

Bacharach V.R. & Baumeister, A.A. (2000). Early generic educational intervention has no effect on intelligence and does not prevent mental retardation: The Infant Health and Development Program. *Intelligence*, 28(3), 161-192. (Sep 2000) Published by Elsevier Science Limited (ISSN: 1873-7935).

Early Generic Educational Intervention Has No Enduring Effect On Intelligence and Does Not Prevent Mental Retardation: The Infant Health and Development Program(1)

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ABSTRACT

Early intervention programs designed to increase intelligence and prevent mental retardation have long been a mainstay of pedagogical ideology. The paramount objective is to overcome intellectual disadvantage that some children experience because of unlucky draw from the genetic deck, adverse environmental exposure, and social misfortune. A number of "premier" projects completed over the past two decades have commanded wide professional and public attention. The most thorough and methodologically sophisticated is the Infant Health and Development Program (IHDP), a comprehensive preschool program to avert health and intellectual impairments sometimes associated with premature low birthweight. Despite claims that IHDP successfully raised intelligence and prevented mental retardation, close examination of project data reveal that these assertions are without foundation. IHDP failed to produce any enduring and meaningful effect on cognitive development. Among others, two primary reasons for this unsuccessful outcome are failures to consider genetic influences and to individualize intervention in terms of etiological specificity at biological and psychological levels. Prevention of premature low birthweight is more biologically plausible, more effective, and more cost-efficient. These issues are discussed in the context of broader issues concerning the nature of intelligence and its mutability. It is now time to design specific interventions that are commensurate with individual differences and the distinctive complexity of myriad problems that give rise to intellectual disadvantage.

Recently, Blair (1999) published in this Journal a wide-ranging discourse in which he alleges serious faults with our critique of the results and interpretations of the much-publicized Infant Health and Development Program (IHDP) (Baumeister & Bacharach, 1996). Endeavoring to characterize our "flawed" analyses within a broad context, Blair takes on a number of consequential but contentious issues including such topics as the heritability of intelligence, enhancing intellectual functioning through early compensatory education, mutability and immutability of intelligence, causal parsing and partitioning of genetic and environmental variance, effectiveness of special education, biobehavioral theorizing, the developmental-difference distinction, logic of clinical trials, applied versus basic science, and related public policy issues. Even the Coleman report is resurrected as a choice example of how Baumeister and Bacharach are similarly guilty of "... error of interpretation" (p. 105).

During his moral crusade, Blair encounters a lot to be alarmed about. He casts a sweeping net to capture perfidious creatures who have concocted major errors of both omission and commission. Apparently, his mission is to illustrate the considerable damage to scientific integrity and informed public policy caused by shoddy, misguided, and even ignominious analyses of intervention research.

After staging this lamentable backdrop, Blair summons Baumeister and Bacharach front and center to the public tribunal in his quixotic quest to set matters right. Although Blair finds any number of features concerning our critique that make him unhappy and that offend his sensibilities, apparently, his paramount concern is with continuing and even growing misconceptions of the authentic purposes and effectiveness of early intervention--that is, to surmount the handicaps of unlucky pedigree, adverse biological exposure, and social misfortune. He somberly declares that "The logic and scientific progress of early intervention research ... are not well understood" (p. 93). Blair numbers us among the uninformed.

Pursuant to our critique of the IHDP, we are indicted for wanton and perverse disregard for principles of respectable science and rational contemplation. Assuming gross misconception of the purposes and logic of that study, it follows that not only did we commit serious errors in re-analysis and interpretation but were manifestly inaccurate in assessment of the scientific importance and policy relevance of IHDP.

Blair cites our critical analysis as a specific example of an epidemic of crippling and "creeping misunderstanding" (p. 98) that is making matters all the more perplexing for proper interpretation and utilization of findings from intervention research. He claims: "The expectation seems to be that early intervention can eradicate differences between individuals and ensure untrammelled success in life." Anything less "... is destined to disappoint" (pp. 98-99).

Straw men make inviting targets. Who in the world has expressed such an outlandish expectation? Certainly not Jensen, as Blair intimates. Nor did we find fault with IHDP because of their failure to eradicate individual differences. We are reasonably confident that that was not their intent because it cannot be done. The very tests they used to measure intelligence are designed to maximize individual differences.

THE REACTION RANGE REVISITED

If through some social magic we could equalize environments to the extent that every child has equal and unfettered access to all good things, while eliminating differences in exposures to all deleterious things, phenotypic variation due to environmental variations would be immensely reduced. Then, as others have pointed out over the past four decades, heritability would increase with the result that individual and group differences would be even more greatly tied to heredity.

In this regard, the reaction range concept (Gottesman, 1963) is still particularly germane. As environments are modified, changes in phenotypic expression of a characteristic will not be the same for different genotypes. Depending on the genotype, some individuals may benefit from a particular feature of an environment while others may even be harmed. "Heredity is the capacity to utilize the environment in a particular way." (Fuller, 1954, p. 8)

Environmental influences on a trait are predictable probabilistically from the distribution of phenotypic reactions associated with a particular genotype. Both a main effect of genotype and a G x E interaction are implicated in that, except under the most extreme environmental conditions, the intellectually rich start out ahead and will get relatively richer as the environment is enriched. Differences exist from conception and birth, not only of class and privilege, but of adaptation, motivation, and other attributes. These are qualities that can be enhanced by environmental intervention, but never instilled (barring breakthroughs in genetic engineering).

An important implication of the reaction range model is that as the environment improves, the contribution of genetic differences to phenotypic differences enlarges--i.e., [h.sup.2] increases (Bouchard, 1997). Genetic potential is actualized more in supportive than in restrictive environments.

Although the reaction range is an appealing concept to explain G x E interaction, there are many other possible modes of G x E interactions. The reaction norm (Platt & Sanislow, 1988) is the most frequently advanced alternative, at least by those committed to the upward modifiability principle. Here, rank orders of individuals on some trait can potentially change substantially and unpredictably when the environment is altered. Response contours of different individuals will not necessarily be parallel as environmental conditions change. An essential difference is that norms of reaction are not monotonic, meaning that different environmental resources may be necessary to produce equivalent performances for different genotypes.

Fervent environmentalists, Blair among them, typically embrace the norm of reaction notion in that they assume intellectual development of children reared under disadvantageous conditions is generally impaired, with the disadvantage arising from differential opportunities associated with class and child-rearing practices. We observe in this viewpoint enormous indeterminacy because human development is not a wide-open free-ranging process, and under typical conditions, is relatively uniform. Within the range of ordinary environments that encompass American life, the course of intellectual development has not been shown to be greatly affected by social/environmental variations except under conditions of extreme deprivation or impairment (Scarr, 1993, 1996).

Those who adopt the view that measures of intelligence are culturally grounded, of course, do not accept such a deterministic interpretation (e.g., Miller, 1997). That appears to be Blair's position. Blair and others, who in defense of early intervention as an intelligence booster, and who subscribe to a culturalistic position founded on humanistic impulse, not only must clearly articulate an alternative falsifiable theory, but must also unambiguously define the independent variable and clarify their key concepts such as "environment." In short, what precisely is "early intervention?"

GENOCENTRISM

Notwithstanding accumulating evidence concerning the lack of durability and specificity of effects in generic efforts to "fix" the child, Blair appears to remain sanguine, even entirely sunny, about the prospects of early preschool intervention research in furthering scientific understanding of intelligence and, consequently, to improve cognitive functioning among children with or at risk for mental retardation. At the same time, he finds it unfortunate and regrettable that "... scientific research on intelligence has been lurking closer and closer to a model of genetic determinism" (p. 109), citing Rowe (1997) and Scarr (1992) as examples of threats to biobehavioral thinking in intervention research.

Here again, Blair's reasoning is murky, because both Scarr and Rowe have applied "biobehavioral thinking" to the ways in which genes and environment jointly influence development. Scarr (1996, 1997) has shown how Socialization Theory and Opportunity Theory, so culturally value-laden and tethered to advocacy demands, could have much greater explanatory power by the incorporation of genetically informative designs and recognition of biosocial approaches. Rowe (1997) made the case that biobehavioral considerations, particularly those arising from behavior genetics can inform public policy. In fact, he points out, as others have done, that behavior genetic analysis is a powerful method of understanding influences of environments.

We wonder if rapidly accumulating empirical evidence, particularly from the biological sciences, has anything to do with the "lurking" genocentrism that Blair considers so vexatious. Actually, genocentrism is not prowling about, but has become a respected and contributing citizen to the community of ideas and, inevitably, to public policy. Scientists such as Richard Dawkins, Robert Wright, and especially E.O. Wilson have advanced the position that realization of biological destiny has become the central imperative of human existence.

A great paradox has been exposed: How can you reconcile the premise of equality at creation with indisputable evidence that sequences of nucleotide bases create genetically heterogeneous populations, differences arising from heritable traits? Inequality of outcome is assured, even if we could environmentally equalize opportunity (and we never will because of biological determinism). Moreover, in human affairs the environment consists primarily of other people who are also products of their genes. Honest reflection demands the conclusion that all people do not begin the race through life even; nor, despite the most sincere dedication to

egalitarianism, will all people finish even. Treating all people as equals will not make them so. Nothing else is logical.

Given his steadfast allegiance to the cult of culturalism, it is of little wonder that Blair sounds a "danger" alert in that a deterministic focus ("in psychology") may produce short-sighted and premature conclusions regarding mild mental retardation and special education. It is curious that Blair is so dismayed about growing genocentrism "in psychology," as if psychology exclusively owns the issue. We perceive this malady is much more widespread. Margaret Mead's *Coming of Age in Samoa*, written in the 1920s, became a classic in anthropology and for a long time predicated the dominant orthodoxy that culture, not biology, was the primary determinant of human behavior.

Many years later, Freeman (1983), published *Margaret Mead and Samoa: The making and unmaking of an anthropological myth* (in 1996, the title became *Margaret Mead and the Heretic*), in which he shows that because of extreme cultural bias, Mead's observations were mostly in her imagination. Freeman found himself (not unlike Jensen) at the center of one of the greatest controversies in the field of anthropology because there, as in psychology, the Hundred Year War was being waged between biological and cultural determinists. The intensity of the dispute according to Brown (1998) showed "... a vigor that is readily gauged by the vociferous dismissal of Freeman's book when it received the public notice deserved by a book designed to demolish one of anthropology's hoariest myths" (p. 19).(2)

GREAT AND DESPAIRING MISUNDERSTANDINGS ACCORDING TO BLAIR

Particularly troubling to Blair is the increasing number of insidious propagators of polemic and grievous misinterpretation both of the science and the public policy implications of early intervention research. In his view, this pernicious attitude is "curiously" comparable to Jensen's famous "misstatement ... that compensatory education has been tried and failed" (p. 99). Blair persists in the all-purpose tradition of Jensenism bashing.

Actually, what Jensen said was that compensatory education "... apparently has failed ..." and that what really failed are the theories of the "average-child concept" and the "social-deprivation hypothesis" (Jensen, 1969). Jensen asserted then, as Galton before him and as Herrnstein and Murray after him, that individual differences in mental abilities require a range and diversity of environmental options and opportunities that are just as assorted as the range of abilities. Just how pernicious is it to recognize and accept the fact that individual differences interact with environmental circumstances?

In any case, in response to the argument that at the time (late 1960s), such conclusions were premature, Jensen agreed that more opportunity should be given to determine where these efforts would take us. Now we have a much better sense and knowledge of efficacy or effectiveness of enriched preschool interventions, thanks in great measure to such projects as the IHDP. When it comes to prevention and cure of mental retardation through pedagogical

methods, not much has changed over the past century and a half (Baumeister, in press; Spitz, 1986).

The hysteria and fury unleashed by Jensen's (1969) article in the *Harvard Educational Review* was greatly fanned by the popular media. Jensen's argument that native endowments of intelligence are not equally distributed across social class and races was seen as a malevolent strike at the very foundation of the towering aspirations of the Great Society, the War on Poverty, and the civil rights movement.

The enormous controversy concerning theoretical, social, and policy issues immediately following publication of that article obviously continues to this day. But if there is any one sentence in Jensen's article that captures the essence of the national outrage, it is the one about black--white differences: "The preponderance of the evidence is, in my opinion, less consistent with a strictly environmental hypothesis than with a genetic hypothesis, which, of course, does not exclude the influence of environment and its interaction with genetic factors" (Jensen, 1969, p.76). The last part of his sentence was never allowed to catch up with the first part. In fact, in a subsequent study of severely deprived black children in the rural south, Jensen (1977) did provide evidence that under extreme rearing conditions, the cumulative deficit hypothesis can explain some, but not nearly all, racial performance differences.

Later, Jensen (1998) concluded that individual and population differences in heritable traits, such as *g*, are controlled by the same genetic principles. He called this the "default hypothesis," and presented an impressive account of evidence in empirical support. Alternatively, the "dual hypothesis," favored by culturalists such as Blair, attributes variability within groups to both genetic and environmental sources, but does not permit the possibility that genetic factors contribute to mean differences between groups. No one has articulated this alternative view more succinctly than Neisser (1999, p. 132), who criticized Jensen's default hypothesis with the comment: "All groups should be assumed to have the same genetic endowment until there is clear evidence to the contrary, or until every plausible environmental explanation of the differences has been disproven." This sort of dismissive reasoning can go on ad infinitum because there is no end to alternative explanations, no matter how remote their plausibility, that might possibly account for a difference. The preponderance of scientific evidence favors the Jensen default hypothesis.

A principal thrust of Jensen's thesis is that uncritical and total allegiance to environmentalism, in order to equalize outcome, is not in the public interest. Wilson (1998, p. 142), in commenting on "idealized societies," suggested that the message from geneticists is: "Choose the society you want to promote, then prepare to live with its heritabilities." We agree with Wilson and with Zigler (1970, p. 83) who remarked three decades ago in 1968 before publication of Jensen's (in)famous review: "... our nation has more to fear from unbridled environmentalists than from those who point to such [biological] integrity as one factor in the determination of development ... a naive or premature environmentalism is just as erroneous as a naive or premature hereditarianism."

THE BELL CURVE AGAIN

We did not bring up The Bell Curve. Blair did because he apparently sees our criticism of IHDP as a reflection and continuation of the "social manifesto" promulgated by Herrnstein and Murray (1994). Currently, they are the most notorious miscreants, because their message is that our increasingly technologically driven society is ineluctably creating class divisions separating the intellectual "haves" from the "have nots." Blair, like so many others, is outraged and accusingly asserts that they are guilty of "political expediency" in their "comprehensive subversion" of the scientific analysis of intelligence (p. 94). They are condemned for basing their conclusions on a passe, "specious," and "undemocratic" interpretation that intelligence is a unidirectional, genetically determined human attribute that can be validly measured by standardized tests. The Bell Curve is "... a social manifesto ... based on an outmoded [italics added] interpretation that intelligence is a genetically determined characteristic validly measured by standard measures such as the Stanford-Binet test" (p. 94). All this is manifestly a non sequitur.

Yet a paragraph hence, Blair goes on to say there is no doubt ("beyond question," p. 95) that intelligence has a strong genetic component and, furthermore, standardized tests are valid measures of intelligence. There seems to be more than a little inconsistency here and we are not entirely sure where he stands except to say that apparently, the real issue concerning genetic determination "... is the exact size and nature of the genetic contribution" (p. 95). So, The Bell Curve is "half-right."

To declare that Herrnstein and Murray completely discount environmental variation, giving full credit to the genetic code, is indeed a distorted reading, not only of them but of every other heritability position that Blair challenges. Their point is that heritability (in the range of .4 to 0.8) constrains the extent to which enrichment of the environment can increase population intelligence. Moreover, they explicitly warned against the "ecological fallacy," generalizing from the aggregate to the individual.

The very expression of genetic differences implicated in intelligence always seems to launch a turbulent tempest, unleashing gales of unfavorable reactions both in the public media and in the professional press. Well over a century ago, Sir Frances Galton, in his Hereditary Genius, made the case that individual differences arising from genetic variations contribute in major ways to different life-long outcomes, including inequalities in social status, educational productivity, racial intelligence differences, and so on. Some reviews of Galton's books were every bit as strident as many commentaries on The Bell Curve. Galton was the prototypical serpent in the pristine garden of environmentalism.

This is certainly not the place for us to consider in detail Blair's curious conclusion or condemnation that the "Bell Curve's errors, misstatements, and inaccuracies are legion ..." (p. 95). There have been literally hundreds of well-rehearsed op-ed pieces, papers in professional journals, and books inspired by The Bell Curve (e.g., Devlin, Fienberg, Resnick, & Roeder, 1997). By no means are they all negative (e.g., Gottfredson, 1997a).

Blair's oversimplified and prejudiced representation of The Bell Curve loads heavily on a general factor of density and group factors of incongruity and political bias that permeate the entire

article. Blair gripes that "The notion of a genetically predetermined intellectual potential that is only minimally modifiable through experience is abhorrent [*italics added*]" (p. 98). Then astoundingly, without any apparent sense of contrition or contradiction, he goes on to proclaim that "Polemic and sweeping generalizations are taking the place of reasoned scientific debate" (p. 99). Just how does one measure the attribute of "abhorrent?" Evidently, one person's perverse polemics are another's unalloyed gospel.

Herrnstein and Murray (and Jensen), asserting that heritability (in the broad sense) is high, did not confuse heritability with hereditary. Phenotypic variation in a population is an expression of both genetic and non-genetic variations, based on empirical scientific facts having nothing to do with humanistic concepts, democratic or undemocratic. Variability is inherent in nature, human or otherwise. Blair's sensitivities are evidently offended by the idea that people are neither created equal nor end up that way. The very suggestion that genetics contributes to group differences in intelligence is anathema to early interventionists.

Whatever our current and evolving research tells of genetic predetermination of *g*, the fact is that general intelligence is the most robust predictor of important social behaviors and effects, educational outcome, occupational success, health, and many other consequences--more so than any other variable known in the social sciences (e.g., Gottfredson, 1997b; Lubinski & Humphreys, 1997). If IQ is unimportant or trivial, it would simply go away. At least for the foreseeable future, parents' IQ will remain a much better predictor of a child's IQ (and all that follows) than prediction from a DNA test.

THE DEVELOPING GENETICS

Much of Blair's introduction is a naive rendition of the wearisome argument about nature versus nurture. In recent years, there have been major developments, both in research and theory, concerning separate and covarying contributions of genetic variations (additive and non-additive) and environment (shared and non-shared) to expression of individual and group differences, new understanding of neurochemistry and other biological substrates, development of more powerful quantitative models, and startling advances in molecular biology (Plomin, 1997; Thompson, 1997).

In drawing inferences, behavioral geneticists traditionally have relied primarily on correlations between traits derived from individuals of varying consanguinity, particularly twins and adoptees--a top-down approach to the question of heritability of *g*. Because of recent cytogenetic and molecular genetic advances, the issue can also be addressed at a basic chemical level. It is now possible to identify DNA segments that, by coding for biosynthesis, influence phenotypic variations in many behavioral traits, including intelligence. There is not a gene for intelligence, but some that can be identified through DNA markers (linkage) and, more recently, allelic association that additively and interactively contribute to variance in a population (Plomin & Petrill, 1997).

Plomin (1997) distinguishes between the one-gene, one-disorder (OGOD) genetic perspective and the quantitative trait loci (QTL, formerly polygenic) model. The former refers to single gene disorders, in which a particular gene is both necessary and sufficient. Already over several hundred single gene disorders, many of which are phenotypically associated with mental retardation, have been tracked to the point where the specific biochemical perturbation has been identified (Baumeister & Woodley-Zanthos, 1996). As sequencing proceeds, along with gene cloning, many more genes and their particular mutations that produce variations in intelligence will be discovered and catalogued. The QTL model, on the other hand, represents the effects of multiple genes with varying effect sizes on IQ (Thompson, 1997).

This distinction is not really new. Plomin and colleagues have greatly refined its expression to prescribe a research strategy leading to a much more precise analysis of genetic contributions to phenotypic variations in intelligence. At the same time, we should emphasize that the contrast between OGOD and QTL approaches to understanding complex behavior such as intelligence is not a one-or-the-other choice. In fact, Plomin stresses that both approaches will contribute to understanding genetic influences on intelligence. The point we wish to underscore is that a single gene effect is a complicated matter involving a complex stream of enzymatic catalyzed biochemical processes that produce great phenotypic variability. Variable expressivity may be the result of a range of mutational severity, the contributing effects of other genes, as well as environmental influences. Furthermore, while a single gene may be "necessary" to create a certain disorder, it is not always "sufficient." A dichotomous distinction between QTL and OGOD models may be somewhat misleading because they both follow the same genetic and biochemical laws.

Another consideration commonly not addressed is the meaning of "disorder." Genetic mutations are expressed at different phenotypic levels, vary in severity, typically involve several organ systems, and are usually associated with multiple comorbidities, which in turn vary in degree and timing during organogenesis and later development. Genetic effects are represented along various continua, both at genotypic and phenotypic levels.

INTELLIGENCE AT THE MOLECULAR LEVEL

Recent genetic studies have identified QTL (a marker for IGF2R on Chromosome 6) associated with high general intelligence (Chorney et al., 1998) and specific gene loci (mutations in GDI1 on the X chromosome) associated with nonspecific mental retardation (D'Adamo et. al, 1998). In the case of IGF2R, the detected effect size is small (about 4 IQ points); in the case of GDI1, only two families were involved, but the impairments were severe. Even if effect sizes are small for specific allelic associations with distribution of intelligence, it would not take many polymorphisms across genes to show, at a molecular level, that general intelligence has a sizeable genetic component. Through DNA pooling across people, more sites have already been associated with high IQ.

For all the talk about genes as the basic biological element of heredity, we should be reminded that genes technically do not cause anything. As segments of DNA, genes contain the

information, a blueprint as it were, for the biosynthesis of protein and enzyme products. It is this sequence of cascading chemical events that result in variable phenotypic expression from molecular to behavioral manifestations. This is more than mere trifling with words, for these products have varied functions, affect the activity of other gene products, are variably expressed, frequently manifest themselves in more than one way, and, we emphasize, can only be signified in the context of specific environmental circumstances, broadly construed to include all exogenous events from the most molecular chemical influences to complex social structures.

Even the well-known single or major gene disorders frequently associated with low intelligence, such as inborn errors of metabolism, are characterized by variability, both at genotypic and phenotypic levels. Intelligence, in these cases, is not only influenced by the particular mutation at some genomic DNA region on one or more alleles, but by some other gene(s) controlling homeostasis.

Hyperphenylalaninemias as the Example

Practically everyone knows that classical phenylketonuria (arbitrarily defined as blood phenylalanine greater than 16.5 mg/dl) is an inborn error of metabolism that, if untreated early with a medical diet, usually produces serious mental retardation along with other somatic sequelae. If the child is treated early in life with the special low-phe diet, the IQ is usually in the normal range, although there seems to be some decrement when these children are compared with unaffected siblings or when they go off the diet later in life.

When early screening for PKU was introduced in the 1960s, a dispute arose as to how soon (and for how long) should the baby be placed on treatment. In reviewing this literature in the mid-1960s, Baumeister (1967) found cases of never-treated PKU children whose cognitive development was normal or, in some instances, exceptional. The highest IQ in this series of 167 cases was 130+ and belonged to a child who had never been placed on the special diet. This phenotypic variability led Baumeister to speculate that hydroxylase conversion of phenylalanine to tyrosine is a variable process involving different mutations and that other polygenic factors must be implicated in accounting for IQ. Maternal intelligence had to be an important predictor of IQ among PKU babies.

As it turns out, the phenylalanine hydroxylase (PAH) gene is very large, involving more than 100,000 nucleotide bases. This leaves room for many different types of mutations, mutations that will have different biochemical, neurological, and behavioral phenotypes. Over 325 different mutations have already been reported to the PAH Mutation Analysis Consortium Database (<http://www.mcgil.ca/pahdb>).

These mutations, within a single gene, are related to intelligence. In a recent study, PAH gene mutations were examined in 222 hyperphenylalaninemic women (Guttler et al., 1999). The purpose was to examine the connection between the PAH genotype and the biochemical phenotype and the extent to which this relationship affects IQ. WAIS-R IQs were available on 164 women whose genotype could be classified as to severity.

Several results of this study are of particular interest here. One is that IQ was directly related to severity of mutations involving both alleles. A second is that even with one severe allelic mutation, a mild mutation on the other allele is protective. The mean IQ of early and prolonged treated women with two severe mutations was 85.9 and for similarly treated women with one mild and one severe, the mean was 105.9. Even though the treatment (timing, degree, and duration) is important in determining IQ among people with hyperphenylalaninemia, there is a separate influence of the genotype on cognitive development. Furthermore, the authors emphasize that there was a broad range of IQs in all genotype groups, indicating the importance of the "... background genetic disposition for cognitive development ..." (p. 261), referring specifically to maternal and paternal IQ.

A COURSE CORRECTION

We are witnessing a fundamental change in the nature-nurture debate that has been progressing, ever accelerating, since the discovery that the gene is a reality, not a hypothetical construct. We are no longer solely dependent on pedigree studies, on familial resemblances, blood types, and correlational studies to make the connection between heritability and population differences in many traits, including intelligence. Molecular genetics has already turned the page to identify numerous somatic conditions that impact on intelligence: single gene, multifactorial, and chromosome disorders (Baumeister & Woodley-Zanthos, 1996). It turns out that small things do make great differences. The grand structure grows from the chemical details.

The nature of the debate is ineluctably changing because it will turn on hard scientific data, not moralist persuasion. Rear guard defensive actions, such as those mounted by Blair or Gould in *The Mismeasure of Man*, or Lewontin, Rose, and Kamin in *Not in Our Genes: Biology, Ideology, and Human Nature*, or Gardner's *Frames of Mind* are based on beliefs that are increasingly unsustainable in light of accruing evidence from the basic sciences.

INDIVIDUAL AND GROUP DIFFERENCES

The cultural view that undergirds Blair's arguments does not at all dispute the fact that phenotypic variations in intelligence arise considerably from genetic variations. Furthermore, there is no denying that environmental influences (e.g., prenatal exposure to teratogens such as alcohol, or a host of other things) can have large effects on mean differences in IQ--usually by way of depressing scores.(3) Nor is this to say that many children cannot be spared the devastating sequelae of inborn or acquired diseases by appropriate environmental interventions (e.g., a special diet for those infants with PKU or vaccinations for Hemophilus influenza type b).

Different variables are undoubtedly implicated in means and deviations from the mean. We assumed that the intent of the IHDP was to alter mean differences between treatment and control groups (i.e., increase between group variance), not to reduce variability within groups or to eradicate individual differences. As Detterman and Thompson (1997) pointed out in this

regard, it is not always clear what the intention of special intervention is. Yet the implications are profound. Detterman and Thompson observed that special education is ineffective partly because it is not individualized. Like Jensen (e.g., Jensen, 1970), they proposed different curricula for different types and levels of learning abilities. However, special educators and early interventionists favor non-categorical or generic services for children with or at risk for mental retardation and/ or learning disabilities. Indeed, some special educators claim that classification systems based on etiological principles have harmful social consequences (Goodman, 1990).

We insist that it is irresponsible to ignore scientific and clinical realities of the variable causative aspects of intellectual and learning differences, including those of biological origin (Baumeister, 1997). It makes no sense at all to ignore individual differences in cognitive attributes. But that is exactly what happens when standardized educational interventions, such as IHDP, are designed to affect an individual difference variable. In this regard, Blair seems to be in agreement: "If individual biological or psychological limiting factors on the rate of development could be identified, educational practice could be improved" (p. 98). But then, he goes on to say that Detterman and Thompson do not make clear the distinction between group and individual differences. Not so. On the contrary, that was the principal thesis from which they developed their argument. Furthermore, Blair remarks "... provision of educational experiences similar to those of normally developing children ... is most appropriate for children with mild MR" (p. 98). On the other hand, "Tailoring educational experiences to the individual needs of each learner is a laudable goal ..." (p. 98). There is some confusing inconsistency in Blair's pronouncements and predilections.

Certainly, the IHDP was not individualized. But the primary outcome measure used by IHDP was an individual difference variable--the IQ. As Spitz (1993a, p. 253) has so persuasively observed, intervention researchers "... have the worst of both worlds" when they attempt to influence an individual difference variable by an experimental manipulation. As we previously pointed out, the basic problem is that individuals do not respond to the intervention in the same way. An essential consideration is that genetic-mediated individual differences in cognitive development will not be properly understood unless the intervention explicitly takes into account the genotype X environment correlation. That failure is exactly what happened in the IHDP. The IHDP Corp of Social Engineers failed (among other things) to consider the "... Trait X Treatment interaction of individual differences in vulnerability to interventions" (Spitz, 1993a, p. 254).

Innate individual differences assure that a generic treatment applied to all individuals will have differential effects. By now, we should understand that a given intervention does not have the same effects on different children and that each child will construct his/her own environment. This is one of the considerations we addressed in our original commentary, a point that Blair does not seem to appreciate. But as Spitz (1993a, p. 255) correctly observed, "... a social policy based on artifacts rather than facts is bound to fail."

The Birthweight-Risk Gradient

About as close as he comes to acknowledging any merit in our critique that the IHDP was a general, all-purpose, one-size-fits-all intervention that failed to take into account particular individual traits (namely, maternal IQ) is his recognition that the treatment had no effect on the very low birthweight (VLBW) infants. But according to Blair, this is a rather trivial point because these children only "... make up less than 1 percent [sic] of all births ..." (p. 101). We do not see 1 per 100 at-risk births as trivial. (Actually, the U.S. rate of VLBW increased from 1.37 percent in 1996 to 1.42 percent in 1997; Ventura, Martin, Curtin, & Mathews, 1998).

Blair goes on to say that our criticism of the "... IHDP because it fails to help a segment of the population does a great disservice to the large segment of the population who benefit by it" (p. 101). At 5 years, children in the control and intervention groups had mean full-scale WPPSI IQs of 91.4 and 91.6, respectively (Brooks-Gunn et al., 1994). Heavy (2,001 to 2,500 g) babies in both the control and treatment conditions had mean IQs in the normal range with a 3.0 difference that was not significant ($p = .09$), based on IHDP data deposited with the National Auxiliary Publication Service. So where is the benefit?

As we emphasized, a great deal of longitudinal research makes clear that as a group, VLBW premature ([is less than] 1,500 g) infants are at differentially greater risk for adverse long-term outcomes. The birthweight-risk gradient is also stressed by IHDP investigators (IHDP, 1990). Nonetheless, Blair sees this as a "stick" that we used to beat the tar out of the IHDP.

We find it more than a little revealing that in planning the IHDP, "Neonatologists favored the inclusion of only [*italics added*] VLBW infants ([is less than] 1,500 g), who are known to be at greatest risk for developmental disabilities and are now surviving in increasing numbers" (Kraemer, Gross, Spiker, & Wiese-Slater, 1997, p. 62). On the other hand, there were those who claimed that "... no empirical evidence existed about [*the proposed interventions*] effectiveness [*italics added*] for LBW ([is less than] 2,500 g) children" (p. 62). So, in order to "balance" these two views, a decision was made to stratify the sample into two weight groups, but with a 2:1 ratio in favor of "lighter" babies ([is less than] 2,000 g).

Despite Blair's assertion that the VLBW infants comprise only a small proportion of low birthweight (LBW) babies ("14%"), it is evident that some of the planners were cognizant of the differential risk for adverse outcomes associated with VLBW. Reflecting on the lack of benefit for the VLBW infants, Richmond, (1997, p. xxxvii), who served on the IHDP National Advisory Committee, observed: "Because this is the group most vulnerable for developmental disabilities, this lack of effect is disappointing."

Commenting on their recruitment and retention analyses and small but significant effects of some variables (maternal education, birthweight by treatment group interaction, and others), Constantine, Haynes, Spiker, Kendall-Tackett, and Constantine (1997, p. 138) indicate that "... subgroup-specific treatment effects should be considered part of the overall interpretation of the IHDP results." That is what we thought, too.

Etiological Specificity

Another of Blair's observations that seems to favor our interpretation is his comment: "Baumeister and Bacharach are correct in considering the importance of etiology in the assessment of intervention effectiveness" (p. 101). But then he goes on to add that we were "incorrect" in criticizing the IHDP for neglecting this consideration. What is odd is that Blair seems to think that we equated birthweight strata with etiology, evidently because we made the point that by their own analyses, the IHDP had no effect on those in the high-risk birthweight group ([is less than] 1,500 g).

Now it is generally true, as Blair indicates, that prematurity and LBW "... have been resistant to nearly all efforts to reduce their rates of occurrence" (p. 102). In support of this statement, he cites Klein and Goldenberg (1990) who, like others, raised questions concerning the benefits of antenatal care. We too described evidence for effectiveness of prenatal care for reducing incidence of prematurity and LBW as "equivocal" (p. 81). The major impact of any prenatal care appears to be limited to gestationally mature infants (Alexander & Korenbrot, 1995).

Failure to reduce premature LBW through standard prenatal care does not mean that we know nothing about etiologic factors. An important consideration is that there are two distributions of birthweight: a normal distribution where those falling below the conventional (and arbitrary) standard of 2,500 g are considered LBW, and those in a residual distribution ([is less than] 1,500 g) who, as a group, are at relatively elevated risk for compromised outcomes. It is this latter distribution that is of the greater concern from a public health perspective. The same can be said of gestational age and prematurity. LBW, prematurity, and small-for-gestational age are not the same conditions. Although there are commonalities, these birth outcomes are controlled by different variables (Goldenberg, 1994). More is known about specific etiological conditions implicated in the residual distribution of premature LBW than about the general LBW distribution.

Blair acknowledges that there have been some recent advances in knowledge of etiological factors that have implications for primary prevention although he does not say what those are. In our discussion of primary prevention (e.g., p. 99), we emphasized that spontaneous preterm labor (PTL) and premature rupture of membranes (PROM) together account for most preterm births. We noted further that recent large-scale studies implicate bacterial vaginosis, genital tract colonization with microorganisms, pathogenic organisms in amniotic fluid, and chorioamnion infection as major etiological contributors to PTL and PROM. Treatment with antibiotics has been shown to reduce risk of PTL and PROM and associated co-morbidities such as low IQ. Is this not an environmental intervention? Maternal pre-eclampsia is a major cause of iatrogenic preterm birth. Low-dose aspirin prevents pre-eclampsia in some women with reduction in fetal growth retardation and prematurity. Maternal smoking, malnutrition, and drug ingestion are associated with LBW. We could give other examples. What goes on in the intrauterine environment sometimes has a considerable bearing on intellectual development, as well as other health conditions that may not appear until very much later in life (Nathanielsz, 1999). Furthermore, there is also a genetic influence on premature LBW that varies across ethnic groups (Amante, Borgiani, Gimelfarb, & Gloria-Bottini, 1996).

In our critique of the IHDP intervention strategy, we posed a simple question with respect to etiology: If LBW is associated with or causes health and developmental problems, then is it not more sensible to prevent premature LBW by focusing on specific etiologic factors than to apply some vague, imprecise, generic, and costly intervention that is demonstrably ineffective? Knowledge about etiology and specific causes permits treatments that are not only more biologically plausible, but that are more effective and inexpensive.

The distinction between primary and secondary prevention Blair (and others, such as McCarton, 1997) raises in support of IHDP, although well-ingrained in our vocabulary, is merely descriptive and is imprecise, implying as it does some sort of discontinuity. From the standpoint of prevention, it is the pathogenesis of disease that counts: Where does it begin, what are the signs, how does it unfold, how is it mediated, and where are the most cost-beneficial and efficacious points of intervention?

EFFICACY VERSUS EFFECTIVENESS IN CLINICAL TRIALS

Blair expresses unhappiness with some recent formulations and misunderstandings regarding "efficacy" as distinct from "effectiveness" of these programs. In particular, we are said to have confused this crucial distinction in that the IHDP "... was conducted to demonstrate efficacy of the intervention rather than its effectiveness" and that "In efficacy trials a decision regarding whether or not a given treatment worked is paramount" (p. 100). Then he goes on to add that the IHDP "... demonstrated the intervention to be efficacious for the general population of low birth weight infants" (p. 101). This is an especially flagrant misstatement of the outcome, both with respect to our analyses and their own reported outcomes at 5 and 8 years. In this context, we should again emphasize that the IHDP was not a population-based study nor were the families selected at random. Blair may wish to assume that these samples (at eight selected sites) were representative of the general population of LBW infants. But data from epidemiological studies tell us otherwise.

Furthermore, there was a "drift" across sites in terms of enrollment, baseline characteristics, and outcome. Control infants at the Harvard site had an average IQ at 3 years of 97 as contrasted with 68 at the Miami site, even though mean birthweights and gestational ages were equal (IHDP, 1990). Mothers differed markedly on variables such as IQ, education, and race.

The distinction between "efficacy" and "effectiveness," so central and essential to Blair's argument, is not the grand and decisive question in clinical trials research that Blair would have us believe, as a review of current discussions of the logic of clinical trials clearly reveals (Friis & Sellers, 1999; Meinhart, 1986). The contrast between efficacy and effectiveness offered by Blair is so vague and shallow as to be of no practical significance with respect to IHDP.

We certainly agree that clinical trials, although not without their limitations, are generally considered the best study design to evaluate efficacy of a prevention or therapeutic intervention particularly when randomization is a major component. However, randomized clinical trials are difficult to conduct, are expensive, often present ethical challenges, and are subject to

confounds from participant attrition and uncontrolled variables. The IHDP was not immune to these problems.

Apparently, our explicit reference to the IHDP as an "efficacy" trial (p. 82) does not satisfy Blair because we claimed, after a re-analysis of their data at 3 years and after reviewing their findings at 5 years, that the treatment did not have the effect they claimed. That is, we asked the questions: Did it work? If so, for whom and how durable was the effect? If an effect was observed, what is its meaning? Are there other explanations that might account for whatever effect was reported? Were there inconsistencies in their own reported data?

By whatever name one wishes to call this trial, the fact remains that the IHDP simply did not produce meaningful and lasting effects on cognitive development. His claim that "The controlled trial demonstrated the intervention to be efficacious for the general population of low birth weight infants" (p. 101) is not only unsubstantiated, but flies in the face of overwhelming evidence to the contrary.

IHDP was an expensive randomized clinical trial that failed to show any enduring effects of early intervention, a point to which we return. In her extensive and careful review of intervention research, Farran (in press) concludes in regard to the IHDP: "The lack of effect for such an ambitious, well mn, and expensive program was unanticipated and troubling."

Policy Issues, Effectiveness-Efficacy and IHDP

Because we supposedly did not understand the difference between "efficacy" and "effectiveness," not only were our analyses and conclusions erroneous, but our pronouncements regarding policy implications of IHDP are inappropriate because, putting carts before horses, we questioned effectiveness of IHDP rather than its efficacy.

Despite Blair's construction of events, we are not the ones who asserted that the IHDP "... has vast policy relevance...." (p. 103). They did so repeatedly in an unabashed manner not constrained by semantic niceties. He is correct in that we countered "... that the IHDP has no relevance to policy ..." (p. 103), except insofar as the results demonstrate that this is the wrong approach. As we observed, in this sense, the IHDP does have important policy ramifications.

Blair is relentless, returning to the efficacy-effectiveness theme several times. He says that this distinction is a major feature underlying the logic of clinical and community thais and that a two-stage process is necessary. First, efficacy must be established to determine whether a procedure or treatment works. Once efficacy is established, then effectiveness can be pursued to identify factors related to different treatment outcomes.

Effectiveness studies, according to Blair, are conducted to identify "... real-world variation in effects" (p. 100). Could it be that IHDP, as an "efficacy" trial, was not concerned with "real-world" effects? Are LBW and intelligence not "real-world variations?"

Apparently, IHDP investigators were not constrained either by this distinction because they repeatedly refer to effectiveness. Authors of the many IHDP reports clearly intend to influence public policy and do not hesitate to talk about "effectiveness" (e.g., IHDP, 1990). They leave little doubt about the social and educational implications of their work: "The results of this study [IHDP] are especially timely and relevant to the concerns at the federal and state levels for providing appropriate interventions for children at risk for developmental delay" (Gross, 1997, p. 153; IHDP, 1990, p. 3041). In an accompanying editorial, Richmond (1990, p. 3069) concluded that: "the results have considerable clinical and potential public policy significance." This is a theme repeated many times in numerous publications of those affiliated with IHDP. Even a major health entity of the U.S. Government got into the act. The Centers for Disease Control and Prevention issued a "Request for Proposal" to implement the IHDP program in a number of states.

For Blair to denounce us for discussing public policy issues that IHDP investigators have emphasized in so many publications (and that he himself has endorsed) is plainly ludicrous, by whatever designation he wishes to apply to this trial. Because "... an effectiveness trial might be some time in coming, it is reasonable to estimate information on effectiveness using the IHDP database" (p. 102). What a nifty two-step shuffle. It is acceptable for them to wax enthusiastically about the social import of their work, but not for us to comment.

ABECEDARIAN AS THE MODEL

According to Blair, effectiveness studies are based on efficacy trials. So if we follow his enigmatic reasoning, then IHDP must necessarily be considered as an effectiveness study because purportedly, it was based on the Abecedarian Project. To quote: "In the Abecedarian Project on which the IHDP was based ..." (Brooks-Gunn et al., 1994; p. 1260). And from Blair: "... the IHDP was itself a replication and expansion of its predecessor, the Abecedarian Project ..." (p. 102). Effectiveness studies address how well a treatment works in different populations-- in this case, different subpopulations at risk for mental retardation. In the Abecedarian Project, the population of interest consists of children purportedly at risk for mental retardation because of social disadvantage; in the case of the IHDP, different birthweight groups. The intervention, according to the IHDP investigators, was modeled after Abecedarian. So is the IHDP not an extension to another subgroup (LBW children) of a "proven" method for averting mild mental retardation? We aver that Blair is engaging in a shallow diversion of slippery and confusing obfuscation.

Actually, it is an uncomfortable stretch for Blair (and the IHDP investigators) to claim that the IHDP was "... a replication and extension of the findings of its predecessor, the Abecedarian Project ..." (p. 102). The principal purpose of the Abecedarian Project was to avert mild mental retardation by providing enriched and intensive early intervention to young children at risk owing to sociocultural disadvantage. The IHDP was about premature LBW, conditions that always involve biological antecedents. Apparently, IHDP was conceived and given birth by Abecedarian under the guise that this early preschool intervention has conclusively demonstrated that large

IQ gains (in the order of 8 to 20 points) can be achieved by children at risk for mental retardation owing to their social disadvantage.

Although many references are made to the Abecedarian Project as the model for IHDP, a more direct methodological link is with Project CARE carried out by the same investigators (Ramey, Bryant, Sparling, & Wasik, 1985). The CARE Project which "verified the efficacy" of a combination intervention, "... using three modes of intervention, was the basis for the IHDP approach" (Ramey, Sparling, Bryant, & Wasik, 1997, p. 17). The "efficacy" or "effectiveness" of CARE is dubious, as we shall show.

We emphasize that inherent in the strategy adopted by IHDP is the assumption that conditions giving rise to birth complications such as prematurity and LBW are the same as those implicated in social disadvantage. This is a bad assumption. There is strong and convincing evidence that variables affecting developmental status of disadvantaged or poor NBW children are different in type and/or degree than those modifying outcomes of LBW children of less intellectually competent mothers (McGauhey, Starfield, Alexander, & Ensminger, 1991; Siegel, 1982). The social risk profile of LBW children is different, applying not only to specific health measures, but to broader performance indicators such as school achievement (Starfield, 1992). In view of these considerations, the Abecedarian (and CARE) intervention models and strategies were inappropriate in the IHDP.

OTHER FLAGRANT MISINTERPRETATIONS

Blair is not the only antagonist to bong and bang The Bell Curve with the club of early intervention studies, particularly Abecedarian (e.g., Sternberg, 1995). There are others who stoutly and enthusiastically harmonize with Blair the refrain that IHDP and the Abecedarian Project conclusively demonstrate "efficacy" or "effectiveness" of early intervention for children at risk for diminished intelligence.

As an example of a particularly egregious misrepresentation of the results of the Abecedarian Project and the IHDP, Wahlsten (1997) presents a figure (Fig. 4.6, p. 83) in which he claims that IQ differences between control and experimental groups were 13.7 for the Abecedarian Project (at 48 months) and 14.5 for IHDP (at 3 years, using just those intervention children [in the 2,001 to 2,500 g range] who received 500 home visits plus days in day care).

These data, together with his creative construction of results from Project CARE and the Mother-Infant Transaction Program (for LBW children in Vermont), lead Wahlsten to declare triumphantly: "These four studies of disadvantaged children in the United States demonstrate conclusively that enriched educational experiences early in life can substantially improve performance on IQ tests" and that "... they indicate a substantial elevation in intelligence of children in an entire country could be achieved ..." (pp. 83-84). To cap off his reasoning, Wahlsten draws an analogy with space travel. Referring to advances in aeronautical engineering that allowed us to escape the shackles of gravity and to reach Mars, Wahlsten proclaims "Gradually and against great resistance from behavior genetics, developmental

psychology is discovering ways to lift humanity from the fetters of poverty" (p. 85). We find it difficult to imagine how behavioral geneticists are responsible for "poverty."

Wahlsten, like Blair, also lashes out at Herrnstein and Murray in take-no-prisoners combat, but he surely is careful to avoid causalities due to friendly fire. So the studies he cites are spared the analytical intensity he applies to others less congenial to his conviction. Perhaps that explains why he does not mention that in the Vermont study, the treatment subjects were initially more socially advantaged than the controls. The authors themselves acknowledge this could be a troublesome confound, leading them deliberately to refer to the "apparent" effect of intervention (Achenbach, Phares, Howell, Rauh, & Nurcombe, 1990).

Wahlsten does not bother to point out that at 48 and 54 months, the group of children in Project CARE, who received a combination of center-based and family home visitation, did no better than controls. Both of these groups were superior to the group that received only home visitation. In other words, the proper interpretation would be that the center-based intervention had no effect and that the home intervention had a negative effect. While this may have been a fluke, results from Project CARE led Farran (in press) to observe: "Even so, it is sobering to think that it [family education] might actually have slowed the children's development, proof that one needs to be quite cautious in intervening in high risk situations."

Based on our multivariate analysis of maternal IQ, maternal age, family income, HOME scores, and 3-year Stanford-Binet scores, we (Bacharach & Baumeister, 1998) were led to conclude that standardized family interventions (in the IHDP) intended to improve cognitive outcome will have only very minimal, perhaps even detrimental, effects on children of poor and intellectually comprised mothers. There is the risk of "intervention-dependence" particularly among intellectual and financially impoverished mothers, as Stone, Bendell, and Field (1988) warned pursuant to their finding that early family interventions failed to produce any long-term effects on normal-birth-weight or LBW children born to poor teenage mothers.

This exercise by Wahlsten shows just how adeptly the life of an endangered theory can be spared by judicious selection of data and deft interpretations. Thomas Huxley observed: "You have no notion of the intrigues that go on in the blessed world of science."

PROBLEMS WITH THE ABECEDARIAN PARENT

Notwithstanding all the exorbitant praise from culturists, there are numerous and profound problems with the Abecedarian Project as Spitz (1986, 1992, 1993b, 1999) has trenchantly detailed. Farran (in press) also counseled "some caution" in interpretation of results from publications describing results of the Abecedarian Project. For instance, rather than reporting means and standard deviations on assessment measures, graphical data are often presented "... in figures which have a tendency to inflate group differences." We think of this ingenious technique of data analysis as "the ordinate stretch effect-size calculation."

In addition to some artful analysis procedures, numbers of children assessed vary across different measures making it difficult to determine group means. Numbers of subjects reported

also varies across publications. Again, Farran states: "At a minimum some explanation of the differences would be helpful. These variations can lead to an impression of the manipulation [italics added] of readers rather than straightforward reporting ..."

Analytical incongruities notwithstanding, a major unexpected problem is that the control children did not behave according to the plan in that their mean IQ did not fall within the range of mental retardation (a five-point IQ difference at 12 years, 94 vs. 89). Spitz (1999, p. 282) recently observed that this is hardly a "... propitious outcome as far as the Project was concerned, because the Project's purpose was to prevent mental retardation.... " The Milwaukee Project, for all its problems, was at least conceived on the basis of a far better risk indicator for mental retardation: low maternal IQ.

At age 15, the 4.6 point WISC-R IQ difference in the Abecedarian Project was not statistically significant (Farran, in press). The mean ability test score of the intervention group was somewhat higher than the control group's at 6 months, shortly after they entered the Project. Although their score remained in the average range throughout, by 18 months, it was appreciably higher than the control group's only because the mean score of the control group had declined until it began, by 48 months, a steady recovery. In general, the experimental group never increased in IQ, but remained in the average range. Nor did the control group decline into mental retardation. The final IQ difference, not incidentally, was about the same as the difference at 6 months; a difference that Ramey, Yeates, and Short (1984) admit cannot be attributed to the intervention.

In regard to this conspicuous lack of enduring effect in the Abecedarian Project, Spitz (1999, p. 283) raised a question and then proceeded to answer it: "What happened during those first 1.6 months at the day care centre to produce an effect worth 6 points, whereas an additional 4 1/2 years of massive intervention ended with virtually no effect? It seems to me that it is not unreasonable to infer that nothing happened, but rather, some initial difference in the control and intervention groups had (by chance) escaped randomisation, and revealed itself at six months of age." We found similar problems with the IHDP.

After a scrupulous, detailed, and even-handed reevaluation of both the Abecedarian and IHDP projects Bruer (1999, p. 172) also concluded they "... hardly support a claim that early interventions have substantial, long-lasting, and positive effects on lifelong intelligence and school achievement." He goes on to add: "One of the greatest abuses to the cause of children is misrepresenting the effects of early-intervention programs" (p. 173). The pathetic collective tragedy arising from such naive environmentalism and misguided egalitarianism is that all the wasted resources could be used much more productively to target specific health-related and social problems with some beneficial results, on a more realistic and less idealistic and grandiose scale to be sure.

Precious little in either project supports Blair's assertion that "Examination of individual and group differences in response to early intervention makes it very clear that mild mental retardation is preventable" (p. 108). This is a blatantly nonsensical statement. Is Blair referring to all mild mental retardation? Some proportion? Mild mental retardation associated with certain types of exposure, such as lead or virus? Those without demonstrable brain damage? Familial

retardation? Who is he talking about? There are many conditions causing mild and serious mental retardation, both genetic and acquired, that can be prevented. For a few examples, along with measures of cost effectiveness, see Baumeister, Bacharach, and Baumeister (1997). Our argument is that mild mental retardation cannot be prevented in any meaningful manner by generic, broad-brush, early educational intervention programs.

ON VARIANCE ASSOCIATED WITH MATERNAL IQ AND INTERVENTION

Another of Blair's principal criticisms of our re-analysis of IHDP is that we inappropriately partitioned child IQ variance between maternal IQ and intervention effects. At this point, we should correct the erroneous statement by Blair that we were the ones who originally referred to the Peabody Picture Vocabulary test as a measure of maternal IQ in the IHDP. Brooks-Gunn and Benasich (1992, p. 66) stated that "IHDP wanted to measure maternal IQ ..." because "maternal IQ could serve as an important covariate in some statistical analyses." The choice of the PPVT as a measure of maternal intelligence was theirs, not ours. In any case, the PPVT is demonstrably a good measure of *g*.

We reported that 50 percent of child IQ variance is associated with maternal IQ variance in the IHDP, whereas the zero-order correlation between intervention and child IQ at 36 months accounted for only 4.4 percent of child IQ variance. That is the fact. We did not square the maternal-child IQ variance because it is a kinship correlation. Blair says that this comparison is inappropriate because these terms cannot be compared unless both terms are squared. In our summary description of results (p. 86 and Table 1), the unsquared correlations involving six predictor variables are compared showing that maternal IQ was the most potent ($r = .50$) and that intervention ranked fifth ($r = .21$).

We remind Blair that these were zero-order correlations. For a more thorough account of our multivariate analytical strategy, the reader may wish to examine the hierarchical analyses described in our article. We never conducted a regression analysis in which we compared squared intervention effects with unsquared maternal IQ effects. In each hierarchical procedure, we reported covariance statistical values (partial correlations). Furthermore, we presented multiple R-values with and without maternal IQ in the equation. We did not "partition" these variances except when examining relative contributions of maternal education and IQ to child IQ. In this case, between-groups sources of variations were not compared with within-groups sources of variation.

In this context, it is peculiar that Blair brings up Carver's (1975) re-analysis of the Coleman Report. One of Carver's main arguments was that some psychological assessment instruments produce scores (such as IQs) that are impervious to intervention. This, of course, is our contention as well. Carver's other main point relates to evaluations of change or gain scores, basing his reasoning on the assumption that there are initial group differences that need to be reduced or eliminated. No such assumption operates in the case of the IHDP where they assumed that the treatment and control groups were not initially different. Furthermore, Carver was criticizing a correlational study; the IHDP was a randomized control study.

The Home Environment

In the tradition of Socialization Theory, a great deal of emphasis is always placed on home environment. Blair maintains that the home environment as measured by the HOME instrument exerts a "large" effect, "... accounting for 20 percent [sic] of the variance" (p. 104). He is, of course, referring to the zero-order correlation. This zero-order correlation ignores confounding of genetic with environmental effects. How can we measure the influence of family environment without taking into account well-established genetic differences? We are ill-served by a stratagem that exclusively focuses on causes that allegedly can be manipulated to the exclusion of those causes, manipulable or not, that have greater explanatory power. Behavior-genetic theory provides both the theoretical and methodological means to quantitatively parse complex environmental and genetic effects. Socialization theory does not.

We showed (Baumeister & Bachrach, 1996, Fig. 1, p. 88) that HOME scores are greatly influenced by maternal IQ. Furthermore, in a subsequent study of IHDP (Bacharach & Baumeister, 1998), we conducted another hierarchical analysis including maternal IQ, maternal age at parturition, family income, and quality of home (HOME scores) entered in that order into an equation to predict child IQ at 36 months. In this analysis, maternal IQ had large direct and indirect effects, mediated by family income and HOME. After controlling for the other variables in the equation, home environment accounted for only about 2 percent of child IQ variance.

The family intervention model, as used in the IHDP, simply had little effect on outcome. We conclude from this and other studies of home-based intervention (e.g., St. Pierre, Layzer, & Barnes, 1995; Wasik, Ramey, Bryant, & Sparling, 1990; Watson, Kirby, Kelleher, & Bradley, 1996) that family-focused support programs, at best, have very weak and transient effects and that intellectually compromised parents simply cannot use the information provided them, even assuming the information might otherwise be of help. In a recent report of the results of the Comprehensive Child Development Program (a two-generation program involving 4,410 families for 5 years), St. Pierre and Layzer (1999, p. 134) concluded: "... the results of this evaluation do not support home visiting as an effective means of social service delivery and parenting education for low-income families."

Maternal Intelligence

For prediction of child IQ, maternal intelligence overwhelms other variables and, like child IQ, is extremely resistant to upward modifiability. In this regard, "effects" of treatment are conditional on the intellectual competency of parents, especially mothers. Those women with low IQs are more likely to have a child with low IQ. Unfortunately, they are less susceptible to parent training programs than more intellectually able parents who, in any case, do not require any such intervention.

We did, as Blair indicates, "... make a great deal about the importance of maternal IQ ... and [we did] criticize the IHDP planners for using maternal education rather than the PPVT scores in the final analysis" (p. 105). He adds that by our own analysis, we showed that it does not matter

which of these is used as an initial status variable. That assertion is just plain wrong because we did explicitly show just how great the difference is.

Controlling for maternal IQ, the partial correlation of maternal education and child IQ at 36 months was .09. Controlling for maternal education, the correlation between maternal and child IQ was .43. Furthermore, our regression analyses (including intervention, birthweight, and the intervention by birthweight interaction) revealed that with maternal IQ in the equation, the squared multiple R was .31; without maternal IQ, the squared multiple R was .07. At that point, we judged effects of the intervention package to be "trivial" (p. 88), all the more so because even this small effect was subject to confounding.

Intensity Effects

Blair, Ramey, Wahlsten, and others make pronouncements about the crucial importance of intervention intensity. In response, we broke out the three components--home visits, parents' meetings beginning at 12 months, and day care days--of the intervention bundle to examine their separate effects on child IQ at 36 months. Only parents' meetings were significantly related to child IQ. Furthermore, children of those mothers who attended the most meetings had significantly higher Bayley scores at 12 months, before the mothers even attended any meetings.

So how does Blair explain this? He does so by suggesting that the child Bayley score difference is attributable to home visitation rather than selection bias. But home visits had no effect on child IQ, as we demonstrated. Also, why is it that families with relatively bright babies were visited more often than families with less able babies? Would that circumstance not lead one to predict a positive correlation between number of visits and IQ? Which is cause and which is effect? Given that the IHDP was modeled after the Abecedarian Project, we also wonder how Blair can reconcile his interpretation with the statement made by Ramey et al. (1984, p. 1922): "We found that developmental functions were alterable only after 12 months." Furthermore, as we emphasized earlier, in the CARE Project, children assigned to home intervention actually performed worse than controls.

Attrition

In any longitudinal study, including randomized clinical trials, recruitment bias and attrition represent serious threats to validity and interpretations of outcomes. Accordingly, a great deal of attention was directed at these issues during the planning phase and execution of the project (Constantine et al., 1997). In the case of the IHDP, these considerations become particularly salient in light of small and dubious effect sizes and because, as we demonstrated, results are confounded by uncontrolled variables (such as maternal IQ) known to be related to outcome--variables that exerted differential effects once the project was underway.

Of the 1,302 families deemed eligible for enrollment in the program, 274 (21%) refused to give consent. After consent was granted and subjects were randomly assigned to conditions, 26 (2%) were not enrolled "... because, for a variety of reasons, group assignment could not be presented" (Constantine et al., 1997, p. 128). In addition, 17 (1.3%) rejected group assignment (mostly to the intervention condition). Enrollment rates differed significantly across sites. Blacks and Hispanics were more likely to enroll than whites/others. Race/ethnicity and birthweight are variables known to be associated with child IQ. (Data on maternal education were not available at this point.) Mothers of heavier infants were significantly less likely to enroll. Another 72 dropped out of the study, many (n = 30) in the early phase at or before the 4-month assessment. Mothers with lower levels of education were significantly more likely to drop out.

In predicting retention, two variables interacted significantly with treatment condition: birthweight and maternal age. A greater number of VLBW children were retained in the treatment group, while for the heavier infants, a greater proportion was retained in the control condition. Children of older mothers were more likely to be retained in the control group; infants of younger mothers ([is less than] 18 years) were more apt to be retained in the treatment group.

Whatever the effects of these variables, they can be expected to bias outcome in some manner. Attrition was not random, either at the point of enrollment or during the course of the study. To which real-world population can these results be generalized? Only a hypothetical population defined in terms of the sample.

There is still another type of attrition bias that is equally or even more problematic: variable participation in the program. For instance, 14 percent of children in the intervention condition never attended the child development center. We showed that those who attended the most (and had higher IQs at 3 years) also had higher Bayley scores before the day program began.

In this connection, Blair finds groundless our criticism of the participation study reported by Ramey et al. (1992) in which they allegedly showed that there was a nine-fold ("8.9 to be exact") reduction in the number of children with mental retardation among those who participated the most intensively in the program. We attributed this result to a selection effect and presented data to support that attribution.

Although we have already addressed the issue of policy implications, one other point that Blair makes in this regard warrants further comment here. He refers to our criticism of Ramey et al. (1992) as "rhetoric" in that we made the point that their conclusion "... is laden with important policy implications ..." (p. 89). Blair says that because Ramey et al. "... are appropriately circumspect in their discussion of policy implications" (p. 103), we unjustly discredited the finding. We demur. The finding is discredited on the basis of data, not because they did or did not speak to policy implications. Nor were they entirely moot as to their impressions of the importance of their findings, as a look at their discussion will reveal. Also see Ramey and Ramey (1992).

Notwithstanding Blair's reproach, we still are impressed that a nine-fold reduction in incidence of mental retardation represents a contribution of immense proportions worthy of public attention.

The problem is, as we demonstrated previously, that intensity study is so badly flawed it is not worth serious regard.

An even more insidious and problematic variant of attrition occurred because different numbers of children were evaluated at separate points in time. In addition, data on different numbers of subjects were reported for different measures even at the same assessment point (e.g., IHDP, 1990).

Extreme and Intrusive Intensity

In various publications (e.g., Ramey et al., 1992; Ramey & Ramey, 1992), the point is stressed and reiterated that the benefits of IHDP and other early intervention programs are directly related to "intensity" of subject participation. It is alleged that the more intense, the higher the IQ and the better the related outcomes. Even assuming that is true--which we do not--troublesome and disturbing ethical, social, political, and even legal issues are raised by the prospect that families with children deemed by some "authority" to be "at risk" for adverse developmental consequence, should consent to surrender themselves to control by vested social handlers who purportedly know what is best for them and their children. Now, of course, it is true that participants in these intervention projects were "volunteers" who had the absolute, if not the relative, right to decline participation. But this begs the question as to what exactly is "informed consent," especially for those families whose choices may be constrained by financial circumstance, social condition, or limited abilities to truly comprehend what they are consenting to; who may be intimidated by professional authority, or who would be only too eager to shift child-rearing responsibility to some agency.

One is reminded of Watson's famous statement of so many years ago that given a dozen "healthy" infants, he would make of them whatever might be wished. Watson, no doubt, was quite aware at the time in making such an outlandish declaration that no one would take him up on it. But now, we confront a much more insidious form of big brotherism in which some vulnerable families may be induced, bribed, misled, or cajoled to enlist themselves and their infant children to participate in programs of dubious benefit, such as early compensatory intervention, with greater rewards attached to greater involvement, but with a concomitant reduction of independence. The very concept on program intensity as described by Ramey and collaborators does, in fact, mean seizing control from families. This is not the egalitarian scenario it purports to be. Carried to an extreme, such excesses of egalitarianism are as ethically daunting as creating "designer" babies through genetic manipulations, only not as effective.

EFFECT OF THE INTERVENTION

The primary motivation behind IHDP, as articulated by Blair and by us in our critique, was to provide early intensive educational interventions over the first 3 years of life "... to improve cognitive and behavioral outcomes of children born both preterm and low birth weight" (p. 99).

This theme is presented in virtually every publication emanating from the project. So we can all agree as to the fundamental purpose. The difference is that IHDP investigators, including Blair, maintain that they achieved their objective. We claim they did not.

Certainly, the purpose was not to show an IQ effect at 3 years and let it go at that. Unquestionably, their intention was to have a more enduring influence on cognitive development. According to IHDP (1990, p. 3041): "We conclude that this comprehensive and intensive early intervention program shows substantive promise of decreasing the number of LBW premature infants at risk for later [italics added] developmental disability." McCarton et al. (1997, p. 130) state: "Although it was hypothesized that the effects of early intervention would be most evident in the prevention of school failure, no differences were found in the percentage of children who repeated a grade or who were placed in special education." After reviewing the results of the Abecedarian Project, Project CARE, and IHDP, Ramey and Ramey (1992, p. 341) concluded: "Health and education policy is being viewed with an eye toward the likelihood that early interventions will be a positive factor in leading to long-term benefits in developmental status, educational progress, and, ultimately, constructive participation in the social and economic life of our society."

Despite the clear and reasonable intention to influence long-term outcome Blair insists "... the study was designed to test explicitly the question of whether or not the specified intervention program would have an impact on the intellectual development over the first three years ..." (pp. 99-100). Why should anyone wish simply to raise IQ scores at 3 years by providing enriched family and education experiences and stop there, particularly in light of the well-documented wash-out effect? IHDP investigators were quite aware of regression, as they have indicated in numerous publications. The 3-year IQ is a moderately good predictor of later IQ, which, in turn, is a strong predictor of socially and educationally meaningful criteria. If the only interest was to affect outcome at 3 years, why is it that these children were followed at 5 and 8 years? Because it makes sense to ask about durability of effects, IHDP investigators were quite correct in their efforts to determine how stable any IQ effect might be, and whether the intervention has any broad transfer beyond IQ.

Blair imparts the impression that IHDP had large and important effects on outcome at 3 years. By their own preset effect-size criterion of .5, only one effect reached that level: Stanford-Binet IQ for the heavier group (2,001-2,500 g). Based on our previous re-analysis of their data, in which we included maternal IQ, we questioned the meaning of this finding. There is no point in repeating ourselves.

Fig. 1 in the Blair article shows that 3-year child IQ regressed on maternal PPVT IQ for both treatment and control groups. His points are that maternal IQ and child IQ are correlated for both groups, and that the intercept, reflecting treatment effects, is different between groups. We are not sure what Blair intends to accomplish with this figure, because it represents the same data we presented. The fact is that the overall treatment condition accounted for merely 4.4 percent of child IQ variance at 3 years, not taking into account confounding variables.

Only in passing does Blair mention the 5- and 8-year outcome data, which he correctly describes as "... largely negative ..." (p. 99). He asserts this cheerless outcome leaves room for

"... considerable misunderstanding as to what IHDP accomplished and why" (p. 99), making it all sound like a great scientific mystery that only early interventionists seem to appreciate.

When the IHDP presented their 5-year follow-up analysis (Brooks-Gunn et al., 1994), one general conclusion comes through distinctly: Whatever effects they claim to have obtained when the children were 3 years of age had evaporated. WPPSI full-scale scores were 91.6 and 91.4 for the treatment and control groups, respectively. The one small remaining effect they attempted to salvage (as reported by Brooks-Gunn et al., 1994) was a 3.7 mean IQ difference ($p = .03$) favoring heavier infants in the treatment group. But when we examined their summary data deposited with the National Auxiliary Publication Service (NAPS), the mean difference between heavier birthweight babies was 3.0 points ($p = .09$).

We (Baumeister & Bacharach, 1997) maintained that the NAPS data were superior in that they represented the same children tested at both points in time. Simple arithmetic reveals that Brooks-Gunn et al. (1994) included children at 5 years who had not been tested when they were 3 years. Furthermore, at the 8-year follow-up (McCarton et al., 1997), where the reported difference (for the heavier infants) was still about 4 IQ points, still other numbers of children were tested. We concluded that there is a bias in these data simply too blatant to be ignored and that could easily account for any small difference, statistically significant or not.

In her response to this criticism, McCarton (1997, p. 1279) justified the different sample sizes by saying that "... we followed the intention-to-treat principle used in randomized controlled trials to minimize bias: every available randomized child was analyzed without any post-hoc exclusions." This statement defies logic. Attrition, for whatever reason, creates bias. An RCT trial does not excuse departures from suitable analysis and, at the very least, sampling irregularities should temper their conclusions. Perhaps, if the IQ differences between groups were enormous, one might reasonably conclude that this sampling breach might not have injected a bias so powerful as to completely distort their findings. But in this case, the difference between groups (of heavier infants) was holding on by a slim thread. Moreover, they do not attempt to explain the difference between what they reported in *The Journal of the American Medical Association* and what they put on file with NAPS. But the disparity is such that these two data sources lead to very dissimilar conclusions. We maintain that his omission of this critical issue attests to the selective and highly partisan nature of Blair's reasoning.

Given that at the 5-year assessment the results were so unimpressive, the possibility has been raised that "... the intervention services need to be of longer duration than 3 years to maintain the magnitude of the intervention group's IQ advantage seen at 3 years" (Brooks-Gunn et al., 1994, p. 1260). Whatever other quarrels we might have with the IHDP strategy, increasing duration of intervention is a highly dubious proposition because the intervention subjects showed only a 2.0 IQ decline from 3 to 5 years. The control children, on the other hand, rebounded by 7.2 points (Brooks-Gunn et al., 1994, Table 1; Also see our Fig. 3, p. 94). Rebound effects have been reported in many early intervention studies (Farran, in press). Furthermore, Wilson (1985) found that among twins, one born smaller than the other, LBW did not confer any long-term IQ disparity by the time they were in school. As we have shown in the

case of IHDP, Wilson also concluded that maternal intellectual competence was the decisive factor in predicting the final IQ among the twins.

So we must ask: If the control subjects are showing substantial gains over time, without the benefit of IHDP, while the treatment children lose just a little, if any, how can a more extended (and hugely expensive) IHDP intervention be justified? The IHDP may have provided some (heavier) babies with a bit of an edge by moving their starting line ahead a tad. But within 2 years, they got winded and the controls got stronger. By 5 years the race was even. At age 8, both groups were about 2/3 of a standard deviation below the normative mean. Was this attributable to their LBW? We expect that some VLBW children did in fact pull the mean down. But a much more likely source of the average lower IQ of these children was their mothers' lower-than-average IQ.

As practically everyone knows, including Ramey and Ramey (1992), maternal IQ is the variable with the most potent direct genetic and indirect environmentally mediated effects on child IQ. On a scientific basis, we find it difficult to understand why the IHDPers did not include maternal IQ as an initial status variable. But in light of the sundry hot-house issues that set Blair ablaze at the beginning of his discourse, along with a prevailing interventionist disdain for behavior genetics as an explanation of IQ variability, their exclusion of maternal IQ is much more readily understandable as a manifestation of political correctness. This is an example of "political expediency" that Blair finds so offensive.

BIOBEHAVIORAL THEORIZING

We not only agree with Blair that advances in biobehavioral theory have important implications for research on mental retardation, but that biobehavioral synthesis is vital to our understanding of mental retardation (Baumeister, 1997). Notwithstanding our general agreement on this matter, we are not entirely sure what Blair means when he says "The key implication of the synthesis [presumably referring to biobehavioral constructs] for research on mild MR, however, is that previously deterministic thinking on the role of genotype in development is being replaced with a dynamic non-linear systems model" (p. 108). How is such a "model" supported by findings that events in the intrauterine environment can influence intelligence in "coaction" with genes? This is hardly a novel revelation that inspires biobehavioral analysis of intelligence differences. How does biobehavioral theory lead to a "new understanding of the way in which ontogeny is related to phylogeny" (p. 108)? What is the point? In any case, the topic of biobehavioral theory is far too expansive to be addressed here in any detail.

THE DEVELOPMENT-DIFFERENCE CONTROVERSY

Here again, Blair ventures into a theoretical and methodological jungle without the proper provisions. The distinction has a long and acrimonious history, with important bearings on social, educational, and even legal conceptions of intellectual differences (Elliott, 1987). Furthermore, the development-difference differentiation implicates intelligence theory and

definition of mental retardation, structural features versus control processes, research design (e.g., MA vs. CA matches), and intervention strategies, especially generality versus etiological specificity. (Even research grant funding decisions have turned on the distinction. It was, at one time, adaptive for research grant applicants proposing comparative studies to include both CA and MA matches in their protocols submitted for "peer" review).

Providing a superficial explanation, Blair concludes: "A large literature supports the developmental position in the practice of special education" (p. 98). By this statement, Blair demonstrates that he either does not understand the origins and controversial history of the distinction along with contradictory evidence or hopes his reader does not. While it is possible that the development-difference notion may have bearing on certain fundamental issues raised by Blair, particularly concerning what might be modifiable and how to do it, any respectable treatment of this convoluted issue deserves a more extended discussion than can be offered here.

Any further discussion not likely to be productive. In his imitable style of careful and detailed analysis of controversial matters, Spitz (1983, 1984) has shown that this is not a productive issue because the developmental-Ziglerian position is so circular as to be "dizzying," with no possibility of proof or disproof because the very fact of mental retardation ensures that retarded and non-retarded persons can never have the same experiences. Furthermore, all mental retardations have a biological basis, rendering the distinction between "organics" and "non-organics" meaningless.

CLOSING ARGUMENT

Blair starts out with the generalization that questions about the nature of intelligence "have potentially strong policy implications" (p. 94), but that connection between policy and science is indirect, uncertain, and subject to "confusion". But rather than precisely explicating how and why he believes this state of affairs has come about, Blair buries whatever signal he may offer under the noise of lubricious verbiage and reactionary obscurantism.

To reject, as we have done, the cabalistic phantasms that early interventionists have foisted on a susceptible society apparently is to be judged as epistemic malfeasance, and undemocratic as well. Starting with a biologically flawed assumption of intrinsic equality, Socialization and Opportunity theories skate on the surface of polite and populist politically correct discourse, because to do otherwise is undiplomatic, and presumably a violation of justice and charity. But this has become a damaging distortion and one hopes, not a vital lie. As technology expands, the gap will only widen. The fact that people are endowed with varying gifts, capabilities, and talents does not at all diminish their humanity. But to engage in the deceit of equalized achievements is demeaning and defeating. Equality of opportunity does not equate with equality of achievement.

Certainly, the many substantive issues that Blair raises in response to us are of great consequence, despite his superficial and distorted treatment of these. Nevertheless, in the final

judgment of soundness and validity of our particular criticisms, they are beside the point. Simply stated, our central contention was that the IHDP failed to meaningfully and lastingly boost intelligence of premature LBW children or to prevent mental retardation--nothing more, nothing less. It is one more proof that not only can we not equalize outcome, we cannot even equalize opportunity. The best we can do is to assure that each individual has the chance to explore a range of opportunities, as she or he intrinsically defines "opportunity."

Setting out to examine the connection between research on intelligence and public policy, Blair hoped to clear up much of the confusion to which we allegedly contributed. On the matter of putting things right, we circumspectly offer for Blair's consideration a Samoan proverb that Mead also would have been well-advised to heed: "O le seuseu ma la fata" or "Fish with a proper net."

NOTES

(1.) We wish to thank Dr. Herman Spitz and Dr. Pamela Woodley for invaluable suggestions, insights, and commentary on an earlier draft.

(2.) In 1983, the American Anthropological Association voted to express its displeasure with the editors of a scientific magazine for recommending Freeman's book as a good holiday gift. The New York Times was officially criticized by another anthropological organization for its role in the publicity campaign for the book. This public relations "spin" by the environmentalists sounds pretty familiar, predictable actually.

(3.) The empirical distribution of IQ is not "normal." There is a much larger proportion in the lower tail of the distribution than would be predicted from the statistical model. The percentage of excess in the IQ range 0-20 is 185,400 percent; in the IQ range 20-50, 125 percent; in the 50-70 IQ range, 1 percent (Dingman & Tarjan, 1960). The bulge at the bottom of The Bell Curve has come to be known as the "bump of pathology."

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