observable characteristics of blacks and whites are

not that different. For example, black deficits in education or in socioeconomic status place the average black below only about 60 to 70 percent of whites.<sup>20</sup>

The heritability of cognitive ability is also crucial to a second type of indirect evidence for a role of genetic differences in explaining the black-white gap. Arthur Jensen has advanced what he calls “Spearman’s Hypothesis,” after the late intelligence researcher Charles Spearman, who observed that people who had large vocabularies were good at solving mazes and logic problems and were also more likely to have command of a wide range of facts. Spearman posited that a single, largely genetic, mental ability that he called *g* (for general mental ability) explained the correlation of people’s performance across a wide range of tests of mental ability. Researchers now know that a single underlying ability cannot explain all the tendency of people who do well on one type of test to do well on another.<sup>21</sup> But it is possible to interpret the evidence as indicating that there is a single ability that differs among people, that is subject to genetic influence, and that explains much of the correlation across tests. Other interpretations are also possible, but this one cannot be discounted. In a series of studies Jensen and Rushton have argued that different types of tests tap this general ability to different degrees; that the more a test taps *g*, the more it is subject to genetic influence; and that black-white differences are largest on the tests most reflective of the underlying general ability, *g*.<sup>22</sup>

Using several restrictive assumptions about the nature of genetic and environmental influence on genetic ability, researchers can use this information to estimate the fraction of the black-white gap that is due to differences in genetic endowment. The more the



pattern of black-white differences across different tests resembles the pattern of genetic influence on different tests, the more the statistical procedure will attribute the black-white differences to genetic differences. Using this method, David Rowe and Jensen have independently estimated that from one-half to two-thirds of the black-white gap is genetic in origin.<sup>23</sup>

### **A Problem for the Indirect Arguments: Gains in Cognitive Ability over Time**

Over the past century, dozens of countries around the world have seen increases in measured cognitive ability over time as large as or even larger than the black-white gap.<sup>24</sup> The phenomenon has been christened the “Flynn Effect,” after James Flynn, who did the most to investigate and popularize this worldwide trend. The score gains have been documented even between a large group of fathers and sons taking the same test only decades apart, making it impossible that the gains are due to changes in genes. Clearly environmental changes can cause huge leaps in measured cognitive ability. Although it might not seem plausible that the average black environment today is below the 5th percentile of the white distribution of environments, it is certainly plausible that the average black environment in the United States today is as deprived as the average white environment of thirty to fifty years ago—the time it took for cognitive ability to rise by an amount equal to the black-white gap in many countries. These gains in measured cognitive ability over time point to a problem in the argument that high heritability estimates for cognitive ability preclude large environmental effects.

Gains in cognitive ability over time also challenge the logic of Jensen’s genetic explanation for the pattern of black-white differ-

ences across different types of tests. All studies show that gains on different tests are positively correlated with measures of test score heritability, and most studies show that gains are positively correlated with the extent to which a test taps the hypothesized general cognitive ability.<sup>25</sup> There is little doubt that applying the same method as Rowe and Jensen used to data on gains in cognitive ability over time would show them to be partially genetic in origin, something we know cannot be true.

So, what is it that is wrong with the logic of these two arguments, that the high heritability of cognitive ability limits the possible effect of the environment and that the pattern of black-white differences across different tests shows those differences to be genetic in origin? And in particular, where is the problem in the first?

It is important to detect the flaw, because if the logic of the argument were sound, the case for environmental causes of black-white differences would be difficult to make, and the possibility of remedying those differences would be remote. But before I explain, I want to cite two other pieces of evidence marshaled by advocates of the hereditarian position that suggest the limited power of the environment to change cognitive ability (and therefore to explain the entire black-white gap). The first is that the heritability of cognitive ability rises with age. It does so at the expense of the effect of family environment, which disappears nearly completely in most studies of late adolescents and adults.<sup>26</sup> The disappearance of the effect on black children of being raised in white families, which I have already noted, is just one case of a general finding from several different types of studies. A second piece of evidence is the fade-out of the effect of preschool programs

on cognitive ability. Although such programs have been shown to have profound effects on the measured ability of children, the effects fade once the programs end, leaving little evidence of any effect by adolescence.<sup>27</sup> Is it possible to reconcile the high heritability of cognitive ability with large, but transient, environmental effects?

## The Interplay of Genes and the Environment

To explain this puzzle, James Flynn and I have proposed a formal model in which genes and environment work together, rather than independently, in developing a person's cognitive ability.<sup>28</sup> The solution involves three aspects of the process by which individual ability is molded that are overlooked by the logic that implies small environmental effects. We illustrate our argument with a basketball analogy.

How can genes and environment both be powerful in shaping ability? Consider a young man with a small genetic predisposition toward greater height and faster reflexes. When he is young, he is likely to be slightly better than his playmates at basketball. His reflexes will make him generally better at sports, and his height will be a particular advantage when it comes to passing, catching, and rebounding. These advantages by themselves confer only a small edge, but they may be enough to make the game more rewarding for him than for the average person and get him to play more than his friends and to improve his play more over time. After a while, he will be considerably better than the average player his age, making it likely that he will be picked first for teams and perhaps receive more attention from gym teachers. Eventually, he joins a school team where he gets exhaustive practice and professional coaching. His basketball ability is now far su-

perior to that of his old playmates. Through a series of feedback loops, his initial minor physical advantage has been multiplied into a huge overall advantage. In contrast, a child who started life with a predisposition to be pudgy, slow, and small would be very unlikely to enjoy playing basketball, get much practice, or receive coaching. He would therefore be unlikely to improve his skills. Assuming children with a range of experience between these two extremes, scientists would find that a large fraction of the variance of basketball playing ability would be explained by differences in genetic endowment—that basketball ability was highly heritable. And they would be right to do so. But that most certainly would not mean that short kids without lightning reflexes could not improve their basketball skills enormously with practice and coaching.

The basketball analogy so far illustrates two of the considerations that Flynn and I believe are important for understanding the implications of behavioral genetic studies of cognitive ability. First, genes tend to get *matched* to complimentary environments. When that happens, some of the power of environment is attributed to genes. Only effects of environment shared by all children in the same family and effects of environment uncorrelated with genes get counted as environmental. Second, the effect of genetic differences gets *multiplied* by positive feedback loops. Small initial differences are multiplied by processes where people's initially varying abilities are matched to complimentary environments that cause their abilities to diverge further.

In theory this same multiplier process could be driven by small environmental differences. But to drive the multiplier to its maximum, the environmental advantage would



have to be as constant over time as the genetic difference, because in the absence of the initial advantage there will be a tendency for the whole process to unwind. For example, suppose that midway through high school the basketball enthusiast injures a leg, which makes him less steady and offsets his initial advantage in height and reflexes. Because of all his practice and learning, he will still be a superior player. But his small decrement in performance could mean discouragement, more bench time, or not making the cut for the varsity team. This could lead to a further deterioration of his skills and further discouragement, until he gives up playing on the team entirely. Although each individual's experience will differ, the theory that Flynn and I lay out would have people with average physical potential reverting to average ability over time, on average.

The transitory nature of most environmental effects not driven by genetic differences helps explain why environmental differences do not typically drive large multipliers and produce the same large effects as genetic differences. That same transience helps explain why environment can be potent but still cause a relatively small share of the variance of cognitive ability in adults.<sup>29</sup>

### **Social Multipliers and the Effect of Averaging**

If most external environmental influences are transitory and transitory environmental effects are unable to drive multipliers, what explains the large gains in cognitive ability over the past century? That question has two answers. One is the social multiplier process. The other is that many random transient environmental effects that lean in one direction when *averaged* together can substitute for a single persistent environmental cause. This is the third point missed by the argument that

claims that high heritability implies small environmental effects.

Another basketball analogy will help explain social multipliers. During the 1950s television entered many U.S. homes. Professional basketball, with its small arena, could not reach as wide an audience as baseball, but basketball translated much better to the small screen. Thus public interest in basketball began to grow. The increased interest made it easier for enthusiasts to find others to play with, thus increasing the opportunities to improve skills. As skills improved, standards of play rose, with players learning moves and skills from each other. As more people played and watched the game, interest increased still further. More resources were devoted to coaching basketball and developing basketball programs, providing yet more opportunities for players to improve their skills. In the end, the small impetus provided by the introduction of television had a huge impact on basketball skills.

A similar process may well be at work for cognitive ability. An outpouring of studies in recent years suggests that social effects have an important influence on school performance.<sup>30</sup> One study of an experimental reduction in school class size resulting in major achievement score gains suggests that a very large fraction of the gains came through the children's extended association with their peers, who shared the experience of small class sizes.<sup>31</sup> In this case an arguably minor intervention had large and long-lasting effects largely owing to a social multiplier effect.

But improvements in cognitive ability could have many triggers, rather than a single one. Many such triggers over the past half-century averaged together could be acting to raise cognitive ability. Increasing cognitive de-

mands from more professional, technical, and managerial jobs; increased leisure time; changing cognitive demands of personal interactions; or changing attitudes toward intellectual activity could all be playing a role. And small initial changes along any of these dimensions would be magnified by individual and social multipliers.

## Genes and Environment and the Black-White Gap

The black-white gap in measured cognitive ability may come about in a similar way, but it could have even more triggers. Segregation and discrimination have caused many aspects of blacks' environment to be inferior to that of whites. Averaged together, the total impact can be large, even if each individual effect is small. Suppose, for example, that environment relevant to the formation of cognitive ability consists of 100 factors, each with an equal effect. If for each of these 100 factors the average black were worse off than 65 percent of whites, he would be worse off than 90 percent of whites when the effects of all the environmental factors were considered together. (The disparity is the necessary result of accumulating a large number of effects when two groups have slightly different means for all the effects.)<sup>32</sup> Taking the total effect of environment in this way, considering the underestimate of the total effect of environment because some of its power is attributed to genes, and considering individual and social multipliers, a purely environmental explanation for black-white differences becomes plausible despite high estimates for the heritability of cognitive ability.

Moreover, our model also has explanations for the correlation of the heritability of scores on different tests with the size of the black-white gap on those tests and the anomalous correlation of the size of gains in cognitive

ability over time on different tests with the heritability of those test scores. Those cognitive abilities for which multiplier processes are most important will be the ones that show the largest heritability, because of the environmental augmentation of the genetic differences. But they will also be the ones on which a persistent change in environment will have the biggest influence. Thus we might expect that persistent environmental differences between blacks and whites, as well as between generations, could cause a positive correlation between test score heritabilities and test differences.<sup>33</sup> Rushton and Jensen's indirect evidence of a genetic role in black-white differences is, therefore, not probative.

## Implications and Conclusions

The indirect evidence on the role of genes in explaining the black-white gap does not tell us how much of the gap genes explain and may be of no value at all in deciding whether genes do play a role. Because the direct evidence on ancestry, adoption, and cross-fostering is most consistent with little or no role for genes, it is unlikely that the black-white gap has a large genetic component.

But what if it does? What would be the implications for the school readiness of children? Much of the variance in human behavior, including cognitive ability and achievement test scores, can be traced to differences in individuals' genetic endowments. But as indisputable as is the role of genes in shaping differences in outcomes within races, so is the role of environment. Studies of young children show that environmental differences explain more variation than do genetic differences. And even studies showing an important role for genes in no way rule out the possibility of improving the school performance of disadvantaged children through

interventions aimed at enhancing their school readiness. Interventions should stand or fall on their own costs and benefits and not be prejudged on the basis of genetic pessimism.

In fact, studies of the role of genes and environment in determining school readiness offer some useful lessons in designing and evaluating interventions. These studies show that normally occurring differences in preschool resources or parenting practices in working- and middle-class families have only limited effects on school readiness once the correlation due to parents' and children's genes is taken out of play.<sup>34</sup> Thus small interventions that make only modest changes in the allocation of resources or the nature of parenting practices will have limited to modest effects at best. Effects will likely be somewhat larger if interventions target very disadvantaged families, probably because the room for improvement is greater.<sup>35</sup>

Achieving permanent effects on cognitive ability is harder than achieving large effects. Most environmental effects on cognitive ability seem to be like the effect of exercise on physical conditioning: profound but short-lived. But even short-lived improvements in cognitive ability can be valuable if they mediate longer-term changes in achievement—for example, if improved cognitive ability for some period of time allows students to learn to read more quickly, putting them on a permanently higher achievement path. And evidence suggests that programs aimed at improving cognitive ability do have long-term effects on achievement even if they have no significant long-term effects on cognitive ability. However, if interventions make even

small permanent changes in behavior that support improved cognitive ability, they can set off multiplier processes, with improved ability leading to better environments and still further improvements in ability. If we knew what aspects of preschool programs help elevate cognitive ability, and if we could get children to continue to seek out such stimulation after they leave preschool programs, their increased ability could lead them to associate with more able peers, to have the confidence to take on more demanding academic challenges, and to get the further advantage of yet more positive stimulation from these activities. This, in turn, could further develop their cognitive ability. Long-lived effects are more likely to be large effects.

Effects are particularly likely to be large if an intervention saturates a social group and allows the individual multiplier effects to be reinforced by social multipliers or feedback effects. If students find themselves among others with greater ability, individual interactions and group activities are more likely to give rise to further improvements in cognitive ability. In this same vein, evaluations that do not take into account the social effects of the intervention on children who did not directly take part may be missing an important aspect of the effects of an intervention.

Although much of normal environmentally induced variance in cognitive ability seems to be transient, if interventions could induce even small long-lasting changes in behavior, they might produce very large effects through the multiplier process. Taking advantage of such processes may make it possible to overcome the black-white gap and put black and white children on an even footing.

## Endnotes

1. The review necessarily highlights only the most important studies; a complete review of all the arguments on both sides of this debate would require hundreds of pages and be beyond the scope of this article.
2. Heritability is estimated by examining the similarity of people with different degrees of genetic similarity raised in similar sorts of environments, and there is some reason to believe that most estimates are somewhat overstated by existing methods. Robert Plomin and others, *Behavioral Genetics*, 4th ed. (New York: Worth Publishers, 2001), in chapter 5 and the appendix, provide a thorough discussion of the methods used to estimate heritability. Mike Stoolmiller, "Implications of the Restricted Range of Family Environments for Estimates of Heritability and Nonshared Environment in Behavior-Genetic Adoption Studies," *Psychological Bulletin* 125 (1999): 392–409, shows that adoption studies probably overstate the degree of heritability and speculates on reasons why some other methods may as well.
3. Robert Plomin and others, *Behavioral Genetics* (see note 2), examine learning disorders on pp. 145–49, ADHD on pp. 227–29, and personality in chapter 12. For the effects of genes on cognitive ability, see Marcie L. Chambers and others, "Variation in Academic Achievement and IQ in Twin Pairs," *Intelligence* (forthcoming); Lee Anne Thompson and others, "Associations between Cognitive Abilities and Scholastic Achievement: Genetic Overlap but Environmental Differences," *Psychological Science* 2 (1991): 158–65; and Sally J. Wadsworth, "School Achievement," in *Nature and Nurture during Middle Childhood*, edited by John C. DeFries, Robert Plomin, and David W. Fulker (Oxford: Blackwell, 1994), pp. 86–101.
4. James R. Flynn, *Race, IQ, and Jensen* (London: Routledge, 1980).
5. Richard Nisbett, "Race, Genetics, and IQ," in *The Black-White Test Score Gap*, edited by Christopher Jencks and Meredith Phillips (Brookings, 1998), pp. 86–102.
6. Arthur Jensen, *The g Factor* (Westport, Conn.: Praeger, 1998), pp 350–531.
7. Jane R. Mercer, "What Is a Racially and Culturally Nondiscriminatory Test? A Sociological and Pluralistic Perspective," in *Perspectives on "Bias in Mental Testing,"* edited by Cecil R. Reynolds and Robert T. Brown (New York: Plenum Press, 1984); Jonathan Crane, "Race and Children's Cognitive Test Scores: Empirical Evidence That Environment Explains the Entire Gap," mimeo, University of Illinois at Chicago, 1994; and Jeanne Brooks-Gunn and others, "Ethnic Differences in Children's Intelligence Test Scores: Role of Economic Deprivation, Home Environment, and Maternal Characteristics," *Child Development* 67, no. 2 (1996): 396–408.
8. This is based on the account by James R. Flynn (*Race, IQ, and Jensen*, pp. 84–87; see note 4) of Klaus Eyferth, "Leistungen verschiedener Gruppen von Besatzungskindern in Hamburg-Wechsler Intelligenztest für Kinder (HAWIK)," *Archiv für die gesamte Psychologie* 113 (1961): 222–41.
9. Flynn, *Race, IQ, and Jensen*, pp. 84–102 (see note 4).
10. Barbara Tizard, "IQ and Race," *Nature* 247, no. 5439 (February 1, 1974).
11. Flynn, *Race, IQ, and Jensen*, pp. 108–11 (see note 4).
12. Elsie G. J. Moore, "Family Socialization and the IQ Test Performance of Traditionally and Transracially Adopted Black Children," *Developmental Psychology* 22 (1986): 317–26; and Lee Willerman and others, "Intellectual Development of Children from Interracial Matings: Performance in Infancy and at 4 Years," *Behavioral Genetics* 4 (1974): 84–88.

13. Sandra Scarr and Richard A. Weinberg, "IQ Test Performance of Black Children Adopted by White Families," *American Psychologist* 31 (1976): 726–39; and Sandra Scarr and Richard A. Weinberg, "The Minnesota Adoption Studies: Genetic Differences and Malleability," *Child Development* 54 (1983): 260–67.
14. These are IQ scores, which have a mean of 100 and a standard deviation of 15 in the U.S. population.
15. Sandra Scarr and others, "The Minnesota Transracial Adoption Study: A Follow-Up of IQ Test Performance at Adolescence," *Intelligence* 16 (1992): 117–35.
16. But see Arthur Jensen, *The g Factor*, pp. 477–78 (see note 6), on whether late adoption can explain the difference.
17. One body of evidence is difficult to judge. See J. Philippe Rushton, *Race, Evolution, and Behavior: A Life History Perspective*, 3rd ed. (Port Huron, Mich.: Charles Darwin Research Institute, 2000). Rushton has proposed a theoretical framework that would explain a genetic gap in cognitive ability between blacks and whites and has marshaled evidence for it. But because much of the evidence was known before the theory was proposed, some view the theory as nothing more than post hoc rationalization for hereditarian views on the black-white gap. At most it suggests that some of the black-white gap may be genetic, but it does not suggest how much.
18. Arthur Jensen, *Educability and Group Differences* (New York: Harper and Row, 1973), pp. 135–39, 161–73, 186–90; Arthur Jensen, *Educational Differences* (London: Methuen, 1973), pp. 408–12; Jensen, *The g Factor*, pp. 445–58 (see note 6); and Richard Herrnstein and Charles Murray, *The Bell Curve: Intelligence and Class Structure in American Life* (New York: Simon and Schuster, 1994), pp. 298–99.
19. Plomin and others, *Behavioral Genetics*, p. 177 (see note 2); and Ulric Neisser and others, "Intelligence: Knowns and Unknowns," *American Psychologist* 51, no. 2(1996): 85.
20. Author's calculations from the 1979 National Longitudinal Survey of Youth.
21. John B. Carroll, *Human Cognitive Abilities: A Survey of Factor-Analytic Studies* (Cambridge University Press, 1993), is the most comprehensive survey of what is known about the correlation of scores on different types of mental tests.
22. See J. Philippe Rushton and Arthur Jensen, "Thirty Years of Research on Race Differences in Cognitive Ability," *Psychology, Public Policy, and Law* (forthcoming), for a review of this evidence and citations to the original studies.
23. David Rowe, Alexander Vazsonyi, and Daniel Flannery, "Ethnic and Racial Similarity in Developmental Process: A Study of Academic Achievement," *Psychological Review* 101, no. 3 (1994): 396–413; Jensen, *The g Factor*, pp. 464–67 (see note 6).
24. James R. Flynn, "Massive Gains in 14 Nations: What IQ Tests Really Measure," *Psychological Bulletin* 101 (1987): 171–91; James R. Flynn, "IQ Gains over Time," in *Encyclopedia of Human Intelligence*, edited by Robert J. Sternberg (New York: Macmillan, 1994), pp. 617–23; James R. Flynn, "IQ Gains over Time: Toward Finding the Causes," in *The Rising Curve: Long-Term Gains in IQ and Related Measures*, edited by Ulric Neisser (Washington: American Psychological Association, 1998), pp. 551–53.
25. Existing evidence suggests that IQ gains across subtests are probably positively correlated with *g* loading. See Roberto Colom, Manuel Juan-Espinosa, and Lu s F. Garc a, "The Secular Increase in Test Scores Is a

- ‘Jensen effect,’” *Personality and Individual Differences* 30 (2001): 553–58; and Manuel Juan-Espinosa and others, “Individual Differences in Large-Spaces Orientation: *g* and Beyond?” *Personality and Individual Differences* 29 (2000): 85–98, for much stronger correlations between *g* loadings and IQ gains. Jensen, *The *g* Factor*, pp. 320–21 (see note 6), reviews a number of studies of the relation between subtests gains and *g* loadings, all of which show weak positive correlations. J. Philippe Rushton, “Secular Gains in IQ Not Related to the *g* Factor and Inbreeding Depression—unlike Black-White Differences: A Reply to Flynn,” *Personality and Individual Differences* 26 (1999): 381–89, finds that a measure of *g* developed on the Wechsler Intelligence Scale for Children has loadings that are negatively correlated with subtest gains in several countries. But see James R. Flynn, “The History of the American Mind in the 20th Century: A Scenario to Explain IQ Gains over Time and a Case for the Irrelevance of *g*,” in *Extending Intelligence: Enhancement and New Constructs*, edited by P. C. Kyllonen, R. D. Roberts, and L. Stankov (Hillsdale, N.J.: Erlbaum, forthcoming.) for an argument that IQ gains are greatest on tests of fluid *g* rather than crystallized *g*. He finds a positive (though statistically insignificant) correlation between a measure of fluid *g* he develops and IQ gains in the data used by Rushton. Olev Must, Aasa Must, and Vilve Raudik, “The Flynn Effect for Gains in Literacy Found in Estonia Is Not a Jensen Effect,” *Personality and Individual Differences* 33 (2001); and Olev Must, Aasa Must, and Vilve Raudik, “The Secular Rise in IQs: In Estonia the Flynn Effect Is Not a Jensen Effect,” *Intelligence* 31 (2003): 461–71, find no correlation between *g* loadings and gains on two tests in Estonia, but these are achievement tests with a strong crystallized bias.
26. Plomin and others, *Behavioral Genetics*, pp. 173–77 (see note 2).
27. Irving Lazar and Richard Darlington, “Lasting Effects of Early Education: A Report from the Consortium for Longitudinal Studies,” *Monographs of the Society for Research in Child Development* 47, nos. 2–3 (1982).
28. William T. Dickens and James Flynn, “Heritability Estimates versus Large Environmental Effects,” *Psychological Review* 108, no. 2 (2001).
29. This is not to say that there are no permanent or long-lasting environmental effects on cognitive ability. The effects of brain damage can be severe and permanent. However, such permanent environmental effects evidently explain only a small fraction of normal variation in cognitive ability. Shared family environment plays a large role in explaining variance in cognitive ability when children are spending most of their time in the home, with their activities strongly influenced by their parents. But that effect fades as they spend more of their time away from home and in self-directed activities.
30. Eric A. Hanushek and others, “Does Peer Ability Affect Student Achievement?” Working Paper 8502 (Cambridge, Mass.: National Bureau of Economic Research, 2001); Caroline Hoxby, “Peer Effects in the Classroom: Learning from Gender and Race Variation,” Working Paper 7867 (Cambridge, Mass.: National Bureau of Economic Research, 2001); Dan M. Levy, “Family Income and Peer Effects as Determinants of Educational Outcomes,” Ph.D. diss., Northwestern University, 2000; Donald Robertson and James Symons, “Do Peer Groups Matter? Peer Group versus Schooling Effects on Academic Achievement,” *Economica* 70 (2003): 31–53; Bruce Sacerdote, “Peer Effects with Random Assignment: Results from Dartmouth Roommates,” *Quarterly Journal of Economics* (May 2001): 681–704; David J. Zimmerman, “Peer Effects in Academic Outcomes: Evidence from a Natural Experiment,” *Review of Economics and Statistics* 85 (2003): 9–23.



31. Michael A. Boozer and Stephen E. Cacciola, "Inside the 'Black Box' of Project STAR: Estimation of Peer Effects Using Experimental Data," Discussion Paper 832 (Economic Growth Center, Yale University, 2001).
32. In statistics this is referred to as the law of large numbers—that the variance of a mean falls as the number of items being averaged goes up. See Eugene Lukacs, *Probability and Mathematics Statistics: An Introduction* (New York: Academic Press, 1972). It applies whether or not the weights being put on the elements are equal. Because the variance and standard deviation of the mean fall, while the average difference stays the same, the difference in standard deviations grows. The example assumes that the effects are all uncorrelated with each other and that each has a normal distribution in the white and the black populations. If the effects were assumed to be correlated or the weights unequal, the results would be less dramatic, but with observed values for correlations of environmental factors, increasing the number of items to be averaged could produce the same results.
33. Dickens and Flynn, "Heritability Estimates vs. Large Environmental Effects" (see note 28).
34. Plomin and others, *Behavioral Genetics*, p. 201 (see note 2).
35. Eric Turkheimer and others, "Socioeconomic Status Modifies Heritability of IQ in Young Children," *Psychological Science* 14, no. 6 (2003). Their own study finds that shared family environment explains 60 percent of the variance of an IQ test score in low-socioeconomic-status seven-year-olds, which is a much larger share than other studies have found. For example, see Kathryn Asbury and others, "Environmental Moderators of Genetic Influence on Verbal and Nonverbal Abilities in Early Childhood" (Institute of Psychiatry, De Crespigny Park, London, 2004).