Psychological Review
2001, Vol. 108, No. 2, 346–369

0033-295X/01/$5.00 DOI: 10.1037//0033-295X.108.2.346

Heritability Estimates Versus Large Environmental Effects: The IQ Paradox Resolved

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Some argue that the high heritability of IQ renders purely environmental explanations for large IQ differences between groups implausible. Yet, large environmentally induced IQ gains between generations suggest an important role for environment in shaping IQ. The authors present a formal model of the process determining IQ in which people's IQs are affected by both environment and genes, but in which their environments are matched to their IQs. The authors show how such a model allows very large effects for environment, even incorporating the highest estimates of heritability. Besides resolving the paradox, the authors show that the model can account for a number of other phenomena, some of which are anomalous when viewed from the standard perspective.

Jensen (1973a, pp. 135–139, 161–173, 186–190; 1973b, pp. 408–412; 1998, pp. 445–458) and Herrnstein and Murray (1994, pp. 298–299) argue that widely accepted estimates of the heritability of IQ—the fraction of the variance of IQ in a population caused by differences in genetic endowment—render environmental explanations of large IQ differences between groups implausible. These authors apply their analysis to differences between racial groups, but it is just as applicable to groups separated in time.

For all group differences, this analysis poses the question: If the observed variance in environment accounts for so little variance in adult IQ, how could environmental factors cause large differences? Yet, a growing body of evidence suggests huge environmental effects on IQ—particularly the evidence of massive IQ gains over time. Dutch gains between 1952 and 1982 are 20 IQ points,[1] and Israeli gains are similar (Flynn, 1987, 1994, 1998b, pp. 551–553). The fact that IQ gains are mainly environmentally caused turns the problem into a paradox: We know that potent environmental factors exist; Jensen's analysis suggests that they should not exist. How can this paradox be resolved?

One could challenge existing heritability estimates. However, a committee of highly respected researchers convened by the American Psychological Association concluded that by late adolescence, heritability is "around .75" (Neisser et al., 1996, p. 85). Future research may change this value, but we do not choose to dispute it. We suspect that when the dust settles, the value for h^2 in adults will be high enough to allow Jensen to make his argument. As Herrnstein and Murray (1994, pp. 298–299) point out, a value for h^2 of .6 works nearly as well as .75 or .80. Therefore, we challenge the analysis itself. We replace the causal model that produces the paradox with a formal model of our own. Our model posits strong reciprocal causation between phenotypic IQ and environment. That reciprocal causation produces gene × environment correlation. The model has three features that allow for potent environmental effects while accommodating high estimates of heritability.

First, the reciprocal causation between IQ and environment leads to a positive correlation between environment and genotype that masks the potency of environment. Because of this correlation, both direct effects of genotype on IQ and indirect effects through induced environments are measured by standard heritability estimates. Judging the size of the environmental effects by the fraction of variance not explained by genotype will understate its full magnitude because to do so ignores environmental effects induced by differences in genotype. Second, reciprocal causation

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We would like to acknowledge the able research assistance of Jessica Cohen, Jennifer Eichberger, Cyd Fremmer, Sharon Hoke, Megan Monroe, Neil Siegel, and particularly Kate Withers (who checked all the math). We would also like to thank the following for their helpful comments: Henry Aaron, George Akerlof, Sam Bowles, Colin Cameron, Bryan Caplan, Curtis Crawford, Mary Alice Fisher, Robin Hanson, Judith Harris, Christopher Jencks, Arthur Jensen, Melvin Kohn, David Levine, John Loehlin, Carmi Schooler, Eric Turkheimer, and participants in seminars held at the Psychology Department at the University of Virginia; the Economics Departments of the University of California at Berkeley, Davis, and Santa Cruz, George Mason University, George Washington University, and Harvard University; the Schools of Public Policy at Georgetown University and the University of Chicago; and the Institute on Race and Social Division at Boston University. The research was supported by National Science Foundation Grant SBR-970925 and the National Community Development Policy Analysis Network's funders: The Rockefeller Foundation, the Annie E. Casey Foundation, and the John D. and Catherine T. MacArthur Foundation.

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[1] With IQ measured on a scale with a mean of 100 and a standard deviation of 15 points.

produces a multiplier effect that inflates both genetic and environmental advantages by a process in which higher IQ leads one into better environments causing still higher IQ, and so on. Third, we hypothesize that at least three aspects of this process lead to averaging of many environmental influences. Because of the law of large numbers, this averaging allows environmental effects to be arbitrarily large relative to the variance of an index of their combined effect—even though they seem small relative to the variance of environmental effects not correlated with genetic endowment.

One factor that produces averaging of environmental influences also produces an additional multiplier effect. We believe that it is not only people's phenotypic IQ that influences their environment, but also the IQs of others with whom they come into contact. The latter is influenced by society's average IQ. Therefore, if some external factor causes the IQs of some individuals to rise, this will improve the environment of others and cause their IQs to rise. We call this the *social multiplier,* and it can play an important role in determining the impact of society-wide changes in our model.

Our model not only shows how environmental influences that appear small can have large effects on IQ, it also explains other observations that may appear anomalous from the perspective of a naive model. It explains increases in heritability as people age, the disappearance of shared environmental influences in adulthood, increases in the stability of IQ as people age, differences in the rate of gain and rate of decay of the effects of compensatory education, and the effects of adoption and cross-racial parenting. Our model can explain these things, but only if the direct effects of environment on IQ are large, though perhaps short lived, and only if phenotypic IQ has a large reciprocal effect on environment.

We are not the first to suggest that there is reciprocal causation between IQ and environment that leads to correlation of genes and environment. Both Jensen (1973a, p. 235; 1973b, p. 417) and Scarr (1985) have warned against interpreting correlations between environmental factors and IQ as proof of environmental potency, and both have emphasized the possibility of reciprocal causation (Jensen, 1998, pp. 179–181; Scarr, 1992; Scarr & McCartney, 1983). In fact, the model that Scarr (1992) described is very similar to our own, but our formal analysis of the model leads us to different conclusions. Bell (1968) and Bell and Harper (1977) examined the role that even very young children may play in shaping their environment. Jencks (1972, pp. 66–67; 1980), Jensen (1975), and Goldberger's (1976) response to Jensen (1975) show that all three authors had a clear understanding of how correlation between genetic endowment and environment could mask environmental effects. The notion of reciprocal causation of IQ and environment is at the core of Bronfenbrenner's bioecological model of development (1989) and kindred work by Ceci (1990) and Bronfenbrenner and Ceci (1994). Kohn and Schooler (1983) and Schooler, Mulatu, and Oates (1999) have estimated the reciprocal effects of the complexity of work and a measure of individual intellectual flexibility. The latter article also shows that intellectual flexibility is highly correlated with several more standard measures of cognitive ability. Jensen (1998, pp. 179–181) and Neisser et al. (1996, p. 86) precede us in suggesting that reciprocal causation of IQ and environment may explain the rise in heritability from childhood to adulthood. Harris (1995; 1999, p. 247) and Harris and Liebert (1991, p. 58) also discuss reciprocal effects and describe what we term multiplier effects as feedback loops and vicious cycles. Harris (1999, pp. 248–251) precedes us in arguing that social interaction can contribute to understanding large IQ differences between groups. Turkheimer and Gottesman (1996) and Turkheimer (1997) have developed an even more complex model than our own with illustrative simulations. Their simulations show high heritability coexisting with potent environmental factors. Winship and Korenman (1999) work out an example of environmental multiplier effects due to reciprocal causation between IQ and schooling.

With our model, we attempt to systematize and formalize what might seem to be a miscellaneous body of contributions and rigorously work out their implications. We believe that this clarifies the processes at work or at least renders coherent fruitful insights whose significance is not yet widely appreciated.

The article is divided into five sections. The first section states Jensen's argument against large environmental effects and the paradox implied.[2] The second provides a verbal introduction to our model by way of an analogy that shows how the introduction of television might have caused the large gains in basketball ability apparent in young people today. The third offers a brief account of how the model might work to explain IQ gains. The fourth presents a sequence of mathematical models that show how small environmental effects can have large consequences for IQ. The fifth section discusses the formal model, explores its explanatory potential, and discusses how it might be tested. Our conclusion reviews our findings and contributions.

Heritability Estimates, IQ Gains, and Factor X

Heritability estimates are often interpreted as assigning the dominant role in determining individual differences in IQ to genes, leaving environment with a minor residual role. Yet, massive IQ gains over time signal the existence of environmental factors of enormous potency over periods during which environmental change looks modest. For example, 18-year-old Dutch men tested in 1982 scored 20 IQ points (SD = 15) higher on a test derived from Raven's Progressive Matrices than did 18-year-old Dutch men in 1952. The gain was verified by comparing the scores of a random sample of the 1982 cohort (95% of it took military tests) with the scores of their own fathers (Flynn, 1994).

This last result shows that the main candidate for a genetic explanation could not be playing a significant role in IQ gains because differential reproduction patterns are not involved. The other genetic factor usually mentioned, hybrid vigor or outbreeding, may have played a role in the first half of the century. But the day when most Dutch were mating within small isolated groups is well in the past; certainly, no great change of this sort affected those born in 1954 and 1964, cohorts who show an eight-point IQ gain over only 10 years.

Data from other nations show large gains on a variety of different tests. Indeed, every one of the 20 nations analyzed to date

[2] To the best of our knowledge, Jensen (1973a) was the first to formalize the argument, though others before him and since have made similar arguments. The version of the argument we will present is a modified version of the original (see Footnote 4).

show sizable gains since 1950 (Flynn, 1994), which poses the question: Why do environmental factors explain so little variance in heritability estimates?

The paradox is best seen by considering the argument made by Jensen (1973a, pp. 135–139, 161–173, 186–190; 1973b, pp. 408–412) and more recently adopted by Herrnstein and Murray (1994, pp. 298–299). These authors develop a formal argument based on standard estimates of heritability of IQ that suggests that a purely environmental explanation for the difference in average IQs between Black and White Americans strains credulity. In this article, we are not going to address the question of whether differences in environments can explain the Black–White IQ difference. We believe that a somewhat elaborated version of the model presented here could shed some light on this question, but proper treatment would require us to develop that model and consider a range of evidence tangential to our main point.[3] However, Jensen's logic is equally applicable to differences between generations where environmental change must be the cause.

An application of this argument to IQ changes over time would begin with the assumption that the value of h^2 for IQ has not changed and is about .75. Further, assume that environmental differences causing IQ differences within a generation are the same as those causing differences over time. Finally, assume that environment and genetic endowment are uncorrelated at each point in time. If IQ gains are due solely to environment, then people from an earlier year can be treated as if they were a sample from a later year selected on the basis of an inferior environment for the creation of IQ. Imagine a sample of people alive today whose environmental quality for IQ was 2.00 standard deviations below the population average. If environmental factors account for 25% of IQ variance, this gives a correlation of .5 between environment and IQ. The correlation tells us how far the sample would regress towards the mean IQ of the population for each standard deviation of environmental deficit eliminated. So, a deficit of 2.00 standard deviations of environment would be needed to account for a 1.00 standard deviation IQ deficit.

If anything, this is an underestimate of the environmental deficit needed. As Jensen has pointed out, only a part of the environmental proportion of IQ variance is relevant to between-group differences. If we were to focus solely on the percentage of IQ variance that is both environmental and relevant to group differences, the value might be as low as 10 or 15%. If we put the percentage at, say, 11%, then an environmental deficit of "only" 2.00 standard deviations would be insufficient to account for a 1.00 standard deviation IQ deficit. The square root of .11 is about .33, and 1.00 standard deviation divided by .33 gives 3.00 standard deviations.[4]

Note the implications of this arithmetic: Dutch 18-year-old men gained 20 points (1.33 *SD*s) between 1952 and 1982. By this logic, a minimum of a 2.67 standard deviation gain in environmental quality would be necessary to account for their IQ gains. If we take into account that the passage of time cannot have had much effect on some significant fraction of environmental causes, the necessary gain for relevant environmental factors might be as much or more than 4 standard deviations. So, assuming a normal distribution for environments, the average Dutch man of 1982 must have had an environment whose quality was well into the highest percentile of the 1952 Dutch distribution. This hardly seems plausible if we think about the types of things that might have changed and about their potential impact on IQ taking Jensen's argument as given.

The evidence for IQ gains over time is overwhelming as is the argument for a primarily environmental cause. Thus there must be something wrong with the analysis that suggests that an environmental cause is implausible. A prime candidate is the assumption that the environmental factors operating between the generations are the same as those operating within each generation. This brings us to the notion of Factor X. In the literature, Factor X has been proposed as an explanation for Black–White IQ differences and described as some aspect of the environment that handicaps practically all Blacks and practically no Whites (Lewontin, 1976). If potent, such a Factor X would explain the IQ gap between the races, and because it varies hardly at all within either race, it would make no substantial contribution to IQ variance within races. Therefore, it would be compatible with the low fraction of variance attributed to environment in heritability estimates.

There are problems with the Factor X explanation for Black–White differences (see Flynn, 1980, pp. 56–63), and those problems are clearly insurmountable for a literal Factor X explanation for IQ gains over time. Every plausible factor suggested to explain IQ gains, whether better schooling, better nutrition, altered attitudes to problem solving, smaller families, or the increasing popularity of video games, affected some before others and has a differential impact at any point in time.

One can make the Factor X argument more plausible by modifying it slightly. Effects need not be literally uniform if what is happening is a shift in the mean of the distribution of some

[3] We will return briefly to this issue when we suggest that a slight elaboration of our model can rationalize the results of transracial parenting and adoption studies.

[4] Jensen's (1973a, 1973b) initial version of this argument is somewhat different from what we have presented here. He argued that only sources of between-family environmental variance were candidates for explaining IQ differences between groups. Therefore, he did not use the correlation of all environmental differences with IQ but the correlation of between-family environmental differences with IQ to compute the necessary regression to the mean. A problem with the original version of Jensen's argument is that even though variance may partition neatly into between- and within-family categories, real world causes do not (Turkheimer, 1991). Suppose, for example, that lead paint in a household reduces the IQs of children who eat or breathe the lead. Not every child in the household will inhale or ingest the exact same amount. Thus, the presence of lead paint could produce both within- and between-family differences in IQ, and removing lead from the environment could reduce both sources of variance. Further, there is no reason that group differences in adult IQ cannot be attributed to contemporaneous environmental differences. In fact, evidence and analysis presented later suggest that childhood environmental differences may play a vanishingly small role in explaining differences in individual adult IQ. This is probably why Herrnstein and Murray (1994) adapted Jensen's argument, dropping his distinction and generously considering that both between- and within-family environmental differences could contribute to group differences. Generous because, no doubt, Jensen is correct: Much of what produces "environmental" variance are causes no one could ever hope to manipulate and could not play a major role in explaining IQ gains over time. Jensen (see his discussion, 1998, pp. 445–458) also seems to have moved to the view that it is inappropriate to focus exclusively on between-family effects.

environmental causes over time. A shift in the mean of a distribution can result if some people are affected by a change but others are not. However, that only pushes the problem back to the starting point of Jensen's argument. If we are changing the mean of the distribution of environmental influences, it would appear we would have to change that mean a great deal relative to the existing variance of environmental factors to have a large-enough effect on IQ. It was the very size of that change that we found implausible—and that motivated our consideration of Factor X. Something is wrong with the logic of the argument that heritability estimates imply small environmental effects.

A Sports Analogy

The argument for the weakness of environmental factors just discussed assumes the independence of environment and genetic endowment. Although the possibility that the two are correlated is recognized, the assumption has been that it did not matter whether genes expressed themselves purely through biological mechanisms or through environmentally mediated paths. However, together, the Gene × Environment correlation and the mechanism we believe causes it radically alter the implications of heritability estimates for the potential effects of environment on IQ. Our model of the effects of environment on IQ shows that the potential impact of even very small changes in environment could be very large, even if we accept the largest estimates of the heritability of IQ.

The model is abstract. To make the ideas more concrete, we use a sports analogy. Since 1945, many Americans have turned to basketball over baseball, and basketball skills have escalated enormously. Assume that it could be shown that a reliable measure of basketball-playing ability was highly heritable in the U.S. population (call that measure BP), and that BP showed a substantial upward trend over time. The amount of time people spend playing and practicing basketball has escalated over time, but at any particular time, individual differences for practicing and playing are also large, and by the logic of the argument described in the previous section, those differences could not account for much of the variance in ability. This poses a familiar paradox: The absence of differential reproductive patterns for BP show that genetic causes of BP gains cannot be important, and therefore BP gains must be largely due to environmental factors. However, Jensen-type calculations render environmental hypotheses ludicrous. Either we must posit a mysterious Factor X or an environmental difference between the generations amounting to several standard deviations.

Gene × Environment Correlation: Matching and the Masking Effect

The standard model that poses the paradox assumes that environment and genetic endowment are uncorrelated. Applied to basketball, this implies that good coaching, practicing, preoccupation with basketball, and all other environmental factors that influence performance must be unrelated to whether genes contribute to someone being tall, slim, and well coordinated. For this to be true, players must be selected at random for the varsity basketball team and get the benefits of professional coaching and intense practice, without regard to build, quickness, and degree of interest. Indeed, random assignment must hold at all levels, from informal pickup games on weekends to selection for the NBA or WNBA, and those who hate basketball must participate with the same enthusiasm as basketball fanatics.

This obviously doesn't happen. If someone's genes predispose them to be good at basketball, then somewhat better play alone is likely to lead him or her into an environment supportive of better performance. The match is not perfect, of course, but the tendency is pronounced. To the extent that environmental quality is matched with genetic endowment, there will be a tendency for identical twins to resemble one another for BP because their shared genes make them likely to have environments that are very similar for the production of BP—whether they are raised together or separated at birth. Despite being raised together in the same home, adopted and unadopted siblings may experience very different quality BP environments, ones matched to their differences in genetic endowment. Heritability estimates will credit genes with creating BP differences that would not exist were genes and environment truly uncorrelated—for example, if everyone irrespective of height played only pickup basketball once a month. Here is something that acts as a *mask*. Thanks to the matching of environment and genetic endowment, the standard causal model based on heritability estimates can hide the potency of environmental factors.

Multiplier Effects

Our account of the masking effect might be taken as positing that genes get matched to environments by way of some direct impact, but genes can get matched with environments of corresponding quality only through genetically influenced traits. For example, a father who loves basketball and who has a son with slightly better than average genes for the relevant physical traits is likely to play basketball with his son at an early age, and they are likely to play together more often than most. The son may become a bit better at basketball than others his age and may frequently be an early pick when teams are chosen in the school yard. This makes him feel good, so he begins to prefer basketball to other sports. The extra practice makes him better still, and the better he gets, the more he enjoys basketball. He is far more likely than most to be singled out for membership on a school or recreational team where he will receive expert coaching. Such a young person is likely to become a very good basketball player—much better than he would be if his only distinction was the minor physical and social advantages posited at the outset.

Now imagine what happens if part of the boy's initial advantage is removed. Assume that the boy plays basketball in high school, but also becomes less influenced by his father and discovers an interest in chemistry. He decides he wants to become a chemist, and even though he still loves basketball, he is less focused than are his teammates and spends more time on homework than they do. With less than peak effort and only a moderate biological advantage, he may never be quite good enough to earn a basketball scholarship. In college and adult life, his basketball ability could enter a downward spiral in which he plays less, his skills decay, his enjoyment diminishes, and he plays less still.

This analogy illustrates several things. First, it demonstrates an important feature of the matching process. The boy enjoyed several environmental advantages *as a result of his initially higher*

ability. People who are born with a genetic advantage are likely to enjoy an environmental advantage as a result—though in our analogy there was a confounding of an initial genetic and environmental advantage (the father's interest in basketball). The genetic advantage may itself be rather small. However, through the interplay between ability and environment, the advantage can evolve into something far more potent. So we have found something that acts as a *multiplier*: The process by which the ability of an individual and the environment of an individual are matched can increase the influence of any initial difference in ability—whether its source is genetic or environmental.

High ability may not always be matched to a better environment than low ability. Sometimes being bad at something may lead to an enriched environment. A person with a physical disability may be put in a special program with intense individual instruction to help him or her overcome that disability. However, we are convinced that the positive matching of ability and environment dominates in nearly all circumstances having to do with either basketball or IQ.

Although the boy in our analogy profited from both good genes and environment, the advantage could have been environmental only—the fact that his father enjoyed basketball. Our analogy shows that *anything* that makes someone better at something improves skills that improve environment that improves skills and so forth. Our analogy also shows that there can be a *de-escalation* once the original environmental advantage is removed. Once the boy escaped the ongoing influence and encouragement of his father and withdrew some of his interest from basketball, his skills deteriorated, which led to a further loss of interest and a further drop in skills. This second point will prove significant when we address some problems in the IQ literature: why the effects of intervention and adoption on IQ diminish, why the stability of IQ increases, and why the heritability of IQ increases with age.

Our focus on an individual's life history means that we have not yet addressed the central problem: changes in the average level of basketball playing ability in the population. One solution to the problem might be to assume some change (e.g., that TV popularized basketball) caused a small increase, on average, in people's interest in basketball. Such a change might kick off multiplier effects at the individual level that for many individuals snowball into very large changes in ability. However, imagine a society in which many individuals are playing more basketball and getting better at it. If the mean BP of the whole population rises, this will eventually have an enormous impact on the basketball milieu—on the quality of interaction between players, the interaction between players and coaches, the interaction between coaches, and so forth. In other words, even a modest rise in group mean BP can boost the group mean further, and that boost can mean a further boost. We call this process the *social multiplier*. Although not necessary for our explanation of IQ gains over time, it qualitatively changes the model so that it becomes easier to provide an explanation.

Averaging Transient Environmental Factors

We now shift our attention away from matching, or the process by which environmental factors become correlated with genetic endowment, toward the environmental factors that are not correlated with genetic endowment: the ones that cause the residual variation in BP after the direct and indirect effects of genetic endowment are factored out. For huge BP gains over time to occur, there must be some new environmental factor, also uncorrelated with genes and ability, that proves potent, such as TV enhancing the popularity of basketball. When we say it is potent, we mean that it is very powerful compared with the environmental influences producing the cross-sectional environmental variance that we observe. The reason it is potent has to do with persistence over time and across individuals. It is a consistent environmental influence, whereas many other environmental factors are relatively fickle. Only a consistent environmental factor can rival the potency of genes; after all, our genetic endowment is always with us.[5]

Why do most uncorrelated environmental factors have little persistent or cumulative effect? A father may play basketball with a son one year, but his job may not leave him time for it the next year. In any case, most children go through phases where their interests shift from one pursuit to another, and nearly all boys eventually outgrow playing basketball with their dads and move on to other pursuits.

We posit that a person's ability at any point in time depends on a sort of *average* of all the environmental influences, both good and bad, that have contributed to the total effect of environment on ability over time. That entails something about variance. Purely environmentally induced variance (that part measured by $1 - h^2$) reflects how the average of changing environmental factors over time varies from individual to individual. At each point in time, each individual is subject to some environmental influences that have not been induced by that person's genetic influences. We will refer to these as exogenous environmental influences. Many of these influences will change over time. Treat these experiences as draws from a distribution of possible environmental influences and assume that current ability is affected by an average of current and past draws from that distribution. Then, as the number of experiences being averaged increases, the ratio of the variance of exogenous environmental effects on current ability across individuals to the variance of the underlying experiences will become smaller. This has important implications for the paradox we are considering. It means that if exogenous environmental effects are the average of a sufficiently large number of different experiences over time, a shift in the mean of the distribution of those experiences that is small relative to the variance of the distribution of those influences could be large relative to the distribution of environmental effects on ability across people.

Consider what happens when we go from single flips of a coin to a long sequence of flips. Assign values of 2 to heads and 1 to tails. If we flip the coin a number of times and record either a 1 or a 2 depending on the outcome, the variance of each flip around a mean of 1.5 is .25 and the standard deviation is .5. However, if we compare several sequences of 100 flips, the variance of the mean of each sequence will be much less than that of a single flip. The odds against even one sequence having a mean of either 2.0 or 1.0

[5] Of course, any particular genetic effect may not be constant or even present at all ages. However, we suspect that at least some of the contribution of genes to differences in intelligence is due to physiological differences that they induce, and that those differences are far more constant across a person's life than nearly all environmental influences.

become astronomical. The variance of the sample mean of 100 flips will be .0025 for a standard deviation of .05.

Now suppose we add a persistent environmental factor, even a rather weak one, to a collection of transient factors. In the coin analogy, we will equate this with raising the value of heads from 2.0 to 2.1 and tails from 1.0 to 1.1. It may look as if this would make little difference because the expected average only rises from 1.5 to 1.6. The gain is small compared to the standard deviation of a single flip (0.1 is one fifth of 0.5). But note how large the gain is compared to the standard deviation of the average of 100 flips; the 0.1 added to the mean is twice the standard deviation of 0.05.

The impact of one persistent factor on an average of many changing factors is relevant to our basketball analogy. Assume there are environmental factors uncorrelated with genes and not caused by an individual's ability that affect a person's ability to play basketball. Assume they are constantly changing. Then, at any point in time, a persistent environmental factor that raises the mean of the distribution of transient factors can have a very large impact compared to the standard deviation of individual differences induced by exogenous environmental influences. At any given time, the "fathers playing with sons" environmental factor, taken as one transient factor in a complex of many, is weak. But if your father plays basketball with you every day for 10 years, that becomes a big environmental influence.

Averaging and the Social Multiplier

If averaging over time is potentially important, how might it occur? First, the direct effect of environment on performance will extend beyond the environment at a particular instant and will include environmental influences that have impacted over some period of time before that instant. Second, today's ability and today's environment are correlated, because the better you are at basketball, the more likely you are to be doing something today to improve your skills. So the fact that past environmental influences have affected today's ability makes today's environment a sort of weighted average of all environments experienced in the past. Third, each individual's basketball ability will be affected by the BP of other people. By definition, any enhancement of individual BP raises the population's mean BP and through social interaction that may raise each individual's BP. This collective averaging further diminishes the importance of random individual environmental influences, whereas consistent factors acquire an impact beyond what we would expect viewing the individual in isolation.

The social multiplier provides the last piece in the puzzle of huge basketball-performance gains over time. We will choose a plausible starting point, 1950, as the year that TV viewing became widespread in America. Before that time, baseball was dominant. But the big baseball park lost something when transferred to the small screen, whereas the confined basketball court fit easily. The growth of TV served as a trigger, a causal factor significant enough to shift attitudes, and the attitude shift eventually fueled a social multiplier. The televised games caused many people to take basketball more seriously, and the technique of the best professionals reached into every home. Playing basketball became a more common after-school and weekend activity, beginning young and extending into middle age. The effect on any one person might have been modest, except for the fact that a lot of people were getting interested. That made it easier to find pickup games, the players took them more seriously, and each person's skill escalation gave others something they could imitate and something they could try to match. People started being able to shoot with either hand and make slam dunks, coaches began to expect these skills, schools hired better coaches, lots of people began to watch basketball, and lots of people began to applaud those with good skills. Through such a process, the introduction of television could reasonably be seen as the cause of the massive gains, even though how much basketball people watched was only weakly correlated with their ability (controlling for their genetic endowment).

IQ Gains Over Time

How do the concepts developed in our basketball analogy transfer to explaining IQ gains over time? The potency of environmental factors between generations is undeniable. Why does environment seem such a weak cause of differences when existing heritability estimates are used to deduce their size effects in the context of the standard analysis described in the first section of this article? We assume that within a generation, there is a high correlation between favorable genes for IQ and quality of environment. Support for this assumption can be found in Jensen's recent summary of the heritability literature (1998, pp. 179–181) in which he traces the almost complete collapse of the between-family percentage of IQ variance as people age. As he says, the most parsimonious conclusion is that as people mature and the influence of family wanes, there emerges a match between genes and environmental quality. In addition, recall the familiar correlations between IQ and income, occupational status, years of education, and other factors, all of which are indexes of environmental quality. Much of Herrnstein and Murray's (1994) book *The Bell Curve* was devoted to demonstrating the existence and importance of these correlations. The match between genes and environment means that environmental factors, however potent, to a large extent just reinforce the advantage or disadvantage that genes confer. So the match *masks* the potency of environmental factors.

Between generations, the mask slips. For it to do its work, the worse environment of the earlier generation would have to be matched by worse genes for IQ; and the better environment of the later generation would have to be matched by better genes for IQ. However, because the two generations are equivalent for genes, there is no matching and therefore no masking. The potency of environmental factors stands out in bold relief.

Now let us focus on how environmental factors that are uncorrelated with genes and ability impact on people. One's IQ reflects in part the *average* of many past environmental influences. Once again, this averaging means that factors that both raise the mean IQ and persist over time can loom large compared to the standard deviation of an index of environmental effects. Note that given the structure of the argument, it is not necessary for there to be a single persistent factor to get this effect. A multiplicity of factors that raise the mean of the distribution of environmental influences will be equivalent to adding a constant positive environmental effect. Thus, we need not be looking for changes that are persistent across all individuals at all points in time. We need only find factors that

have changed the mean of the distribution of environmental effects.

We can suggest several possible triggers of this sort. However, we want to emphasize that we cannot, at present, show that any of the specific triggers we suggest have actually contributed to IQ gains over time; and we accept that there may well be triggering factors not on our list. Our purpose is merely to suggest some possibilities, so readers can understand how the model *might* apply.

Thanks to industrialization, it is likely that the cognitive complexity of the average person's job has increased over the last century. There is no doubt that more-demanding educational credentials control access to a wide range of jobs. There are far more people in scientific, managerial, and technical positions than ever before.[6] Increased leisure time is another possible trigger for IQ gains, as some activities undertaken during extended leisure (reading, puzzles, games such as chess) may be honing people's facilities.[7] Radio and television may be factors. It is possible that the machinery we increasingly surround ourselves with (e.g., cars, phones, computers, and VCRs) have increased the demands on our cognitive capacities. The shift to fewer children in each family, affording more time to cater to children's curiosity and richer individual interactions, may have played a role. Some or all of these may have contributed to a significant attitude shift: The current generation may take abstract problem solving far more seriously than preceding generations did.

The direct effects of these changes need not be large. But because they are widespread and persistent trends, they could loom large relative to the many less-constant environmental influences that produce most differences between people. Further, once whatever triggers the process gets it going, the individual and social multipliers begin their work. A person whose school, job, and gadgetry are more cognitively demanding than those of his or her parents may choose more-demanding leisure activities and a more-intelligent spouse. This may boost his or her IQ further, leading to further improvements in the personal environment that the individual creates. At the same time, other people are coping with more-demanding environments as well. So, their cognitive abilities rise and that affects their lives. With many people experiencing rising cognitive ability, the cognitive quality of social interaction escalates. Lunch conversations, family chat, and game and puzzle play move up a notch in sophistication. These social changes produce further changes in individuals, which again work their way through the individual multiplier. In the end, the small initial triggers can produce a huge cumulative change in mean IQ. Everyone will, of course, have their favorite list of triggers. More research is necessary to identify the actual chain of causation.

Three Formal Models

Our models give formal statement to our four key concepts: how the matching of genetic endowment and environment produces a Gene × Environment correlation that can mask environmental effects; how the process that produces matching can act as a multiplier of environmental influence; the significance of the fact that the environmental influence on IQ is the average of a number of environmental effects; and the enormous potential of the social multiplier. All four are embedded in our most elaborate model. However, as an aid to presentation, we will use two simpler models that clarify some of our key concepts without the complexity of the final version.

Model 1: Matching and Masking Environmental Effects

The following linear model can be used to decompose the variance of phenotypic IQ:

$$M_j = aG_j + vE_j. \quad (1)$$

M_j is the measured intelligence of person j, G_j is that person's genetic endowment, and E_j is a measure of how conducive person j's environment is to the development of IQ. The model does not divide E into between-family environment and within-family environment as is usually done.[8]

The coefficients a and v represent the impact of genes and environment on test scores. Now, if M_j and G_j and E_j are all measured in terms of standard deviations from their means, and if G and E are uncorrelated, the correlation of G or E with M will be the coefficient of that variable (a and v, respectively). Also, the square of the correlation coefficient will be the fraction of variance in M explained by the variable.

In other words, if we interpret Equation 1 as a causal model of the process generating IQ and make the assumption that G and E are uncorrelated, then the logic of Jensen's or Herrnstein and Murray's argument is inescapable—it will take a huge change in E, measured in standard deviations, to produce the 1.33 standard deviation change in M (mean IQ) that occurred in the Netherlands. However, the assumption that G and E are uncorrelated is clearly false. All parties to this discussion seem to agree on this.

Therefore, we need to take the next step and ask how the interpretation of Equation 1 changes if G and E are correlated. To answer that question, we write the following equation for the environmental effect on a person's test score (E_j):

$$E_j = rG_j + e_j. \quad (2)$$

The environmental effect is equal to the correlation between individuals' genetic endowments and their environments (r) times their genetic endowments (G_j)—plus a term (e_j) for environmental factors causing a mismatch between genes and environment.

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[6] There is a large literature in sociology questioning whether jobs actually have become more complex. Our reading of summaries such as Spenner (1983) is that even though it is hard to judge whether work has become more autonomous, and examples of individual occupations that have become less cognitively demanding can be found, the change in the occupational composition toward jobs requiring more cognitive skills (as evidenced by their educational requirements) dominates any changes within occupations. Strong evidence of increasing demand for cognitive skills is the stable or increasing return to education in the labor market in the face of a great increase in the supply of educated labor over the last century and particularly in the last several decades (see Burtless and Pierce, 1996).

[7] Schooler and Mulatu (in press) present evidence that leisure time activities affect cognitive skills.

[8] This is because we do not believe that this dichotomy coincides with the dichotomy between group-difference-relevant E and group-difference-irrelevant E. See our discussion of this issue in Footnote 4.

Equivalently, e_j represents environmental influences that are uncorrelated with genetic endowment (G_j) because they are in no sense caused by it. They are the exogenous environmental influences we discussed in the last section. Figure 1 illustrates the path model implied by Equations 1 and 2.[9]

We can then substitute the right-hand side of Equation 2 for the term E_j in Equation 1, which gives

$$M_j = aG_j + v(rG_j + e_j) = (a + vr)G_j + ve_j. \quad (3)$$

Because e_j and G_j are uncorrelated by definition, Equation 3 satisfies the requirements for a unique decomposition of the variance of IQ. Thus the coefficient of G_j must equal the correlation of genetic endowment with IQ (h). The square of the coefficient will be h^2, or heritability, or the fraction of IQ variance explained by genes.

Equation 3 reveals that the correlation of genetic endowment and IQ implied by heritability estimates will be equal to the sum of the direct effect of genes on intelligence (a) *plus* the impact of environment (v) times the correlation of genes and environment (r). In other words, Equation 3 reveals that the matching of genes and environment dictates that genes get credit for some of the work that is actually being done by the environment. The more correlated that genes and environment are, the greater the misattribution.

Equation 3 provides an antidote to the misleading implications drawn from the analysis of Equation 1 under the false assumption that G and E are uncorrelated. Equation 3 shows that it is the variance of ve_j plus heritability that must equal one. Since ve_j is not the full impact of environment, but rather is the part that is due to whatever mismatch there may be between people's genes and their environment, it follows that the total impact of environment may be much larger than the impact of that part alone.

Looking back to Equation 2, and assuming that we continue to measure environment in terms of standard deviations from its mean in the population, the variance of $rG + e$ must equal one. Therefore, the variance of e must be $1 - r^2$, because the variance of G has been assumed to be one. This yields our next equation:

$$1 = (a + vr)^2 + v^2 \operatorname{Var}(e) = (a + vr)^2 + v^2(1 - r^2). \quad (4)$$

Equation 4 makes it clear that the impact of environment (v) need not have an upper limit equal to the square root of one minus heritability ($1 - h^2$)—because the variance of the factors causing mismatch between genes and environment (e) is not assumed to equal one. In fact, it shows that the direct effect of genetic endowment (a) plus the term that arises from the correlation of genes and environment (vr) equals the correlation of genetic endowment and IQ. The correlation of genetic endowment and IQ is h (the square root of h^2 or heritability), so $h = a + vr$. Therefore, Equation 4 gives the following:

$$v = \sqrt{\frac{(1 - h^2)}{(1 - r^2)}}. \quad (5)$$

Assuming Equations 1 and 2 reflect causal processes, Equation 5 provides a measure of the impact on IQ of a 1.00 standard deviation change in environment (E_j). It is equal to the square root of one minus heritability divided by one minus the squared correlation of genes and environment. That correlation could conceivably have any value from zero to the square root of heritability (h). Therefore, the impact of a 1.00 standard deviation change in environment (v) would have the square root of one minus heritability as its lower limit,[10] but its upper limit would be one. The value for v cannot go higher than one because that would imply the impossible, namely that the variance of IQ measured in terms of standard deviations from its mean was greater than one.

Equation 5 also shows that the upper limit of environmental impact is approached as r (the correlation between genes and environment) approaches h (the correlation between genes and IQ). The correlation r can approach this upper limit, but it cannot reach it. Looking back to Equations 4 and 5, $r = h$ only if $(vr)^2 = h^2$ or heritability; and $(vr)^2 = h^2$ only if a (the direct impact of genetic endowment) equals zero (because from Equation 5, $v = 1$ in this case). In sum, environmental impact reaches its upper limit only if the correlation between genes and IQ is entirely due to genes matching environments and if genes have no direct effect on IQ. In the context of our model, this is impossible, so a value of one for v represents an upper bound that cannot be reached.[11]

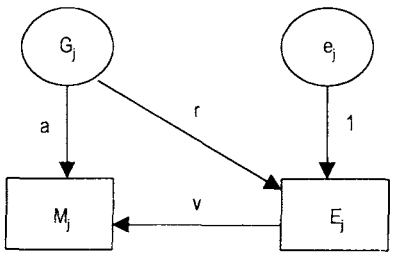

Figure 1. Static model with $G \times E$ correlation (Model 1). G_j = genetic endowment of person j; M_j = measured IQ of person j; E_j = environment of person j; e_j = exogenous environmental influences on person j.

[9] Note that we have assumed that genes cause environment but that environment does not cause genes. It is certainly possible that environmental differences between populations could cause selection that would lead to Gene × Environment correlation. More important for the arguments we are making here, it is also possible that Gene × Environment interaction could cause certain genes to be expressed only in certain environments, and this could be interpreted as environment causing genes. We do not deny that such mechanisms could be at work, but we assume them away to simplify our presentation and help focus the analysis on the effects with which we are mainly concerned.

[10] Assuming genes and environment are not negatively correlated.

[11] Several readers of earlier drafts of this paper have noted that the "direct effect of genes on IQ" may very well be zero, in that there are certainly no genes for IQ scores. This is an overly literal interpretation of what we mean by a direct effect of genes on IQ. We understand a direct effect to be one in which genes predispose one to have some physical characteristic that is useful for performance on IQ tests. We concede that it is possible that even in this sense there may be no direct effects of genes on IQ. For example, it is possible that the genetic effect on IQ arises entirely because some people's genes make them less able in areas other than intellectual pursuits, this causes them to eschew other pursuits in favor of a rich intellectual environment, and this is what causes their IQ to be higher. To model such a process we could add a term for genetic determination of those aspects of environment conducive to IQ (Equation 2). Doing so would complicate the analysis, but would not substantively alter the conclusions we draw from the model.

Recall that the Dutch gained 1.33 standard deviations on Raven's Progressive Matrices between 1952 and 1982. The Jensen and Herrnstein and Murray reasoning implied the need for an environmental change of at least 2.67 standard deviations. Since multipliers approaching one are possible, the environmental shift necessary might be only slightly greater than the 1.33 standard deviations of the change in IQ.

Still, that we must posit an environmental shift of at least 1.33 standard deviations is not reassuring. A table of areas under a normal curve tells us that a shift only slightly larger than that would put more than 90% of the 1982 Dutch above the average environment of the 1952 Dutch. In addition, the environmental gain of 1.33 standard deviations refers to all environmental sources of IQ variance, and some of these are unlikely to alter much over time. Therefore, the change in the kinds of environmental factors likely to differentiate two generations would have to be greater still. Clearly, we are only part of the way to a plausible environmental explanation of massive IQ gains over time. We still need to transcend the upper limit of environmental impact imposed by Equation 5.

Model 2: Matching as a Multiplier of Environmental Influence

We have posited a tendency toward a matching of genes and environment. This section will show how such matching might take place and why it can give enormous leverage to exogenous environmental differences. Our Equations 1 and 2 give no account of how genes and environment come to be matched. Therefore, we rewrite them as follows:

$$M_{jt} = aG_j + vE_{jt-1} \quad (1')$$

and

$$E_{jt} = bM_{jt} + (u + e_j). \quad (2')$$

Equation 1′ is identical to Equation 1 except that we have given IQ and environment subscripts to indicate that they change over time. Today's IQ is shaped by one's genetic endowment (G_j) and past environment (E_{jt-1}). We continue to measure M, G, and E in terms of standard deviations from their respective means. Equation 2′ now specifies that environment is determined by IQ times the impact of IQ on environment (b) plus a term that allows for differences in the extent of mismatch between IQ and environment ($u + e_j$). The variable e_j still represents environmental factors uncorrelated with genetic endowment unique to individual j that influence IQ. Technically, we imagine that it is a mean zero random variable so that the sum of exogenous factors affecting E_{jt} is a random variable with a mean of u. Figure 2 presents the path diagram implied by Equations 1′ and 2′.

What happens to the average IQ if u changes? Initially, there will be a rise in each individual's IQ equal to v (the impact of environment on IQ) times the change in u; of course, in reality, change need not be uniform. However, just as environment impacts on IQ, so IQ impacts on environment. Therefore, the new higher IQ will in turn enhance the quality of environment. This environmental enhancement will be equal to b (the impact of IQ on environment) times v (the impact of environment on IQ) times the

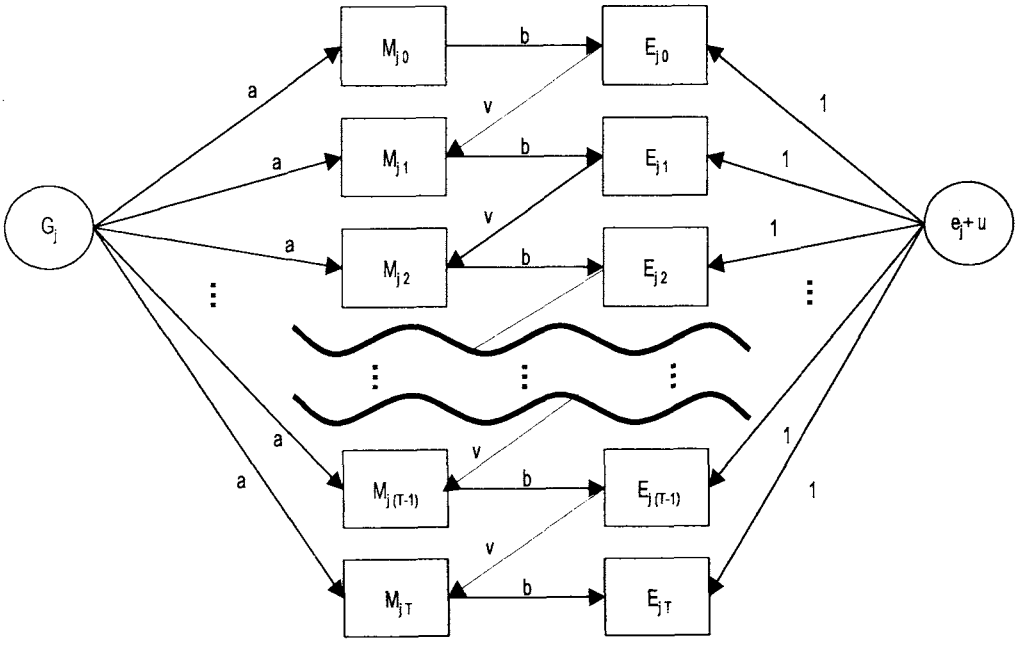

Figure 2. Model of dynamic phenotype–environment interaction leading to $G \times E$ correlation (Model 2). G_j = genetic endowment of person j; M_{jt} = measured IQ of person j at time t; E_{jt} = environment of person j at time t; $e_j + u$ = exogenous environmental influences on person j.

change in u. The process will be both repetitive and cumulative. Ultimately, the rise in IQ will approach the value of the infinite sum: $\Delta u(v + bv^2 + b^2v^3 + b^3v^4 + \cdots)$.

If the impact of IQ on environment and the impact of environment on IQ were sufficiently large, a change in the mean of the distribution of exogenous environmental influences (u) could produce an upward or downward spiral of IQ that would be infinitely large in absolute value. However, plausibility requires a limit. Let us assume that the product of the effect of environment on IQ times the effect of IQ on environment is positive but less than one. Then, even as the process goes on forever, IQ gains or losses will tend toward an upper or lower limit.

The upper or lower limit can be found by solving the difference equation implicit in Equations 1′ and 2′. We will substitute Equation 2′ into 1′, assume that $M_{jt} = M_{jt-1}$ in the long run as the process runs its course, and solve for M_j. That gives us a new equation for IQ,

$$M_j = \frac{aG_j}{1 - bv} + \frac{v(u + e_j)}{1 - bv}. \quad (6)$$

As we know from Equations 1′ and 2′, if we change the mean of exogenous environmental influences (u), then the initial impact on IQ is a change of v times the size of the change in u. However, that change in IQ causes further changes to environment, and those changes in environment cause further changes in IQ. In the end, a one-unit increase in u—that is, a change sufficient to increase E (environment) by 1.00 standard deviation before any multiplier effects—will produce a $v/(1 - bv)$ standard deviation increase in IQ. This is the sum of the infinite series described previously.

How large could the IQ shift be? Assuming that G and e are uncorrelated, Equation 6 can be interpreted in the same way Equation 1 was with respect to the contribution of genes and environment to IQ variance. The variance of the first term will be equal to the fraction of variance explained by genetic endowment or h^2 and this equals $a^2/(1 - bv)^2$. The variance of the second term will be $1 - h^2$ and this equals $v^2\,\mathrm{Var}(e_j)/(1 - bv)^2$. Further, we can substitute Equation 6 into Equation 2′ to get

$$E_{jt} = \frac{baG_j + bv(u + e_j)}{1 - bv} + (u + e_j) = \frac{baG_j + (u + e_j)}{1 - bv}. \quad (7)$$

We now wish to determine the size of v or the direct impact of environment on IQ. The details are spelled out in the Appendix. However, the logic of the derivation runs as follows: Under the assumption that the variance of all environmental influences (E) is one, we can use Equation 7 and the two conditions on the variance of IQ (M) from Equation 6 to derive the following:

$$v = \sqrt{(1 - h^2) + (h - a)^2}. \quad (8)$$

Equation 8 entails a familiar conclusion: The direct impact of environment on IQ (v) approaches its maximum value of one as the direct effect of genetic endowment on IQ (a) approaches zero. Since $1/(1 - bv) = h/a$ (see the Appendix), we can write

$$\frac{dM}{du} = \frac{v}{(1 - bv)} = \sqrt{(1 - h^2) + (h - a)^2}\,\frac{h}{a}. \quad (9)$$

From Equation 6, a one-unit change in u will cause a change in M of $v/(1 - bv)$. Equation 9 shows that as the direct effect of genetic endowment on IQ (a) approaches zero, the effect of changing u goes to infinity.

Once again, putting the direct effect of genetic endowment at zero or even close to zero is implausible in the context of this model. However, the fact that we broke the stricture that held the upper limit of the impact of environmental change at one and now have an upper limit of infinity has a dramatic effect. When the stricture held, setting the direct effect of genetic endowment on IQ (a) at .2 and assuming heritability of .75 implied that an initial 1.00 standard deviation change in environment would cause a 0.84 standard deviation change in IQ. Now, those same assumptions imply that the impact of increasing the mean of exogenous environmental influences (u) by 1.00 standard deviation will be a 3.6 standard deviation increase in IQ. Table 1 shows different values of this multiplier corresponding to different assumptions about the magnitude of the direct effect of genetic endowment on IQ, assuming $h^2 = .75$.

Figure 3 presents a simulation of Model 2. We take an individual with an average genetic endowment ($G = 0$), an initially average environment (u, e, and $E = 0$), and a value for a of .3. We show what happens when, in the sixth time period, e is increased from 0 to 0.5, and then what happens when in the 21st time period it is reduced back to 0. One can see how initially E rises by 0.5 and how that causes M to rise by a fraction of that amount in the next period. The rise in M causes a further, but smaller rise in E, which in turn causes yet another rise in M. As the process continues, M slowly approaches its new equilibrium value of 1.1. In period 21, the initial cause of the increase in measured intelligence is removed and the process reverses itself.

Note that the induced increase in total environment at its peak is large (1.5 standard deviations). And we can now explain why we are deceived into thinking that large increases in environment are implausible. Our intuition about what constitutes a large increase is shaped by perceptions of what sorts of *exogenous* changes have taken place in society. What we have demonstrated here is that relatively small exogenous changes in environment—those consistent with intuitions about the potential magnitude of such changes—can have large effects on both total environment and IQ.

Can we now explain the IQ gains of the Dutch and others? Assume that Dutch society evolved to impose on the average person of 1982 an exogenous environmental improvement of 0.5

Table 1
Multipliers Implied by Model 2 for Different Values of the Direct Effect of Genetic Endowment

Coefficient of genetic endowment (a)	Environmental multiplier ($v/(1 - bv)$)
.10	7.9
.15	5.0
.20	3.6
.25	2.7
.30	2.2
.40	1.5
.60	0.8
.80	0.5

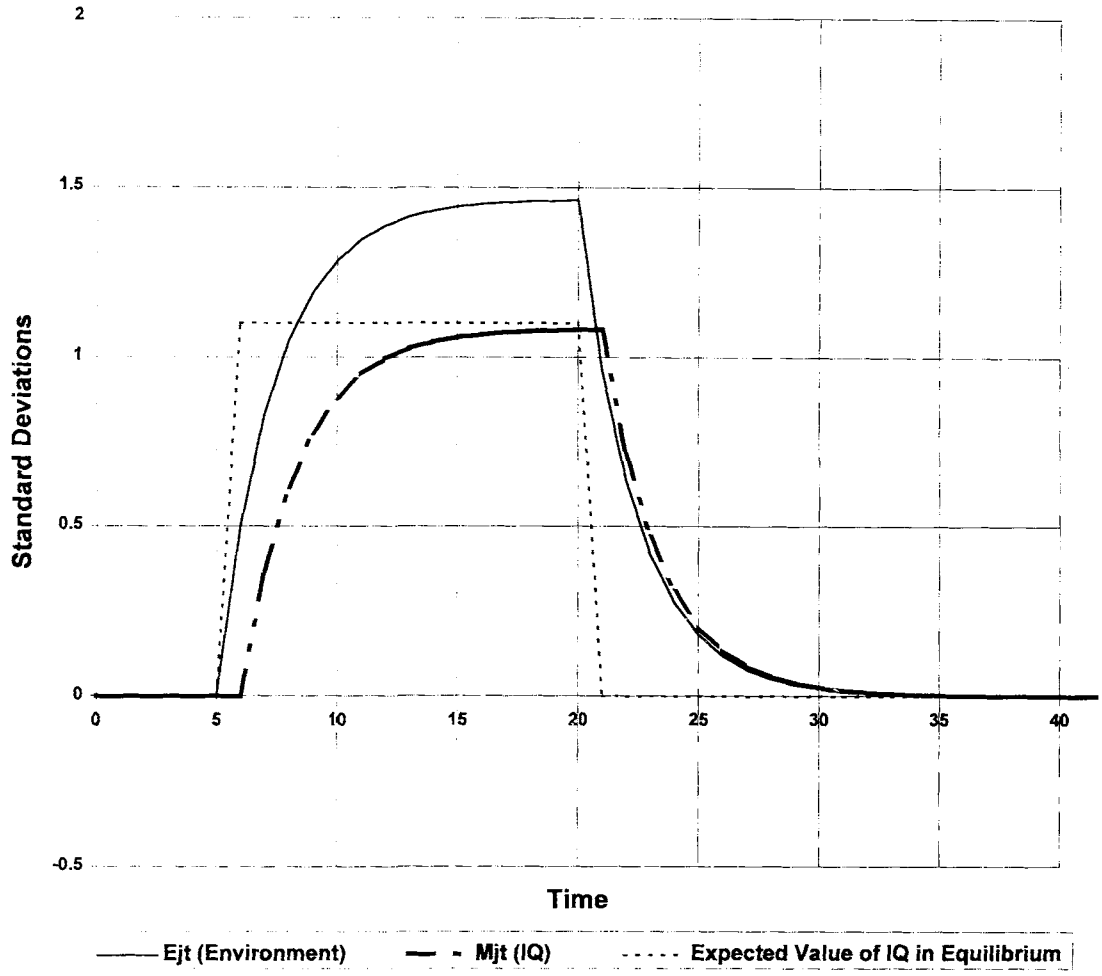

Figure 3. Simulated history for environment and IQ (Model 2, average person with multiplier of 2.2).

standard deviations over the average environment of 1952. Setting a at .25, the multiplier gives a 1.35 standard deviation IQ shift. This is enough to explain the 20-point IQ gain the Dutch enjoyed.

Or is it? Thus far, we have been measuring the exogenous change in environment in terms of standard deviations of the total environmental influence (E) on IQ. Is that the appropriate metric, or should we be measuring changes relative to the variance of exogenous environmental influences ($u + e$), which could be much smaller than the variance of E? It is easy to imagine that a change in u that was small relative to the variance of E could be large relative to the variance of e.

Up to now, we have been vague about what we mean by E because we want it to capture a wide range of effects. We have imagined it as encompassing everything from the cognitive demands of one's job to how individuals internally react to any given environment. We cannot be sure how important influences like the former are relative to the latter. Imagine an extreme case in which the only aspect of total environment (E) that matters for IQ is differences between people in the extent to which they set difficult mental problems for themselves and persevere in solving those problems. Suppose that exogenous environment ($u + e$) has little direct effect on IQ and that all exogenous differences in environment (e) are due to differences between people in the quality of their nutrition—because nutrition influences their ability to concentrate and therefore has some small effect on the intensity with which they undertake mental problem solving. Suppose we wanted to argue that it was a change in nutrition that caused IQ gains over time. If we knew all the parameters of our model, we could compute the magnitude of the change in nutrition that would be necessary to produce the observed gains in IQ over time. Measuring nutrition in terms of its initial impact on IQ, the magnitude of the change could be quite small compared to the variance of total environment because the latter is mainly due to differences in the extent to which people mentally challenge themselves. On the other hand, the necessary change in nutrition could be huge relative to the cross-sectional variance of exogenous environmental influences—differences in nutrition. If this were the case, we might well conclude that improved nutrition was not an adequate explanation of IQ gains. Even when augmented by the multiplier effects we have described so far, the magnitude of the improvement would have to be far too large relative to the variance of nutrition in the population.

In effect, the above assumes a total compartmentalization between E and e. Only internal self-created factors make up E; none of the variation in the exogenous elements of environment ($u + e$) are of this nature. Yet, it is only through changing exogenous environment that environmental factors outside of the person can affect E. If that were the case, it would make no sense to measure changes in u relative to the variance of E. A change in u that was small relative to E could be huge relative to the variance of the exogenous factors (e). Therefore, as a cause of IQ gains over time, it would appear, given only Model 2, to be completely implausible.

Now imagine an alternative extreme case. Suppose that the variance in E is due entirely to differences in the sorts of external factors that we imagine as having changed over time, such as the degree to which doing a job is cognitively demanding, how stimulating available leisure activities are, the intellectual quality of social interaction, or shared attitudes to abstract problem solving. In this case, e (exogenous environmental influences) represents small random factors causing a mismatch between people and the external environmental stimuli to which they are exposed. A change in u that was small relative to the variance of total environment (E) but large relative to the exogenous differences in environment (e) could still provide a plausible explanation for the change in IQ over time. It would be entirely appropriate to measure the magnitude of changes in the mean of exogenous environmental influences (u) relative to the variance of total environment (E), rather than measuring them relative to the variance of exogenous environmental influences ($u + e$).

So, if one believes that total environmental influences (E) as described in our model are mainly external and that the factors that might have triggered IQ changes are representative of the factors that cause E to differ between people, then we are done. But if one believes that total environmental influences mainly reflect differences in how people react to their objective environments, then total environmental influences are not amenable to change, except through the indirect influence of other external environmental influences, and we do not have a complete story. We suspect that the environmental influences that matter for individuals are a blending of the two extremes we have described. Therefore, we think it appropriate to proceed to our third model. It will demonstrate large effects on IQ for environmental influences that are small not only when compared to total environmental influences (E) but also relative to individual differences in exogenous environmental influences (e).

Model 3: Averaging and the Social Multiplier

In the next model, individuals get a new value for e at each point in time instead of keeping the same e as in the last model. The averaging of these es in the production of IQ will allow the effect of environment to be large relative to the standard deviation of the distribution of e at any point in time. How does averaging take place?

First, there are a range of environmental influences that have *directly* affected current IQ. Today's IQ will reflect a sort of average of those effects. Second, to the extent that environment and IQ affect each other over time, then IQ at each point in time will be an *indirect* averaging of that history. Third, to the extent that an individual's IQ is affected by the IQs of others, each individual's IQ will average not only his or her own outside environmental influences but also the exogenous environmental influences affecting others.

These three effects can be captured by rewriting Equations 1′ and 2′ as follows:

$$M_{jt} = aG_j + v(E_{jt-1} + wE_{jt-2} + w^2E_{jt-3} + \cdots + w^tE_{j0})$$

$$= aG_j + v\sum_{i=1}^{t} w^{i-1}E_{jt-i} \qquad (1'')$$

$$E_{jt} = bM_{jt} + cP_t + (u_t + e_{jt}). \qquad (2'')$$

The new parameter w is assumed to have a value between zero and one, so today's IQ is influenced by a geometrically declining weighted sum of all past environmental influences. The impact of past environments will decline slowly if w is close to one and quickly if it is much less than one. We choose this form for the effects of environment because of its analytic convenience. We do not know what the correct functional form is, but we doubt that choosing a different functional form would substantively affect our results. We assume that the individual in question (j) was born during time period 0. The variable P represents the value of the average IQ of the population. The term in parentheses in Equation 1″ captures the first of the averaging effects previously discussed. We are now expressing today's IQ as a function of a weighted sum of past environmental influences. The term bM_{jt} in Equation 2″ retains the assumption that current IQ affects current environment and thereby allows for our second averaging effect. If we substituted Equation 2″ for the Es in Equation 1″, we would get an expression for today's IQ as a function of genetic endowment, average IQ in society, past values of the exogenous environmental influences (e), and past values of M. If we then substituted our new expression for every M on the right-hand side of this new equation, and continued to do that, we would get a complicated sum of past values of the average IQ in society, genetic endowment, and past outside environmental influences. This sum captures our second kind of averaging (of past environmental influences) and shows how it differs from the first.

The term cP in Equation 2″ captures the third averaging effect. P represents the average IQ of a population. Assuming that c is greater than zero, this term allows the average IQ of a person's society to influence that person's environment, which in turn influences the maintenance and development of his or her IQ. This would be true, for example, if the amount of cognitive stimulation that one received depended on the IQ of the people one encountered. Given the way society is structured, people are more likely to encounter others with similar IQs than those whose IQs are very different, but it is harder for people with above-average IQs to find someone with an IQ above theirs than one below theirs. Thus, those with high IQs tend to encounter people whose IQs are intermediate between theirs and the societal average; the same will be true for those with low IQs for much the same reasons.[12] Therefore, everyone is affected by the societal average. The average IQ will include an average of all the environmental effects of

[12] A formal derivation of the presence of the average IQ of a person's social group in the function for his or her environmental influences is available as a typescript from the authors.

all of society's members. By including it, we add a new dimension to the notion that today's IQ is an averaging of many individual time-specific environmental influences.

Including the average IQ of society in the determination of individual environment also introduces a new kind of multiplier effect. An exogenous change that raises the IQs of some members of society by definition increases the average IQ in society and therefore improves other individuals' environments. This acts to increase their IQs—which further increases the average IQ—which further improves the average environment.

Now, if we imagine a particular person affected by an outside environmental influence, and that influence is the sole catalyst that enhances society-wide IQ, the effects must be small. But what if the *average* value of some outside environmental influence affecting a group changes and all members of the group begin reinforcing one another? Then the combination of social and individual multipliers could produce very large effects.

Figure 4 presents the path diagram implied by our third model for someone who is only 5 periods old. Figure 5 shows the results of simulating the model for an individual, holding social effects constant. As before, we assume that the person whose history is being simulated has an average genetic endowment and an average environment (u) before our intervention. However, this time we do allow random shocks from exogenous environmental influences in each period. We choose parameter values that give us a multiplier of 2.6, and in the 6th–20th periods we increase the mean of the exogenous influences (u) by one half a standard deviation of *exogenous environmental influences* (e). This increases the expected equilibrium value of this individual's IQ from 0 to 1.3. Doing this gives the time path for measured intelligence and

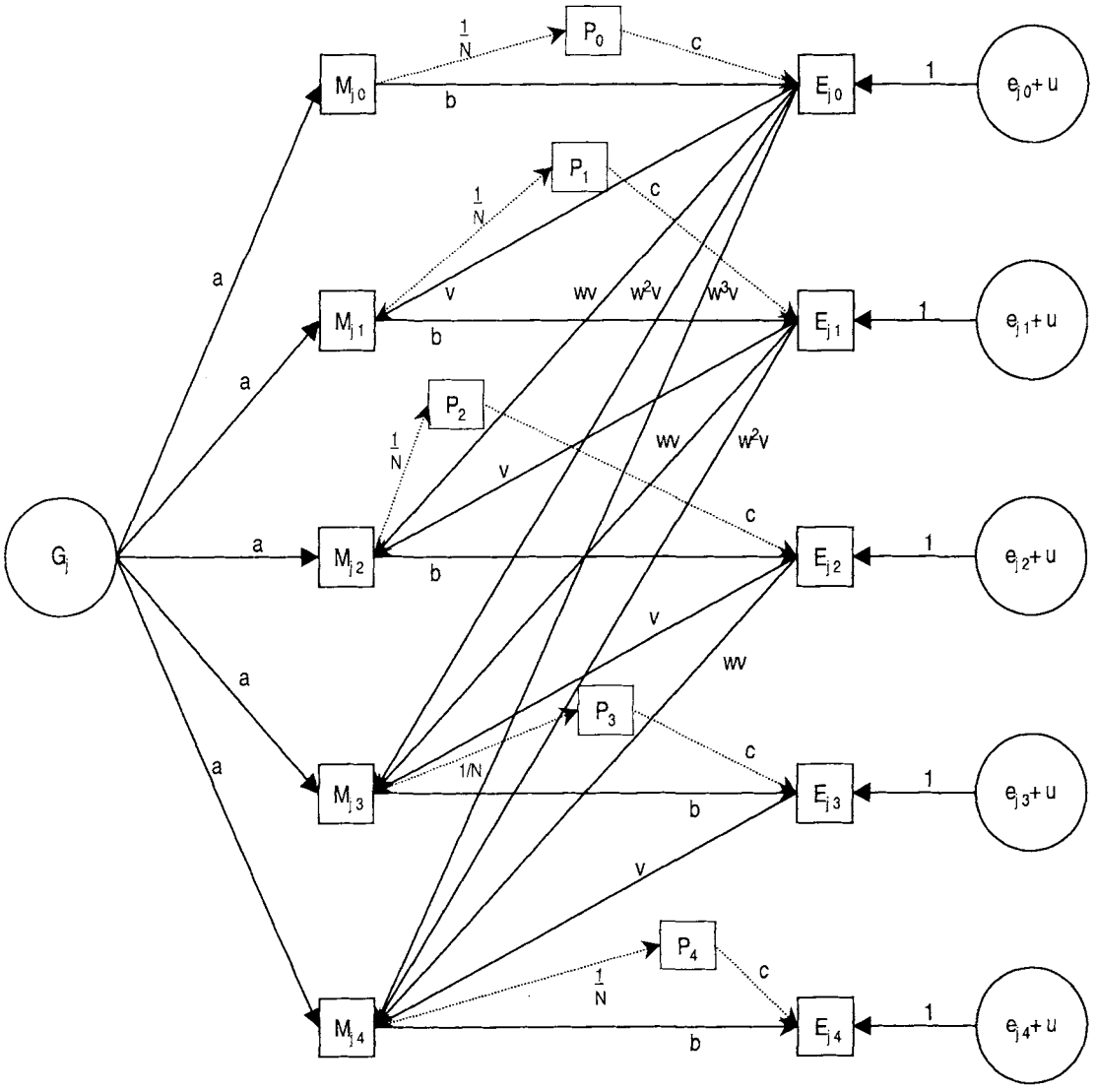

Figure 4. Dynamic interactive model with averaging (Model 3). Person is 5 time periods old. G_j = genetic endowment of person j; M_{jt} = measured IQ of person j at time t; E_{jt} = environment of person j at time t; e_{jt} + u = exogenous environmental influences on person j at time t; P_t = average IQ of population at time t; N = number of individuals in population.

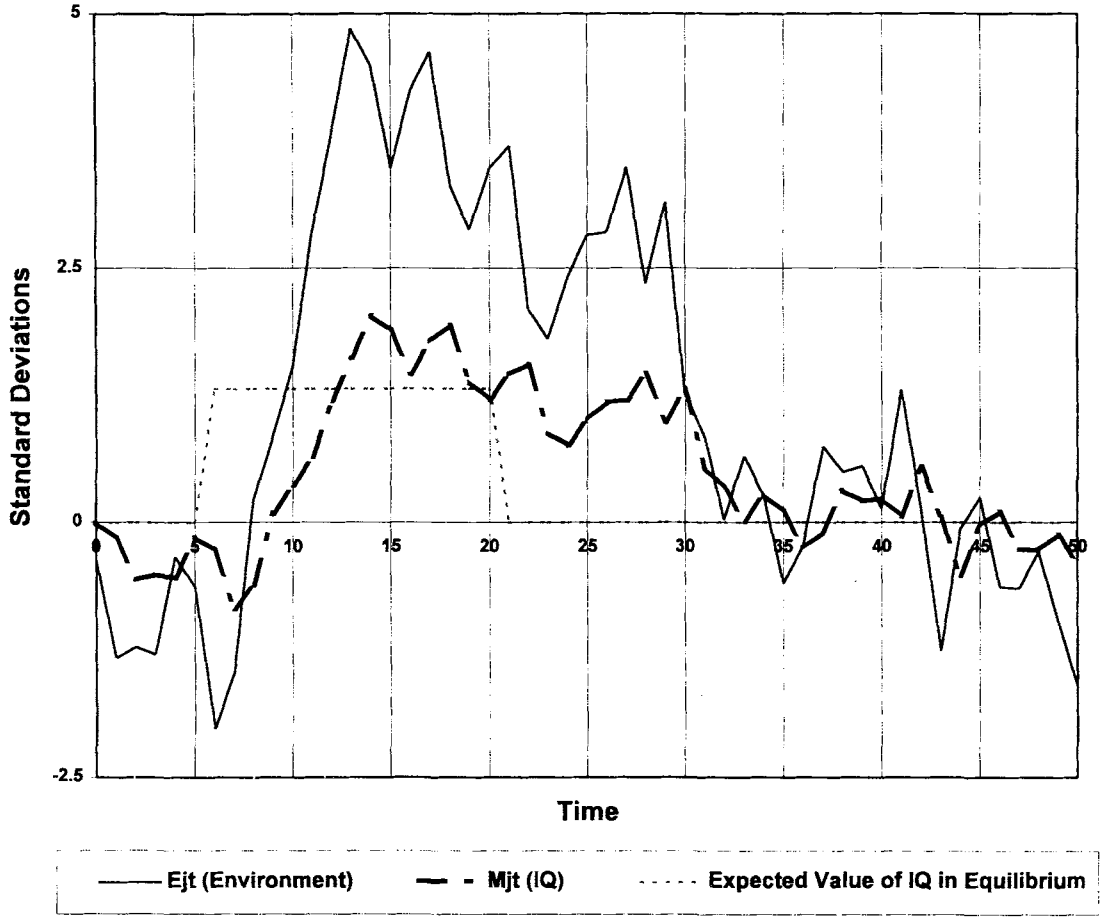

Figure 5. Simulated history for environment and IQ (Model 3, average person with random environmental shocks, individual multiplier of 2.6, and no social multiplier effects).

environment depicted. Note that once again the improvement in the environment causes an increase in IQ, but this time, random shocks to environment cause IQ to rise above its expected target. After the environmental stimulus (the increase in u) is removed, IQ approaches its old equilibrium value and bounces around it as a result of continuing environmental shocks.

We are now positioned to see how a change in the mean of the exogenous environmental influences (u) will affect the average IQ of an individual and society as a whole. We will present our last two equations: Equation 10 shows the effect of changing u for an individual while ignoring social feedback, and Equation 11 shows the effect of the combined individual and social multiplier. These equations have been derived from analysis of Equations 1″ and 2″. Unfortunately, the math involves many steps and therefore has been relegated to the Appendix. The expected effect of changing u for an individual (without social feedback) will be equal to

$$\frac{dE(M|P)}{du} = \sqrt{(1 - h^2)\left[\frac{2h}{(1 - w)a} - 1\right]} \qquad (10)$$

where $E(M|P)$ denotes the expected value of measured intelligence for an individual in equilibrium, given a particular value of P.

If we assume that only the current environment matters for the determination of IQ ($w = 0$) and that the direct impact of genetic endowment on IQ (a) is equal to the square root of measured heritability (h), then the impact of changing u by 1.00 standard deviation of the exogenous environmental influences (e) will be the square root of $1 - h^2$, which is to say the result would be the same as in Jensen's analysis. However, if we allow for some averaging of past environmental influences in the creation of current IQ ($w > 0$), we begin to get the effect of the law of large numbers. The effect of a change in the mean of exogenous environmental influences increases beyond what is implied by Jensen's analysis.

If we also allow the direct impact of genetic endowment on IQ (a) to fall below the square root of heritability (h), we introduce our second form of averaging, and the law-of-large-numbers effect becomes more pronounced. As the direct impact of genetic endowment approaches zero, or the coefficient of the geometric weighted average (w) approaches one, the impact of a 1.00 standard deviation change in u approaches infinity.[13]

[13] If we assume the e_ts are correlated over time, the multiplier will not approach infinity. Instead, it will approach the square root of the ratio of $1 - h^2$ to the correlation of e_t and e_{t-1}. Also, for a correlation greater than zero, the multiplier will be smaller for given values of a, w, and h. However, the critical value for the social multiplier to be discussed is unaffected by assuming correlated e_ts.

Table 2
Environmental Multipliers for Model 3 With Social Effects

Direct genetic impact (a)	No social effect (c = 0)		Medium social effect (c = .4)				Large social effect (c = .8)			
	w = 0		w = .5		w = 0		w = .5		w = 0		w = .5	
.1	2.0	7.9	2.9	8.2	10.5	41.3	∞		∞		∞	
.2	1.4	3.6	2.0	3.9	3.1	8.1	10.5	20.3	∞		∞	
.3	1.1	2.2	1.6	2.5	1.9	3.9	4.6	7.1	8.7	17.3	∞	
.4	0.9	1.5	1.4	1.8	1.4	2.3	3.1	4.1	3.4	5.5	∞	
.5	0.8	1.1	1.2	1.4	1.1	1.6	2.4	2.8	2.1	2.9	46.7	54.5

Note. The first entry in each cell is the effect of increasing the mean of everyone's exogenous environmental influences by one standard deviation of the exogenous environmental effects. The second entry is the effect of increasing the mean of exogenous environmental influences by one standard deviation of the total environment. The latter value is computed by multiplying the former by the standard deviation of total environmental influences (E). That can be shown to equal $\sqrt{1 + b^2}$ where b can be shown to equal $(1 - w)(1 - a/h)/v$.

We have no intention of arguing that the direct impact of genetic endowment (a) is anywhere near zero, nor do we believe that the evidence on the persistence of environmental influences would allow a value of w close to 1.[14] However, assume that the value for a is as large as .3. If we also assume that the value of w is no larger than .5 and use a value for h^2 of .75, Equation 10 gives a multiplier of a change in u of 1.6. That is over three times as large as in the Jensen and Herrnstein and Murray analysis. If the direct effect of genes is .25 and w is .9, the multiplier becomes greater than 4.00. In other words, a change in the mean of the distribution of exogenous environmental influences (u) of as little as 0.25 standard deviations of e would cause more than 1.00 standard deviation change in IQ.

But we are not finished. We have not taken the social multiplier into account. Recall our contention that thanks to social interaction, everyone is affected by their social group's average IQ and, therefore, by the average IQ of society. If we increase the mean of the exogenous environmental influences (u) for everyone, we get

$$\frac{dE(M)}{du} = \frac{\sqrt{(1 - h^2)\left[\frac{2h}{a(1 - w)} - 1\right]}}{1 - \frac{h}{a}\frac{cv}{(1 - w)}} \qquad (11)$$

If the impact of the average intelligence on individual environment (c) is zero, Equation 11 reduces to Equation 10. So if the direct impact of genetic endowment on intelligence (a) is equal to h and w = 0, the effects of changing u are limited to no more than the square root of $1 - h^2$. However, Equation 11 reveals something new. We no longer need the direct effect of genetic endowment (a) to go to zero for the multiplier to become infinitely large. Note the inverse of the ratio of the direct impact of genes to the square root of heritability (a/h) in the denominator of Equation 11; and note what happens as that ratio approaches the direct impact of average IQ on an individual's IQ (cv) divided by one minus the weight of past environments in determining current IQ (w). The combination of the individual multiplier (which is growing as $a(1 - w)/h$ falls) and the social multiplier (which is growing as cv increases) becomes so potent that their total effect becomes infinite.

We do not believe the multiplier is infinitely large. Nonetheless, reasonable values for heritability (h^2), the direct effect of genetic endowment on IQ (a), and the initial impact of a rise in the societal average IQ on individual IQ (cv) can give multipliers that, while finite, are still very large. Table 2 contains illustrative multipliers for a range of parameter values.[15]

Table 2 also shows how much difference it makes when changes are measured in terms of the standard deviation of exogenous environmental effects (e) as opposed to total environmental effects (E). The first entry in each column shows the expected effect on equilibrium IQ of increasing the mean of exogenous environmental influences (u) for everyone in the population by 1.00 standard deviation of e. The second entry shows the effect of increasing u by 1.00 standard deviation of E.

Finally, Table 2 allows us to address the question of the relative importance of the different aspects of the model. We have already discussed why multiplier effects are necessary to get environmental impacts greater than 1.00 standard deviation of IQ from a change in exogenous environment of less than 1.00 standard deviation. In Table 2, we can see that the social multiplier is not necessary for large multiplier effects, but it makes it possible to get them without setting the direct effect of genetic endowment (a) to nearly zero. The individual multiplier produces one form of averaging—so multiplier effects always entail averaging. Still, as Table 2 shows, the other two sources of averaging ($w > 0$ or social multipliers or both) make it easier to obtain large effects.

Interpretations, Implications, Applications, and Tests of the Model

Most of the multipliers in Table 2 are large enough to lend plausibility to environmental hypotheses about massive IQ gains. Assume that something (from our list of triggers or some other cause) raises u, the mean of the distribution of exogenous environmental factors, by 0.01 standard deviations (of e) per year.

[14] Unless time periods are imagined to be very short. See the discussion of preschool enrichment programs in the next section.

[15] Our reason for concentrating on values of a less than .6 will be explained in the discussion of increases in heritability with age in the section after next.

These factors are many and vary over time. Moreover, every person's IQ is affected by the average IQ of their social circle and therefore by the societal average IQ, so we also have a social multiplier. We will assume that the triggers have been operating over a long time and that society has reached growth equilibrium. For this example, we will assume that $c = .5$, $a = .2$, and $w = 0$. With these parameter values, a 1.00 standard deviation increase in the mean of the exogenous environmental influences will produce a 4.5 standard deviation IQ gain. So if u has been increasing by 0.01 standard deviation each year for 30 years, the total increase of .30 standard deviations will produce a 1.35 standard deviation IQ gain. This more than matches the Dutch IQ gain on Raven's from 1952 to 1982.

We obtain these changes by hypothesizing a shift in the mean of the distribution of exogenous environmental influences. We claim that this provides a more plausible explanation of IQ changes than does a literal Factor X that must impact each individual in exactly the same way. Even though it is very hard to imagine how a uniform effect across individuals could be changing over time as required by a literal Factor X, it is not hard to imagine that the mean of the distribution of environmental influences could be changing over time. The fact that this mean is changing does not imply uniform effects. For example, if the prime mover is the cognitive demands of jobs that might be changing due to changing technology and growing wealth, our model does not require that all jobs are becoming equally more demanding. Some could be increasing their cognitive demands whereas others could be decreasing.

Of course, if the variance of the cognitive demands of jobs have changed, the variance of IQ would have to change too. This could be a problem for the model because we are aware of no evidence that suggests that the variance of raw test scores has been changing. However, it is easy to imagine that the mean could change without the variance changing. If jobs evolve with one set of tasks replacing another set of tasks, and if the variance of the demands of the new tasks is similar to that of the old, the total variance of cognitive demands will not change. Alternatively, there could be offsetting effects. For example, the mean of the cognitive demands of jobs could be rising because already-complicated jobs are becoming even more complicated. This would increase the variance of the cognitive demands of jobs and tend to increase the variance of IQ in our model. However, if at the same time declining family size is increasing the cognitive demands of family life, this would almost certainly be accompanied by a reduction in the variance of family size due to floor effects (families have to have at least two people) and therefore a reduction in the variance of the cognitive demands of family life. With one trend tending to produce an increase in variance and another a decrease, the net effect on the variance of IQ could be quite small.

Having used our model to answer questions about heritability and IQ gains, let us now reverse direction. Assume our model provides a reasonable representation of the process generating individual and generational IQ differences. What do h^2 estimates and IQ gains tell us about the model?

To accommodate large environmental effects and high heritability, our model requires relatively large multipliers for changes in the mean of exogenous environmental influences, $dE(M)/du$. We may speculate about how big environmental differences between generations have been, but we cannot know for sure. The larger the exogenous environmental change one finds credible, the smaller the multiplier necessary to accommodate observed changes in IQ. On the one hand, there have been large changes in affluence, leisure, the workplace, and the home. On the other hand, the variance component we attribute to "environment" contains differences due to many things that probably could not change over time. Therefore, we would be surprised if exogenous environmental changes have been more than 1.00 standard deviation per generation and suspect that they could be considerably less than that. Thus, multipliers of 1.5 or more seem necessary to explain IQ gains over time. Such multipliers could be produced by any of many different combinations of values of the model's parameters. However, to get the necessary multipliers, the indirect effect of genes on IQ through environment must rival or dominate the direct effect of genetic endowment. This is a different picture of the development of human intelligence than that usually associated with the pronouncement that 75% of IQ variance is genetic.

We believe that our model can shed new light on other phenomena concerning IQ. In the process, we draw implications for the parameters of the model and further implications for our understanding of the process that generates IQ. Our approach is to assume that the parameters of the equation for IQ are fixed whereas the parameters of the equation for environment vary with age and the circumstances of the individual. We would not be surprised if genes and environment did play different roles in the generation of IQ at different ages, but it makes sense to us that the biological system determining IQ would be more stable than would be the social system determining environment. Further, natural assumptions about how the process that determines environment might change do seem enough to explain a wide range of phenomena without assuming any change in the parameters of the IQ equation.

Heritability, Stability, and Age

Jensen (1998, pp. 179–181) and Neisser et al. (1996, p. 86) have suggested that the matching of phenotype (IQ) to environment with age might explain why the value for h^2 rises with age. However, we do not believe that the implications of such a matching process are fully appreciated.

In our model, the phenomena of rising heritability is understood as resulting from an increase in the impact of one's own IQ on one's environment (b) with age. This is probably accompanied by a decline in impact of exogenous environmental influences[16] as individuals take control of more and more aspects of their life. Recall that in our second model (before we introduced averaging), heritability (h^2) equals $a^2/(1 - bv)^2$. A similar, but slightly more-complicated, relation holds in our third model (see Appendix, Equation A14). Since bv must be less than 1 for changes in environment not to have infinitely large effects on IQ, increasing b will increase the magnitude of h^2 estimates. But also note that unless we believe that the direct impact of genes on IQ changes with age, the low h^2 estimates for children place an upper bound on a—the direct impact of genetic endowment on IQ. The review

[16] In our model, this would be represented as a decline in the variance of e.

by Neisser et al. (1996, p. 85) concludes that for children, h^2 is "of the order of .45." If this is correct, the direct impact of genes on IQ can be no larger than the square root of .45—which is .67.

Further, that value is an *upper* bound. If we are correct in thinking that the explanation of IQ gains over time involves a substantial individual multiplier, the direct impact of genetic endowment on IQ would have to be much lower. Unless one assumes that the direct impact of genes on IQ (a) changes with age, large multipliers imply that the direct impact of genes is likely to be substantially less than the square root of even lowest estimates of heritability in children. This is why we focus on values of a of less than .6 in Table 2.

Our analysis suggests the reinterpretation of another phenomenon. It has long been noted that preschool children's IQs are particularly unstable and that IQs become more stable as people age. In terms of our model, this can be understood as resulting from the same factors that increase h^2 with age. As more and more of the environment comes under control of the individual (in our model, a rising b and a falling variance of e), the more IQ reflects a person's genetic endowment. At the same time, the relatively transient exogenous environmental factors explain less of the IQ variance between people. As the fraction of variance explained by genes (both directly and indirectly) grows with age, the constancy of IQ over time grows—because genetic differences are stable. Therefore, it may not be a change in the child that increases the constancy of IQ with age, but rather a change in how much control the child is exercising over his or her environment. The very low correlations between the IQs of very young children and their later IQs (even after correction for reliability) might be interpreted, within the framework we propose, as evidence that the upper bound for the direct impact of genetic endowment on IQ is lower still.

The Disappearance of Shared Environment and the Source of Nonshared Environment

Jensen (1998, pp. 178–197) notes that as heritability grows with age, it does so mainly at the expense of another component of IQ variance, namely shared environment. It accounts for a small but significant proportion of IQ variance among young children, but completely disappears in most studies of late-teenage children and adults.

The influence of shared environment is measured most directly by correlations between the IQs of adoptees who share the same environment but don't share genes with the natural siblings in their adoptive family. As for unrelated children currently residing in the same household, our model would explain the correlation between their IQs in terms of the correlation between their values for the exogenous component of environment (e). The more aspects of their environments they share, the larger the correlation between their es, and therefore the larger the fraction of IQ variance attributable to shared environment. As children age and become more and more independent of their families, living in the same household would mean less and less common environment. After they leave home and live separately, they would have very little environment in common.

A property of our final model is that any transient environmental effect will decay over time. Specifically, it will decay exponentially at the rate $(1 - w - bv)$.[17] As siblings grow older and share less of their environment, their IQs should become less correlated. After some time, they should share almost no environmental effects that are not induced by similar genetic endowment, and the only correlation detectable would be genetic in origin. Thus, our model predicts that the shared environment component of IQ variance will disappear with age.

It can also help us understand why nonshared (as distinct from shared) environmental variance persists in adults. Because all transient environmental differences decay with time, a literal interpretation of our model suggests that childhood experiences cannot be the source of nonshared variance among adults. The source would have to be transient environmental influences closer to the time when their IQs are measured—the random effects that cause a less than perfect match between peoples' environments and their IQs. Therefore, when Plomin and Daniels (1987) say that psychologists should look to environmental differences between siblings for the source of nonshared environmental variance in adults, their suggestion is unlikely to be fruitful; it is no more promising than looking for permanent effects of shared environment.[18] Indeed, Turkheimer and Waldron's (2000) review of studies of the impact of specific nonshared environmental influences finds negligible effect sizes when genetically informed designs are used.

As it stands, our model implies that literally no transient environmental influence has permanent effects. However, it could be altered to accommodate such. For example, we could allow for a neurodeficit caused by inadequate neonatal nutrition by putting a separate term for environmentally induced biological effects into the equation generating IQ. We could allow for other permanent effects by hypothesizing that the current value of IQ has a small permanent effect on the mean of exogenous environmental influences (u). This would "lock in" at least a part of any transient gain or loss in IQ. Take the case of shared childhood environment. At present, there is no evidence of important impacts persisting into adulthood. However, Stoolmiller (1999) has argued that significant restriction of range in adoption studies leads to a profound underestimate of the importance of shared environment. We believe we could accommodate persistent effects of shared family environment without compromising any of our fundamental results.

Compensatory Education

In their review of the effects of early education programs on IQ scores, Lazar and Darlington (1982) note that "The conclusion that

[17] In a recursive model such as this, a disturbance from equilibrium will exhibit exponential decay at a rate equal to the denominator of the multiplier. Since we are dealing with the rate of decay for an individual, it is the denominator of the individual multiplier that is relevant. A typescript deriving the decay rate is available from the authors. One should not assume that one could identify the denominator for a realistic model from the rate of decay of environmental effects. Such a model would have a number of additional terms complicating such an interpretation (see Footnote 19).

[18] Turkheimer (1991) points out the extreme assumptions that are necessary for childhood environment to produce permanent within- but not between-family effects.

a well-run cognitively oriented early education program will increase the IQ scores of low-income children by the end of the program is one of the least disputed results in education evaluation" (p. 44). The wide range of programs they surveyed show other similarities—some of which are less welcome. First, nearly all the gains that treatment children make relative to controls occur in the first year of the program; second, the gains decay when the program ends; and third, they decay far slower than the rate at which they were made. These facts can be explained in the context of our model and shed some light on what the parameters of the model might be.

We would describe the impact of early education as an improvement (increase) in participants' exogenous environmental influences (e) and a decline in the impact of their own IQ on their environment (b) during the time they are in the program. The impact of each child's IQ on environment declines, we believe, because the programs substitute cognitively demanding activities for activities that the children would have chosen for themselves, or that others would have chosen for them, had they not been in the program. Thus the environments of children in the program are less subject to influence by their own IQs.

If children's IQs aren't having much effect on their environments, and if their environments suddenly improve a great deal, a very rapid IQ rise might be expected. In several of the studies reported in Lazar and Darlington (1982, p. 45, Table 14), treatment-control differences are largest in the first year of the program. No study shows any sign of steadily increasing gains over the whole course of the program. This pattern—a dramatic rise in the first year—has implications for our model. It suggests that if the time period assumed in the model is a year, the time-averaging of environmental effects is not important (which means that w is very close to zero). If time-averaging were going on, the children would still be feeling the drag of their old environments as they entered the program. Gains would come only slowly as the effects of past environments wore off.

The fact that there are no additional gains beyond the initial large jump not only implies that the time-averaging of environmental effects is not active, it also reinforces the conclusion that the program renders the effect of the children's IQs on their own environments relatively unimportant. Otherwise, the IQ gains that the children were making would prompt them to make further improvements in their environments, which in turn would produce further IQ gains over time. For children in these programs, the impact of their IQs on their environments must also be close to zero (b must be close to zero, not just w).[19]

Our model implies that a transient environmental influence will decay exponentially. Therefore, we would expect that once the exogenous shock to their environments (the enrichment treatment) goes away, the effects of the treatment on their IQs would begin to decay exponentially. The studies show that the effects do not disappear dramatically but rather decay slowly—in contrast with their rapid appearance. The decay is more rapid at the start and then slows over time as in the examples of the behavior of our model in Figures 3 and 5. This is evidence that when children leave the program, their enhanced IQs are matching them with better environments than they had prior to the program, and that those environments are doing something to keep their IQs higher (which means that b times v must be significantly greater than zero once children are out of the programs).

The contrast between the rapid onset and the relatively slow decay of treatment IQ gains has another implication when interpreted in the context of our model. Even though the rapid onset suggests that the effects of own IQ on environment are virtually nonexistent during the program, the slow decay suggests that the effect of one's own IQ on environment is strong after the end of the program. And if that is true, it suggests something about the character of those aspects of environment that are influenced by individual IQ—intervention programs are able to change them and take children's "control" over them away, which means that the environment that affects a child's IQ must be external to the child or at least subject to manipulation by outsiders. Recall our discussion about what metric should be used to measure the effect size of environment. If the part of total environment that is normally determined by one's own IQ can change independent of one's actions to change it, the standard deviation of total environment (E) may be the appropriate metric for measuring environmental change—not the standard deviation of exogenous environment (e). Thus, the second entries in each cell of Table 2 (the larger values) may be more appropriate for our multiplier than the first entries.

Adoption Studies and Cross-Racial Parenting

Our model can account for the pattern of results from these studies. Adoption is perhaps the most ambitious environmental manipulation possible. When a child from a disadvantaged background is adopted into an upper-middle-class family, the improvement in the quality of environment amounts to a radical change in exogenous environmental influences (e). Studies show large impacts of adoption on IQ in the expected direction while children live in their adoptive homes. Even Lucurto's (1990) skeptical review of adoption studies suggests that the typical adoption moves the child into a better environment and increases the child's IQ by about 12 points.[20] However, those studies in which children have been followed into adolescence (Scarr & Weinberg, 1983; Scarr, Weinberg, & Waldman, 1993) show that as they age, their IQs match their adoptive family less and less and their biological family more and more. Readers comfortable with the model will see that this could be the result of adoptive children gaining control over larger and larger parts of their environment as they age and the consequent decay of shared environmental influences.

Studies of cross-racial parenting and adoption can also be explained more readily by our model than by the standard model. The standard model implies that environment is feeble. If so, why do

[19] Or at least any feedback must be happening very quickly. The model we have presented assumes that only current IQ affects current environment and that environment only affects IQ with a lag. If we were going to apply our model to data on IQ measurements in children that were being made a year or more apart, we would want to elaborate the model to allow for simultaneous determination of environment and IQ and for lagged effects of IQ on environment. In such a model, the finding that nearly all IQ gains happen in the first year would not preclude a multiplier effect in the simultaneous determination of IQ and environment, but it would preclude any lagged effect of IQ on environment or environment on IQ.

[20] More recently, see Duyme, Dumaret, and Tomkiewicz (1999).

the children of Black mothers and White fathers have IQs so much lower than the children of White mothers and Black fathers (Willerman, Naylor, & Myrianthopoulus, 1974)? Mothers are much more important contributors to the typical child's environment than fathers are, but both mothers and fathers contribute equally to a child's genes. In the context of our model, the results of Willerman et al. (1974) suggest that environment plays a potent role in Black–White IQ differences.

The results that are the least friendly to an environmental hypothesis about racial differences are based on comparing Black and half-Black children adopted by White parents with White children adopted by the same parents plus their natural children (Scarr & Weinberg, 1976; Scarr, Weinberg, & Waldman, 1993). While the Black adoptees are young and living primarily at home, the White families have sizable impacts on their IQs. As they age, the adoptees' IQs begin to correlate more with their Black natural parents than with their White adoptive parents and their mean IQ tends to fall well below that of the White adopted children. Taken together, these two phenomena have been interpreted as evidence of a genetic gap between the races: Black children regressing to a genetically determined Black mean that is lower than a genetically determined White mean.

Four points are relevant. First, if family influences become weak in late adolescence, the effects of adoption will fade—no matter what the race of the child. Second, adoptees will tend to return to their preintervention IQs if their postfamily genes and environments are similar to their prefamily genes and environments (the latter is notional, of course). A combination of genes and environment will determine their pre- or postfamily IQs. When the fact that the average mature IQ of Black adoptees is lower than the average mature IQ of White adoptees is cited as evidence that genes rather than environment cause the Black–White IQ gap, that simply begs the question. All possibilities are still open—whether genes or environment or both determine the racial IQ gap. Our model shows that the adoption data makes no prima facie case that environment has a weak explanatory role concerning IQ differences either within or between the races.

Third, as Flynn (1980, p. 104; 1999, pp. 13–14) has acknowledged, it is disturbing that the Black and White adoptees do not exhibit IQ parity while still immersed in the family environment. Perhaps even then, the family does not totally dominate the environment; moreover, the adoptive family cannot level prenatal, perinatal, and early postnatal environmental differences between Black and White, differences that may be of some importance (Broman, Nichols, & Kennedy, 1975; Jensen, 1998, pp. 500–509). Unlike the more-transient environmental influences of later life, very early environmental influences of this kind may have physical effects that are as permanent as the effects of genetic endowment. Nearly all estimates of heritability based on adoption studies would confound such environmental influences with genetic endowment.

Finally, there is Eyferth's (1961) study of the children fathered by Black and White American soldiers with German women after World War II. This is the only study of Black American children totally extracted from their usual environments. The half-Black children had White German prenatal, perinatal, postnatal, and family environments. The study showed IQ parity between Black (half-Black) and White. Flynn (1980, pp. 84–102) has investigated related questions, such as whether the Black fathers were a genetic elite.

This is not the place to reargue the Black–White IQ question. We merely wish to put the cross-racial adoption literature into perspective and point out how these studies can be easily accommodated in the context of our model. In contrast, the standard model of Jensen and others has a very difficult time accommodating the clearly large environmental effects evident when children are still living in their adoptive homes.

Studies of the Effects of Schooling on IQ

It is sometimes contended that the correlation between years of education and IQ shows that people who have higher IQs get more education, not that education raises IQ. Ceci (1991) presents an impressive array of many different types of evidence, and his analysis leaves little doubt that schooling does influence IQ. However, our model suggests a very different interpretation of that phenomenon than the one found in the studies that Ceci, and more recently Winship and Korenman (1997), review.

Several studies claim to find large effects of education on IQ that persist many years after people have left school.[21] There could be no explanation for large permanent effects in our model because our model implies that the effects of past environment should decay over time. As noted above, we could adapt our model to accommodate modest permanent environmental effects. But must we do so?

All studies that find long-lasting effects that we have identified possess a common methodology: In effect, they regress current IQ on a measure of IQ taken when people were still in school, the number of years of school completed, and other variables. A positive coefficient on years of education is taken as evidence of a causal effect of education on IQ. That does not necessarily follow, as our model makes clear. Assuming that years of schooling completed reflects IQ at the time of school completion and that the two measures of IQ and the completion of school take place at points in time sufficiently far apart so that environmental factors determining IQ are essentially uncorrelated, the correlation between the three measures is entirely due to the common element of the individual's genetic endowment. In other words, by regressing adult IQ on years of schooling completed and an earlier measure of IQ, researchers may have regressed one measure of genetic potential on two other noisy measures of genetic potential. If a variable is regressed on two noisy measures of itself, both will have positive coefficients with their magnitude depending on their signal-to-noise ratios. Studies with this design are simply not informative about the effect of schooling on IQ.

However, the literature also contains a number of quasi-experimental studies where factors beyond the control of individuals cause them to attend more or less school. These are exempt from the criticism made above. As far as we can see, IQ effects in these studies have all been measured fairly close in time to the environmental change. Two of the largest estimated effects for

[21] Lorge (1945) claims to find effects of schooling on IQ in his sample of men in their 30s. Jencks (1972) and Wolfle (1980) construct estimates of the effect of education on IQ using path analysis, where the correlations employed are drawn from several different studies.

educational deprivation come from cases in which large groups of children, whose members would have formed each other's peer groups, were deprived of formal education for extended periods of time (DeGroot, 1948; Green, Hoffman, Morse, Hayes, & Morgan, 1964). There are confounding factors in both cases, and both sets of authors have a difficult time convincingly establishing the quasi-experimental counterfactual. Still, the large effects measured in these studies of groups whose members interact, when contrasted with studies of individuals, are at least suggestive of the importance of the social multiplier.

The Positive Correlation of g and Gains Across Subtests

Rushton (1999, p. 382) asserts that if group differences in performance on Wechsler Intelligence Scale for Children (WISC) subtests are environmental in origin, the magnitude of the differences should be negatively correlated with subtests' g loadings and heritabilities. This assertion has some credibility in the context of the standard model. Our Equation 1 represents that model if we assume that G and E are uncorrelated, which implies that $a = h$ and $v = (1 - h^2)^{.5}$. If we also assume that the difference between groups in E is the same for every subtest, and if we allow $h(a)$ to vary across subtests, and posit that g loading is simply an alternative measure of heritability, then Rushton's assertion follows. Of course, there is no reason why an environmental difference between two groups must have a uniform effect across all subtests—for example, if every member of one group were given the correct answers for one of the subtests. Thus, even in the context of the standard model, Rushton is wrong to assert that environmentally induced performance differences are *necessarily* negatively correlated with heritabilities or g loadings. Still, unless there was some reason to posit a correlation between g loadings and the variation in E differences across subtests, there would be no reason to expect the correlation to be positive and some reason to expect it to be negative.

By contrast, our model can more easily accommodate positive correlations between environmentally caused group differences and measures of heritability. If the reciprocal impact of environment and IQ on each other (bv) differs across subtests, and the direct impact of genes on subtest scores does not vary, then environmentally induced gains will be largest on the same subtests that register the highest heritabilities. This follows because larger values for bv imply both larger environmental multipliers and larger values for h^2, all else held equal.

Rushton himself agrees that subtest gains on the WISC are positively correlated with inbreeding depression—an indicator of heritability. However, he emphasizes that they are negatively correlated with a measure of g for the WISC. Neither correlation is statistically significant. On the other hand, Flynn (1994, 1998a, 2000) has shown that IQ gains over time have been largest on tests of fluid g like Raven's Progressive Matrices, tests that measure raw problem-solving ability while tapping a minimum of learned skill. Flynn (2000) presents IQ gains on WISC subtests and shows that they are positively correlated with a measure of fluid g—though again the results are not statistically significant. Similarly, Jensen (1997) finds positive correlations, again not significant, when he relates g loadings to subtest differences induced by adoption. These positive correlations are more easily accommodated in our model than in the standard model.

Estimating and Testing Our Model

Our model was developed to illustrate how we believe environment and phenotypic IQ cause each other. Some of its features were chosen for analytic convenience rather than for realism. For example, the model is recursive in IQ and environment primarily to simplify exposition. Were we to attempt to estimate the parameters of the model with long time periods between IQ measurements—periods typical of those studies with repeated measures—we would undoubtedly have to allow for simultaneous determination of IQ and environment. We would also have to allow for lagged effects of IQ on environment and for correlation across time of exogenous environmental effects. We know of no study that offers rich data on environment and high frequency observations on IQ in the context of a genetically informed design, which is to say we know of no data of the sort that would be necessary to estimate such a model. Were such data available, estimation might tell us a great deal about the model's ability to explain IQ differences between individuals.

Because the simple static model is a special case of our model, it should, in theory, be easy to test our more-general model against the restrictions implied by that standard model. In practice, we suspect that gathering the data needed for estimation of our model would require a massive effort over many years. Is there any relevant data available short of such an effort? We offer a tentative "yes." The exercises performed throughout this section—reviewing salient findings from studies of different aspects of the process generating IQ and deducing their implications for the model's parameters—has, we hope, been suggestive. A complete structural meta-analysis of the literature might identify the parameters of at least a very parsimonious version of our model. If the parameters were overidentified, it might be possible to estimate confidence intervals and test restrictions. Even if such an analysis could not identify all of the parameters of our model, it might succeed in testing the restrictions of the simple static model. At the very least, we could use such a meta-analysis, along with Bayesian techniques, to calibrate our model and explore the sensitivity of parameter estimates to different prior beliefs about the magnitude of environmental changes over time and key model parameters.

However, it is unlikely that this would provide a very convincing test. Rather than attempting to estimate the entire model, it might be more fruitful to explore the model's potential for surprising or novel predictions and test those. For example, it predicts that even in adults, radical environmental change should produce significant changes in IQ. If we could test IQ before and after periods of incarceration, or before and after joining religious cults that significantly restrict people's control over their lifestyles, we might observe large changes in IQ surprising from the perspective of the standard model.

Conclusion

We began with a paradox: If environment explains so little of the IQ variance between individuals, how could changes in environment produce the huge IQ gains that have been observed? We have shown how the reciprocal causation of phenotypic IQ and environment could mask, multiply, and average environmental effects, so that relatively small environmental influences could produce large changes in IQ. We have also sketched how the

model can be used to explain a wide range of phenomena in the IQ literature, how those phenomena can contribute to a better understanding of the implications of the model, and some ways in which the model might be tested and estimated.

The picture that emerges suggests a powerful role for environment in shaping individual IQ. However, we wish to stress that the way environment plays its role is very different from the traditional characterization. It appears that most environmental effects are relatively short-lived.[22] At least for young children, experiences much more than a year old influence today's IQ only because of their effect on past IQ and the effect of past IQ on today's environment. Even then, the effects of environment decay, leaving only a narrow window in which transient environmental effects may influence IQ. If correct, our model suggests that improving IQs in childhood is not the way to raise the IQs of adults. Adult IQ is influenced mainly by adult environment. Enrichment programs may nonetheless be worthwhile because at least some seem to have long-term effects on achievement and life outcomes, and the temporary IQ boosts they provide may mediate those effects. However, our model suggests that such programs would be most likely to produce long-term IQ gains if they taught children how to replicate outside the program the kinds of cognitively demanding experiences that produce IQ gains while they are in the program and motivate them to persist in that replication long after they have left the program. If the programs that were the subjects of longitudinal evaluations were trying to do this, those evaluations suggest that they were unsuccessful.

Our model was motivated by a paradox in the study of IQ, but it may be relevant for describing the development of a wider range of traits and behavior. Any trait that has a tendency to match itself to an environment that reinforces that trait will behave in the fashion our model describes, as long as the effects of environment do not accumulate over time. Even if environmental influences accumulate, that could be accommodated by some simple changes in the model. Setting aside the paradox of genes versus environment in the development of IQ, we hope that our analysis demonstrates something else of importance—the potential of formal models of the development of phenotypic behavioral traits as a product of the reciprocal causation of environment and phenotype.

[22] Environmental factors that have permanent physical effects, as nutrition might, would be exceptions. In fact, by having persistent effects, we would expect that such factors would also have very large effects—their persistence allowing them to rival genes in importance. The virtual disappearance of between-family effects with age in the study of IQ differences suggests that there is little systematic difference between families in the factors producing such effects, at least within the range of families that have been the subjects of IQ studies. However, views on this may change, because some of the most compelling critiques of existing studies of heritability suggest that these studies may profoundly understate the role of shared family environment (Stoolmiller, 1999).

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(Appendix follows)

Appendix

Derivations of the Multipliers

In this appendix, we show the full derivation of the multiplier in Equation 9 from Equations 1′ and 2′ and show how the multipliers presented in Equations 10 and 11 can be derived from the model represented by Equations 1″ and 2″.

Deriving the Environmental Multiplier for Model 2 (Equation 9)

We assume that G_j and e_j from Equations 1′ and 2′ are independent random draws from populations with mean 0, variance 1 for G, and variance σ_e^2 for e. Given individual values for G_j and e_j, Equations 1′ and 2′ represent a system of deterministic difference equations that will have a single equilibrium value for each person j if bv is positive and less than 1 as we assume. The easiest way to find the equilibrium value is to substitute the right-hand side (RHS) of Equation 2′ for E_{jt} in Equation 1′, to assume the equilibrium condition that $M_{jt} = M_{jt-1}$ for all t, and to solve for M_j. That yields Equation 6 in the text. Substituting the RHS of Equation 6 into Equation 2′ yields Equation 7 in the text.

We now note that M_j gives us the IQ of individual j as a function of two independent random variables, G_j and e_j. Thus the variance of M_j can be uniquely partitioned. Assuming that the variance of M_j and G_j have been standardized to 1, and that the expected values of G_j and e_j are 0, the expected value of $M_j = vu/(1 - bv)$. Subtracting that from both sides of Equation 6, squaring them, and then taking expected values yields

$$1 = \frac{a^2}{(1 - bv)^2} + \frac{v^2\sigma_e^2}{(1 - bv)^2}. \tag{A1}$$

If the process generating IQ data in a population was that represented in Equations 1′ and 2′, then the expected value of h^2 would be the first term in Equation A1, and $1 - h^2$ would be equal to the second term. To see this, note that h^2 can be estimated as the correlation of the IQs of identical twins raised apart. Identical twins raised apart would have G in common but their es would be independent, so from Equation 6 the correlation of IQs of identical twins raised apart will be equal to the first term in Equation A1.

Repeating the analysis of Equation 6 done above to find the mean of M_j for Equation 7, we find the mean of E_j is $u/(1 - bv)$. Subtracting it from each side of Equation 7, squaring, and taking expected values yields

$$1 = \frac{b^2a^2}{(1 - bv)^2} + \frac{\sigma_e^2}{(1 - bv)^2}. \tag{A2}$$

Now note that

$$h^2 = \frac{a^2}{(1 - bv)^2} \Rightarrow (1 - bv) = \frac{a}{h}$$

and

$$b = \frac{1 - \frac{a}{h}}{v} \tag{A3}$$

and that if we substitute a/h for $(1 - bv)$ in Equation A2, it implies that

$$\sigma_e^2 = \frac{a^2}{h^2} - b^2a^2. \tag{A4}$$

Next, we use the fact that $(1 - h^2)$ is equal to the second term in Equation A1 and get

$$v^2 = \frac{(1 - h^2)(1 - bv)^2}{\sigma_e^2} \tag{A5}$$

by rearranging terms. Substituting for b in Equation A4 from Equation A3, substituting for the variance of e in Equation A5 from Equation A4, substituting for $(1 - bv)$ from Equation A3, solving for v^2, simplifying, and taking the square root of both sides yields Equation 8 in the text. Taking the derivative of the expected value of Equation 6 with respect to u yields the effect on M of changing u, which is the constant $v/(1 - bv)$. Substituting for v and $(1 - bv)$ using Equations 8 and A3 yields the form for the multiplier in Equation 9.

Derivation of the Multipliers for Model 3 (Equations 10 and 11)

Our strategy for computing these multipliers has two steps. First, compute the derivative of the expected value of measured IQ with respect to a change in the mean of the distribution of exogenous environmental effects (u). Second, show how the elements of those derivatives can be related to quantities we either know or might have intuitions about to derive the results discussed in the text, thus relating the values of the multipliers to a, h, and the impact of population averages on individual IQ.

To begin, we note that Equation 1″ implies

$$wM_{jt-1} = waG_j + v\sum_{i=1}^{t-1} w^iE_{jt-i-1}, \tag{A6}$$

which further implies that Equation 1″ can be rewritten

$$\begin{aligned} M_{jt} &= aG_j + v\sum_{i=1}^{t} w^{i-1}E_{jt-i} + wM_{jt-1} - waG_j - v\sum_{i=1}^{t-1} w^iE_{jt-i-1} \\ &= (1 - w)aG_j + wM_{jt-1} + vE_{jt-1}. \end{aligned} \tag{A7}$$

Substituting for E_{jt-1} from Equation 2″ in Equation A7 yields

$$M_{jt} = (1 - w)aG_j + (w + bv)M_{jt-1} + vcP + v(u + e_{jt-1}). \tag{A8}$$

Equation A8 describes a dynamic stochastic system. As with any dynamic system, analysis requires either that we state starting conditions or we must analyze the system asymptotically. We choose to do both in that we will assume that M and E for each person at time zero will start at values such that at all points in time $E(M_{jt}) = E(M_{jt-1})$ and $E(E_{jt}) = E(E_{jt-1})$—that is, we are assuming that the starting values for M and E are their asymptotic or equilibrium expectations. These assumptions simplify the analysis and will not substantially impact the relation between the model and reality, because there is evidence that the system tends to converge in expectation to its long run equilibrium relatively quickly. Specifically, the decay of the effects of enrichment programs and the disappearance of the influence of shared environment relatively quickly after people strike out on their own suggest that the system quickly seeks a new equilibrium when an environmental influence is removed. Thus, our starting value assumptions should be appropriate for the analysis of heritability estimates in populations of adults. The assumption allows us to drop the time subscript on single variables within expectations. For notational convenience, at this point we will also drop the subscript j for individual.

Considering only how the average individual's intelligence will be determined given a particular level of average intelligence in society (P),

the expected value of M (from Equation A8) treating P as a given constant is

$$E(M|P) = (w + bv)E(M|P) + vcP + vu = \frac{vcP + vu}{1 - w - bv}. \quad \text{(A9)}$$

Thus, holding the average level of IQ constant, the effect of a one-unit change in u on the average individual's IQ is $v/(1 - w - bv)$.

If we ask what will happen to the expected value of IQ in society if u is increased, assume that society is large so that $P = E(M)$ and substitute for P in Equation A9 to get

$$E(M) = (w + bv)E(M) + vcE(M) + vu = \frac{vu}{1 - w - bv - vc} \quad \text{(A10)}$$

or a multiplier for a change in u of $v/(1 - w - bv - vc)$.

We now want to express these multipliers in terms of values we know or might have intuitions about. To do that, we want to relate the parameters of the model to the decomposition of variance in the analysis of heritability. To do that, we note

$$\begin{aligned}\text{Var}(M) &= E([M - E(M)]^2) \\ &= E(\{[M - E(M|G)] + [E(M|G) - E(M)]\}^2). \quad \text{(A11)}\end{aligned}$$

(Note that we are analyzing the individual multiplier so P is held constant, but we have dropped the notation indicating this for convenience.) Since the two terms $M - E(M|G)$ and $E(M|G) - E(M)$ are orthogonal by construction, we have

$$\text{Var}(M) = E([E(M|G) - E(M)]^2) + E([M - E(M|G)]^2). \quad \text{(A12)}$$

or the variance of M can be decomposed into that part which is due to differences in genetic endowment (the first term) and that part which is due to all other factors (the second term). If we continue to assume that we are measuring intelligence in terms of population standard deviations, then the first term on the right-hand-side of Equation A12 corresponds to what is measured as h^2, while the second term corresponds to what is measured by $1 - h^2$.

Taking the expectation of Equation A8 treating G as a constant yields

$$\begin{aligned}E(M|G) &= (1 - w)aG + vcP + vu + (w + bv)E(M|G) \\ &= \frac{(1 - w)aG + vcP + vu}{1 - w - bv}. \quad \text{(A13)}\end{aligned}$$

Defining $D_t = M_t - E(M|G)$ and $F = E(M|G) - E(M)$. Then,

$$h^2 = E(F^2) = E\left\{\left[\frac{(1 - w)aG}{1 - w - bv}\right]^2\right\} = \frac{(1 - w)^2a^2}{(1 - w - bv)^2}, \quad \text{(A14)}$$

because the variance of G is still assumed to be 1. Also,

$$\begin{aligned}1 - h^2 = E(D_t^2) &= E(\{[w + bv][M_{t-1} - E(M|G)] + ve_{t-1}\}^2) \\ &= (w + bv)^2E(\{M_{t-1} - E(M|G)\}^2) + v^2E(e_{t-1}^2) \\ &\quad + 2v(w + bv)E(e_{t-1}D_{t-1}) \quad \text{(A15)}\end{aligned}$$

can be obtained by substituting Equation A8 for M_t, Equation A13 for $E(M|G)$ in D and canceling terms. Since e_{t-1} is uncorrelated with M_{t-1} (and therefore so is D_{t-1}), the expectation in the last term of Equation A15 is 0; and since we will now assume that the variance of e is 1 (so that we are measuring increments in u relative to the variance of e rather than E), we get

$$\begin{aligned}1 - h^2 = E(D^2) &= (w + bv)^2E([M_{t-1} - E(M|G)]^2) + v^2 \\ &= \frac{v^2}{1 - (w + bv)^2}. \quad \text{(A16)}\end{aligned}$$

Rearranging the terms in Equation A14 yields $a^2(1 - w)^2/h^2 = (1 - w - bv)^2$. Therefore

$$\frac{a(1 - w)}{h} = (1 - w - bv) \quad \text{(A17)}$$

and

$$\frac{1 - a(1 - w)}{h} = w + bv. \quad \text{(A18)}$$

Rearranging the terms in Equation A16 yields $v^2 = (1 - h^2)[1 - (w + bv)^2]$ and substituting Equation A18 into that expression yields

$$\begin{aligned}v^2 &= (1 - h^2)\left\{1 - \left[1 - \frac{a(1 - w)}{h}\right]^2\right\} \\ &= (1 - h^2)\left[\frac{2a(1 - w)}{h} - \frac{a^2(1 - w)^2}{h^2}\right]. \quad \text{(A19)}\end{aligned}$$

Taking the square root of both sides of Equation A19 and substituting for v in the multiplier implied by Equation A9 and substituting Equation A17 for the denominator yields Equation 10 from the text. Again, using the square root of the RHS of Equation A19 for the numerator and substituting the left-hand side of Equation A17 for $(1 - w - bv)$ in the denominator of the multiplier implied by Equation A10 yields Equation 11 from the text after some simplification.

Received December 3, 1999
Revision received July 7, 2000
Accepted July 9, 2000 ■