

Why early intervention works A reply to Baumeister and Bacharach

By: Clancy Blair, [Douglas Wahlsten](#)

Blair, C., and Wahlsten, D. (2002) Why early intervention works: A reply to Baumeister and Bachrach. *Intelligence*, 30: 129-140.

Made available courtesy of Elsevier: <http://www.elsevier.com>

*****Reprinted with permission. No further reproduction is authorized without written permission from Elsevier. This version of the document is not the version of record. Figures and/or pictures may be missing from this format of the document.*****

Abstract:

In a wide-ranging critique of compensatory education, Baumeister and Bacharach [Intelligence 23 (1996) 28 (2000)] focus most specifically on the Infant Health and Development Program (IHDP), an intervention program lasting from birth through age 3 for low birth weight (LBW) preterm infants. In response, we identify logical, methodological, and analytical inconsistencies in their critique of early intervention research and offer a balanced assessment of IHDP findings to date. Specifically, we note that Baumeister and Bacharach overinterpret null findings, selectively review the early intervention literature, engage in an inappropriate analytical appeal to variance partitioning, and evidence limited understanding of the ways in which individual differences among program participants and controls may be related to early intervention outcomes. Careful examination of the IHDP study design and database provides a clear indication of what the study accomplished and why. Alternative explanations for the absence of long-term IHDP effects are proposed.

Keywords: Development; Intelligence; Early intervention

Article:

1. Introduction

In a recent issue of this journal, Baumeister and Bacharach continued their spirited defense of the position that early preventive intervention of the type embodied in the Infant Health and Development Program (IHDP), an early compensatory education program from birth through age 3 for low birth weight (LBW), preterm infants, is ineffective and of no value. At the heart of their arguments are findings from IHDP follow-up assessments at child ages 5 and 8 years indicating few long-term program effects on child outcomes, most notably, IQ. Baumeister and Bacharach contend that these findings demonstrate that early compensatory education cannot lead to any enduring and meaningful effects on the development of program recipients. They further contend that negative findings offer support for the idea that the developmental trajectory for intelligence is fixed at birth.

To begin, we all agree that given the strictures of randomized clinical trials, IHDP results at child ages 5 and 8 years, 2- and 5-year intervals following program end, should be seen as largely negative (Brooks-Gunn, McCarton, Casey, et al., 1994; McCarton, Brooks-Gunn, Wallace, et al., 1997). We emphatically disagree with Baumeister and Bacharach, however, on the reasons for the dissipation of treatment effects with age.

2. Problems with logic

Baumeister and Bacharach's critique of the IHDP represents one of those occasions in scientific discourse in which researchers highlight findings from a particular study in order to divert attention away from a much larger body of alternative findings. In their first installment critical of the IHDP study in this journal, Baumeister and Bachrach (1996) asserted that "Once the developmental trajectory is set, it is extremely impervious to significant alteration" (p. 100), and they claimed that effects of the IHDP early intervention were "functionally nonexistent." In their more recent commentary, they go far beyond an argument that the IHDP treatment had no statistically significant effect and claim that the true effect size was precisely zero. Thus, they

seize upon a null hypothesis test as evidence confirming a particular theoretical position. This theoretical position is then utilized to call into question all compensatory education efforts.

Both the logic of data analysis and the weight of empirical evidence, however, are not in their favor. Statistical data analysis cannot prove the truth of any null hypothesis. At best, it delivers a confidence interval for the true size of a treatment effect. In contrast to Baumeister and Bacharach's strong interest in a finding for the null in the IHDP, a large body of evidence points to early environment effects on mental development in the range from 0.25 to 1.0 standard deviation, with intervention over longer periods of time yielding larger effects (Wahlsten, 1997). This body of evidence is consistent with other recent reviews of the literature (Ceci, 1996; Mackintosh, 1998), and it even accords well with twin and adoption studies in behavior genetics that point to substantial sources of environmental variation in intelligence (Plomin, DeFries, McClearn, & Rutter, 1997).

IHDP findings do not prove that the program had no effect nor can they be taken as evidence in favor of the point that all early compensatory education is ineffectual. It may certainly be the case that the IHDP program was simply ineffective but alternative explanations must be examined and considered before such a pronouncement can be made. Baumeister and Bacharach, however, adopt an extreme position by ascribing mental development to only one cause. We suggest that their allegiance to a fixed developmental trajectory leads them to misinterpret an important fact about early experience, namely that control and treatment group differences diminish after the period of intensive intervention ends. They see this as evidence that the effects were not real in the first place. To the contrary, we believe that these findings show there is no early critical period for modification of mental development by experience. By analogy, mental ability can be seen to be like muscle, requiring recurrent exercise to maintain a respectable degree of strength and an extraordinary amount of deliberate practice to achieve expert performance (Ericsson, 1996). As unused muscle quickly becomes flaccid, as witnessed by astronauts who begin their journeys in superb physical condition and return to Earth as weaklings, so do mental abilities such as attention, memory, planning, and problem solving fail to maintain adequate levels of performance if not consistently utilized.

Following the logic of Baumeister and Bacharach, we should not waste public funds on physical education or the teaching of mathematics because their effects dissipate once the instruction ceases. In our opinion, early intervention and related studies demonstrate not only the effectiveness of many kinds of preschool education but also the importance of continuing the enrichment into and beyond the school years. How this enrichment is distributed among the children of the rich and the poor is a question of public policy. Whether enrichment would have a beneficial effect was answered long ago in the affirmative.

The absence of a critical period for setting the trajectory of intelligence poses especially serious difficulties for behavior genetic studies of separated twins. The Minnesota Twin Study, for example, lacked crucial information on twin test scores during the school years and instead examined monozygotic twin pairs more than 10 years on average after they were reunited as young adults (Bouchard, Lykken, McGue, Segal, & Tellegen, 1990). As reported in Table 1 of the widely cited report of the Minnesota Twin Study in Science (Bouchard et al., 1990), it is apparent that the twins had been tested on average 10.6 years after being reunited and were in contact with each other for a substantial amount of time during this interval. The authors of that study dismissed any contribution of this environmental covariance to twin IQ similarity by claiming the "formative period" for mental development had already elapsed. Once the notion of a fixed developmental trajectory is abandoned, however, that twin study is seen to suffer from a major flaw.

Enriched environmental support of the type that Baumeister and Bacharach label as generic has been demonstrated to be associated with optimal child developmental outcomes and sufficient to overcome a myriad of biological and environmental risk factors (Bryant & Maxwell, 1997; Infant Health and Development Program [IHDP], 1990; Ramey & Campbell, 1991 for review). Intervention research has demonstrated that general features of the environment such as enriched language experience, consistent guidance, limit setting, and attention from caring and supportive others, as well as nutritional and hygienic aspects of an ordered and well regulated setting can impact children's intellectual development. The protest is made that there is a

frustrating lack of precision in putative environmental effects on intelligence (Loehlin, 1996; Thompson, 1996). As with Baumeister and Bacharach's (in press) critique of generic intervention, however, these protests represent an ignorance of child study and the great number of correlational studies indicating the types of environmental influences that optimize child developmental outcomes. Early intervention studies provide some limited but convincing experimental support that general features of the environment can influence the ontogenetic course of intelligence.

As with most meaningful areas of research, however, results from preventive intervention studies are not always favorable or uniform. Notable attempts to attack the compensatory intervention endeavor have proceeded logically by attacking perceived methodological difficulties in an attempt to discredit findings before moving on to substance and theory (Spitz, 1992). Baumeister and Bacharach, however, offer selective evidence in their discussion of home visiting and other intervention programs. They fail to consider well-known and widely cited research reports and reviews indicating favorable effects of intervention on mothers and children (Olds, Ekenrode, Henderson, et al., 1997, Olds, Henderson, Kitzman, et al., 1999). They point to instances of negative findings in order to support their position and make selective reference to studies suggesting early intervention to be ineffective for families facing socioeconomic disadvantage (St. Pierre & Layzer, 1999). But they do not consider the full body of evidence.

3. Problems with method

Similarly, as with problems in the logic of their approach to the IHDP and compensatory education, Baumeister and Bacharach's methodological approach to the IHDP is found to be lacking. In their original critique on the program and in their defense of this critique, they make a great deal of the relation between maternal IQ and child IQ. Their indictment of the IHDP is essentially premised on the idea that biological relatedness between mother and child, as reflected in the magnitude of the mother-child IQ correlation, is far more important to intellectual outcome than anything to do with the intervention program. But given the centrality of the maternal IQ variable in their analysis, it is somewhat surprising that they fail to carefully examine its measure, the Peabody Picture Vocabulary Test (PPVT) (Dunn & Dunn, 1981). Given their emphasis on maternal IQ as a relevant individual difference factor in the analysis of IHDP outcomes, one would expect that they would carefully examine the PPVT for all it is worth. However, they blandly state that the "PPVT is demonstrably a good measure of g" and then proceed to treat it as a measure of g rather than the measure of maternal receptive vocabulary that it is. In doing so, however, they ignore the fact that the PPVT is usually obtained in a single testing session lasting no more than 15 min and that as a measure of maternal verbal ability it may represent the effect on child IQ of the language environment that the mother provides as much or more than anything passed from mother to child encoded in DNA. Such a lack of in-depth examination of a variable so central to their analysis that would allow for alternative interpretations of their primary thesis suggests that their interest in the IHDP is ideologically rather than scientifically motivated.

Baumeister and Bacharach's approach to the measure of maternal IQ highlights the extent to which they failed to consider the methodological challenges and intricacies inherent in the attempt to assess genetic and environmental contributions to phenotypic traits in humans. The realities of life in a society that recognizes certain human rights deny behavior genetics the kind of excellent data that can be obtained with controlled experiments on laboratory animals (Wahlsten & Gottlieb, 1997). There is no feasible research design that can unambiguously reveal the precise effect of a multigene difference in human heredity. The same limitation does not apply to research on early experience, however. Random assignment of subjects to control and treatment conditions effectively minimizes any average group difference in genotype and thereby provides a valid indicator of the average treatment effect size. The randomized early intervention design tells nothing about the role of genetic variation and does not separate effects of genetic and environmental variation. Each group mean is an average over many kinds of Genotype x Environment interactions, and variation within a group arises from an unknown combination of effects of genetic and environmental differences as well as developmental variations that are not attributable to either genetic or environmental differences (Gottlieb, Wahlsten, & Lickliter, 1998).

Of course, implementation of a randomized treatment study is never going to be perfect because some parents refuse their group assignments and some families drop out of the study. Fortunately, it is possible to estimate the degree of bias that might arise from these imperfections. Provided that any bias is small in comparison with the magnitude of the observed group difference, the credibility of the primary conclusion about early intervention is not shaken. As Blair (1999) has argued in detail, the IHDP study withstands scrutiny, provided its status as a controlled efficacy trial rather than an effectiveness study is recognized. As Wahlsten (1997, in press) has argued, the results of the IHDP study themselves are consistent with several other converging lines of evidence, including adoption research and studies of the duration of formal schooling. Baumeister and Bacharach accuse Wahlsten of dishonesty in citing the IHDP results, but they evidently missed the point that in his 1997 chapter criticizing *The Bell Curve*, the IHDP values for conditions most similar to three other studies were cited in a figure, i.e., highest birth weight infants with the longest duration of treatment, rather than figures for the entire sample, much of which was not at all comparable with the other studies. The IHDP was mentioned in only one sentence of a much broader review, but apparently Baumeister and Bacharach insist that the study must be discussed at great length if it is to be mentioned at all. Their concerns would be easier to accept if Baumeister and Bacharach applied the same criteria to reviews of twin and adoption studies in behavior genetics, where it is commonplace to cite a single intraclass correlation for monozygotic twins or even a simple heritability estimate without excavating the methodological entrails of each study.

4. Problems with analysis

As with problems with logic and method, Baumeister and Bacharach continue to advocate for an analytical approach to IHDP data that is inadequate. In both their original publication and its defense, they take a “largest correlation wins” approach to the use of statistical analysis to support their position. They make a great deal of the size of the correlation between maternal PPVT and child IQ relative to the size of the correlation between the group assignment variable and child IQ. Such allegiance to the numbers, however, reflects an ongoing problem with the use of statistical analysis in behavioral research (Cohen, 1990; Schmidt, 1992). The point is that an individual difference factor, maternal PPVT, is going to account for a larger proportion of variance in another individual difference factor, child IQ. The group assignment term will account for a smaller amount of variation around the mean. It will, however, be associated with upward shifts of the mean that affect the percentage of individuals falling into the lower IQ tail of the distribution. The relevant statistic for assessing the program effect is the effect size or the odds ratio.

Two examples help to illustrate this point. The first is highly relevant to Baumeister and Bacharach’s attack on the IHDP and concerns findings from the NICHD Early Child Care Network study of child care experience beginning in infancy. The Early Child Care Network study has examined child cognitive outcomes as a function of child and family background characteristics and child day care experience within a longitudinal design. Findings indicate that child and family characteristics overwhelmingly account for the majority of variance in outcomes. Quality of care, defined primarily by language stimulation, and other care predictors accounted for relatively small proportions of variance, between 1.3% and 3.6% in child cognitive measures. However, examination of mean differences between children in high versus low quality care adjusted for child and family characteristics revealed effect sizes associated with quality of care of greater than .40 for child cognitive and language outcomes at 24 and 36 months (NICHD Early Child Care Research Network, 2000). Though the quality of care accounts for relatively little variation around the mean levels of cognitive and language functioning in the sample, the quality of care is associated with a moderate to large effect on child outcomes.

A second example, though one with somewhat different implications for intelligence and early intervention, concerns the Flynn effect (Flynn, 1984), the name given to the upward drift in mean IQ observed at each renorming of standardized measures of intelligence. The upward drift of approximately 3 IQ points per decade does not account for substantial variance in any analysis of IQ between cohorts. The small size of the change per decade relative to the variation present in IQ in any given cohort places a ceiling on the amount of variance that will be associated with it (“The greater the departure from distribution similarity [between predictor and outcome], the more severe will be the restriction on the maximum possible r ,” Cohen & Cohen, 1983, p. 65). However, the upward drift is a major scientific finding with strong implications for definitions and

measurements of intelligence (Flynn, 1999). It suggests that measures of intelligence are highly influenced by environmental factors, because the average population increase of 3 IQ points per decade has been too rapid to have resulted from genetic selection. In fact, the environmentally driven upward drift over the past 70 years is so great as to call into question what it is that IQ tests actually measure and raises the idea that it may be possible to raise IQ without raising intelligence (Flynn, 1996). While the implications of the upward drift are certainly wide-ranging, the important point of both the Flynn effect and the Early Child Care Network Study for present purposes is their implication for Baumeister and Bacharach's argument regarding the "overwhelming" effect for maternal PPVT in the IHDP data. They demonstrate that Baumeister and Bacharach's appeal to variance partitioning in an attempt to belittle the intervention effect simply does not stand up, not on methodological grounds due to failure to comprehensively examine the measure of their key independent variable nor in analysis due to their appeal to variance partitioning.

In addition to the aforementioned problems, Baumeister and Bacharach's approach to the analysis of IHDP data also fails to consider a key point related to the study's design. Because random assignment is done prior to administration of treatments in the IHDP, group membership warrants priority in regression analysis and should be entered into the equation first. One can then ask whether and to what extent additional information provided by, for example, maternal education or receptive vocabulary improves our understanding of childhood test scores, and centered variables can be used to examine interactions (Aiken & West, 1991). We do not agree with the statistical methods invoked by Baumeister and Bacharach (1996) on pages 86 and 87 where they simply pool children in the two groups and then ask which variable has the highest correlation with child IQ, as might be appropriate for a single sample obtained from a population survey.

To illustrate this point, an independent analysis of IHDP data utilizing the primary analysis group data set used in reporting prior IHDP findings (e.g., Ramey et al., 1992) was done by one of us (DW). First, all variables were subjected to analysis of variance with three factors: treatment (control, intervention), site number (1 to 8), and skin color (Black, Hispanic, White). For variables that existed prior to the study, including maternal age, education, and PPVT, as well as birth weight and HOME score, there were no significant differences between treatment groups (all $P > .05$), as expected with random assignment and minor attrition, but there were large variations among sites and skin colors. Multiple regression analysis yielded an equation (Table 1) that accounted for about 44% of variance in Stanford–Binet IQ at 36 months. Most importantly, the first three predictors all had tolerances of at least 0.98 and therefore were essentially independent of each other as well as the remaining predictors, whereas site, skin color, maternal PPVT, and HOME score were so highly correlated with each other that partitioning variance among them was not possible. Site, skin color, maternal PPVT, and HOME score being independent of treatment group, help to understand only the variance within a treatment group. These variables involved some unknown combination of hereditary and environmental factors; hence, this aid to understanding is statistical rather than biological or psychological. The effect of early intervention was a substantial increase in IQ of about 9 points, and this psychologically meaningful effect was without question an environmental effect. Further analyses can be done to study interactions and identify subgroups showing greater or lesser effects of the treatment, but none of these challenge the reality of the treatment effect.

Table 1
Multiple regression equation predicting Stanford–Binet IQ at 36 months

Predictor	Coefficient	Standardized	Tolerance	<i>t</i>	<i>P</i>
Treatment	9.15	0.227	0.990	8.47	.0001
Sex	3.65	0.092	0.986	3.45	.002
Birth weight	0.004	0.083	0.980	3.09	.001
Maternal IQ	0.217	0.229	0.548	6.37	.0001
HOME score	0.792	0.247	0.688	7.70	.001
Skin color	5.85	0.144	0.514	3.88	.001
Sites	0 to -11	0 to -0.19	.53 to .62	0 to -5	.0001

Note: Treatment, sex, and skin color were coded as (-.5, .5), whereas sites were dummy coded in seven variables and entered as one group. There were 782 degrees of freedom for the residual variance and the standard error estimate was 14.8.

5. A balanced approach to compensatory education research

Issues and evidence of the sort raised in this paper do not accord well with Baumeister and Bacharach's arguments. They ascribe naïve cultural-relativist beliefs to early intervention proponents while scrambling for the high ground of gene-environment interaction, paying lip service to the need for environmental factors in development. But their understanding of Gene \times Environment interaction is limited by their viewpoint. As with others before them, most notably Herrnstein and Murray (1994) and Jensen (1997), their approach to environmental effects is one in which environmental factors can only additively effect phenotypic traits and then only in the face of large genetic effects. No rigorous attempt is made to consider a state of nature in which the environment plays an active role in gene expression (Gottlieb, 1998). And no rigorous attempt is made to question what intelligence tests measure and why they predict later life outcomes.

It is our position that no purpose is served by Baumeister and Bacharach's polemical portrayal of compensatory education as a humanistic endeavor. No one actually holds the views that Baumeister and Bacharach ascribe to intervention researchers. Reasonable people understand that both genes and environments jointly determine the expression of any phenotypic trait. As well, reasonable people understand that intelligence tests measure a mix of ability and opportunity. While arguing that early experience can markedly alter intelligence, we do not claim that studies of environment belittle or negate the importance of genetic variation. But it is clear that Baumeister and Bacharach do not share these views when they make the claim that "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." (pp. 171)

There can be no argument regarding the importance of individual differences among children in response to compensatory education. Attention to individual differences is a priority in preventive intervention research (Ramey & Ramey, 1992). Baumeister and Bacharach, however, take the position that intelligence is an innately determined individual difference characteristic that assures differences in the ability of individual children to benefit from intervention. This is a radically different position from that motivating compensatory education. The interventionist's position is developmental. Intelligence is understood to be a developmental construct amenable to combined environmental and genetic influence. While there are differences in children's responsiveness to intervention efforts that are genetically and environmentally determined, enriched environments can benefit all recipients to some extent because of the role of experience in the development of intelligence. The innatist, however, evidences no conception of how a trait can have a genetic component and still be amenable to environmental influence. Unidirectional influence from gene to behavior is assumed. Estimations of the influence of genes on intelligence supported by variance partitioning behavior genetic studies, however, are based on incorrect unidirectional assertions regarding how genes work. The all-important question concerns how genes and environment work together to influence phenotypic traits as gene functions are altered by environmental influences (e.g., Crabbe, Wahlsten, & Dudek, 1999; Gottlieb, 1998; Kandel, 1998).

Within a theoretical framework in which both biology and environment are accorded equal weight, reasonable alternative explanations for IHDP follow-up findings can be considered. The fact that the sample contains children at varying level of risk due to LBW as well as varying level of risk due to socioeconomic factors makes it a useful one for studying the joint contribution of biology and environment to developmental outcome. Baumeister and Bacharach criticize the study for a lack of specificity in risk associated with LBW but provide no evidence to suggest that such information is important for the IHDP's primary outcomes. They simply assume that the study can provide no meaningful information regarding the effect of environmental support on children born LBW. They then further conclude that efforts to prevent LBW are the only viable strategy for preventing the sequelae of LBW. In this, their innatist bent regarding the immutability of intelligence extends to their consideration of the biological risk facing children born LBW. But again the weight of evidence regarding early intervention for children born LBW does not support their arguments (see Aylward, Pfeiffer, Wright, & Verhulst, 1989; Blair & Ramey, 1997 for reviews). Environmental factors play a substantial role in outcome

among children born LBW (e.g., Bendersky & Lewis, 1994; Resnick, Eyler, Nelson, Eitzman, & Bucciarelli, 1987).

As Baumeister and Bacharach contend, the absence of long-term group differences in intelligence and in indices of school adjustment among IHDP participants may simply reflect the long-term ineffectiveness of the program. As noted above, however, the absence of long-term effects may be explainable by the fact that the intervention only continued through age 3. Unlike its predecessor, the Abecedarian Project (Ramey & Campbell, 1991), the IHDP contained no extended intervention component. It may be that restricting the intervention to the child's first 3 years limited the effectiveness of the intervention program and its potential for impacting school outcomes. The Abecedarian intervention, unlike the IHDP, was delivered for the child's first 5 years and up to age 8 for some program recipients. It may be that continued intervention beyond the age of 3 is necessary for the maintenance of early gains.

An additional consideration for the absence of long-term program effects in the IHDP, however, may be related to the fact that the IHDP sample was comprised of LBW, preterm infants. LBW, preterm infants are at particularly high risk for negative emotionality and poor self-regulation due to physiological immaturity at birth. It is thought that high rates of cognitive and social developmental difficulty among LBW children are mediated through nervous system deficits that limit the ability to regulate state (Porges, 1996). Recent analyses of IHDP data provide some evidence suggesting that self-regulation difficulty is central to the intervention's effect. The intervention appears to have worked particularly well for children at high risk for developmental delay due to problems associated with self-regulation difficulty in infancy. Specifically, at program end the intervention was found to be associated with a fourfold decrease in clinically meaningful behavior problems and a fivefold decrease in $IQ \leq 75$ among children characterized by high levels of temperamental self-regulation difficulty in infancy (Blair, in press). These findings suggest that the intervention worked, in part, by offsetting problems associated with self-regulation difficulty that interfere with developmental progress.

Given evidence suggesting the relevance of self-regulation ability to intervention outcome at age 3, it is conceivable that in the absence of intervention services following age 3, self-regulation difficulties among intervention recipients may be confounding expected long term intervention effects. While difficulty with self-regulation is only one aspect of child functioning that may be worth considering in the assessment of long-term outcomes in the IHDP, it is one that provides a plausible, testable developmental explanation for the lack of long-term effects. It is also one that is supported by available research and theory. Though Baumeister and Bacharach may be content to condemn the compensatory education enterprise on the basis of IHDP null findings, in science, it is the posing and testing of alternative explanations for unexpected findings, rather than reliance upon negative results to bolster a particular ideological position, that leads to a healthy and purposeful community of scholarly inquiry.

In conclusion, scientific evidence available at the turn of the 21st century indicates that early intervention can alter the developmental trajectories of children at risk for later cognitive and social developmental problems. As an aspect of the overall finding for the efficacy of early intervention, however, scholars should welcome the long-term IHDP findings. Rather than trying to downplay them or to make more of them than they deserve, long-term results in the IHDP should be seen for what they are: unexpected findings that invite further inquiry. This is what contradictory findings should do. The problem is, however, when individuals view isolated findings as single definitive answers to complex and enduring questions. Rather than viewing IHDP findings as opportunity for further inquiry and open debate on what does and does not work in compensatory education, they too eagerly highlight them in a rush to condemn the whole enterprise. Which is not to say that the early intervention literature generally and the IHDP specifically are without their problems. In particular, there has been an inadequate specification of the mechanisms through which putative early intervention effects occur. A black box model of intervention has prevailed in which treatments are presumed to be monolithic and a clinical trials approach to design and analysis has been accepted as the methodological gold standard. Perhaps by returning null findings in long-term follow-ups, the IHDP can spur advances in compensatory education research that will further understanding of how and why early intervention is effective.

References

- Aiken, L. S., & West, S. G. (1991). *Multiple regression: testing and interpreting interactions*. Newbury Park, CA: Sage.
- Aylward, G. P., Pfeiffer, S., Wright, A., & Verhulst, S. (1989). Outcome studies of low birth weight infants published in the last decade: a meta-analysis. *Journal of Pediatrics*, 115, 515–520.
- Baumeister, A. A., & Bacharach, V. R. (1996). A critical analysis of the Infant Health and Development Program. *Intelligence*, 23, 79–104.
- Baumeister, A. A., & Bacharach, V. R. (2000). Early generic intervention has no enduring effect on intelligence and does not prevent mental retardation: the Infant Health and Development Program. *Intelligence*, 28, 161–192.
- Bendersky, M., & Lewis, M. (1994). Environmental risk, biological risk, and developmental outcome. *Developmental Psychology*, 30, 484–494.
- Blair, C. (1999). Science, policy, and early intervention. *Intelligence*, 27, 93–110.
- Blair, C. (in press). Early intervention for low birth weight, preterm infants: the role of negative emotionality in the specification of effects. *Development and Psychopathology*.
- Blair, C., & Ramey, C. T. (1997). Early intervention for low birth weight infants and the path to second generation research. In: M. Guralnick (Ed.), *The effectiveness of early intervention* (pp. 77–97). Baltimore: Brookes.
- Bouchard, T. J., Lykken, D. T., McGue, M., Segal, N. L., & Tellegen, A. (1990). Sources of human psychological differences: the Minnesota study of twins reared apart. *Science*, 250, 223–228.
- Brooks-Gunn, J., McCarton, C., Casey, P., McCormick, M., Bauer, C., Bernbaum, J., Tyson, J., Swanson, M., Bennett, F., Scott, D., Tonascia, J., & Mainert, C. (1994). Early intervention for low birth weight premature infants: results through age 5 years from the Infant Health and Development Program. *Journal of the American Medical Association*, 272, 1257–1262.
- Bryant, D., & Maxwell, K. (1997). The effectiveness of early intervention for disadvantaged children. In: M. Guralnick (Ed.), *The effectiveness of early intervention* (pp. 23–46). Baltimore, MD: Brookes.
- Ceci, S. J. (1996). *On intelligence. A bioecological treatise on intellectual development* (Expanded edition). Cambridge, MA: Harvard University Press.
- Cohen, J. (1990). Things I have learned (so far). *American Psychologist*, 45, 1304–1312.
- Cohen, J., & Cohen, P. (1983). *Applied multiple regression/correlation analysis for the behavioral sciences* (2nd ed.). Hillsdale, NJ: Erlbaum.
- Crabbe, J. C., Wahlsten, D., & Dudek, B. C. (1999). Genetics of mouse behavior: interactions with laboratory environment. *Science*, 284, 1670–1672.
- Dunn, L. W., & Dunn, L. M. (1981). *Peabody Picture Vocabulary Test—Revised*. Circle Pines, MN: American Guidance Service.
- Ericsson, K. A. (1996). The road to excellence. The acquisition of expert performance in the arts and sciences, sports and games. Mahwah, NJ: Erlbaum.
- Flynn, J. (1984). The mean IQ of Americans: massive IQ gains from 1932 to 1978. *Psychological Bulletin*, 95, 29–51.
- Flynn, J. (1996). What environmental factors affect intelligence: the relevance of IQ gains over time. In: D. Detterman (Ed.), *Current topics in human intelligence. Vol. 5. The environment* (pp. 17–30). Norwood, NJ: Ablex.
- Flynn, J. (1999). Searching for justice: the discovery of IQ gains over time. *American Psychologist*, 54, 5–20.
- Gottlieb, G. (1998). Normally occurring environmental and behavioral influences on gene activity: from central dogma to probabilistic epigenesis. *Psychological Review*, 105, 792–802.
- Gottlieb, G., Wahlsten, D., & Lickliter, R. (1998). The significance of biology for human development: a developmental psychobiological systems view. In: R. Lerner (Ed.), *Handbook of child psychology: Vol. 1. Theory* (pp. 233–273). New York: Wiley.
- Herrnstein, R. J., & Murray, C. (1994). *The bell curve: intelligence and class structure in American life*. New York: Free Press.

- Infant Health and Development Program. (1990). Enhancing the outcomes of low-birth-weight, premature infants. *Journal of the American Medical Association*, 263, 3035–3042.
- Jensen, A. R. (1997). The puzzle of nongenetic variance. In: R. J. Sternberg, & E. L. Grigorenko (Eds.), *Intelligence, heredity, and environment* (pp. 42–88). New York: Cambridge University Press.
- Kandel, E. (1998). A new intellectual framework for psychiatry. *American Journal of Psychiatry*, 155, 457–469.
- Loehlin, J. (1996). Environment and intelligence: a comment. In: D. Detterman (Ed.), *Current topics in human intelligence. Volume 5: the environment* (pp. 151–157). Norwood, NJ: Ablex.
- Mackintosh, N. J. (1998). *IQ and human intelligence*. NY: Oxford University Press.
- McCarton, C., Brooks-Gunn, J., Wallace, I., et al (1997). Results at age 8 years of early intervention for low birth weight premature infants: the Infant Health and Development Program. *Journal of the American Medical Association*, 277, 126–232.
- NICHD Early Child Care Research Network. (2000). The relation of childcare to cognitive and language development. *Child Development*, 71, 960–980.
- Olds, D., Ekenrode, J., Henderson, C., Kitzman, H., Powers, J., Cole, R., Sidora, K., Morris, P., Pettit, L., & Luckey, D. (1997). Long-term effects of home visitation on maternal life course and child abuse. *Journal of the American Medical Association*, 278, 637–643.
- Olds, D., Henderson, C., Kitzman, H., et al (1999). Prenatal and infancy home visitation by nurses: recent findings. *The Future of Children*, 9, 44–65.
- Plomin, R., DeFries, J. C., McClearn, G. E., & Rutter, M. (1997). *Behavioral genetics* (3rd ed.). NY: Freeman.
- Porges, S. W. (1996). Physiological regulation in high-risk infants: a model for assessment and potential intervention. *Development and Psychopathology*, 8, 43–58.
- Ramey, C. T., Bryant, D. M., Wasik, B. H., Sparling, J. J., Fendt, K. H., & LaVange, L. M. (1992). Infant Health and Development Program for low birth weight premature infants: program elements, family participation, and child intelligence. *Pediatrics*, 89, 454–465.
- Ramey, C. T., & Campbell, F. A. (1991). Poverty, early childhood education, and academic competence: the Abecedarian experiment. In: A. Huston (Ed.), *Children in poverty* (pp. 190–221). New York: Cambridge University Press.
- Ramey, C. T., & Ramey, S. L. (1992). Early educational intervention with disadvantaged children—to what effect? *Applied and Preventive Psychology*, 1, 131–140.
- Resnick, M. B., Eyler, F. D., Nelson, R. M., Eitzman, D. V., & Bucciarelli, R. L. (1987). Developmental intervention for low birth weight infants: improved early developmental outcome. *Pediatrics*, 80, 68–74.
- Schmidt, F. (1992). What do data really mean? Research findings, meta-analysis, and cumulative knowledge in psychology. *American Psychologist*, 47, 1173–1181.
- Spitz, H. (1992). Does the Carolina Abecedarian early intervention project prevent sociocultural mental retardation? *Intelligence*, 16, 225–237.
- St. Pierre, R. G., & Layzer, J. I. (1999). Using home visiting for multiple purposes: the Comprehensive Child Development Program. *The Future of Children*, 9, 134–151.
- Thompson, L. (1996). Where are the environmental influences on IQ? In: D. Detterman (Ed.), *Current topics in human intelligence: Vol. 5. The environment* (pp. 179–184). Norwood, NJ: Ablex.
- Wahlsten, D. (1997). The malleability of intelligence is not constrained by heritability. In: B. Devlin, S. Feinberg, D. Resnick, & K. Roeder (Eds.), *Intelligence, genes and success* (pp. 71–87). New York: Copernicus (Springer-Verlag).
- Wahlsten, D. (in press). The theory of biological intelligence: history and a critical appraisal. In: R. Sternberg & E. Grigorenko (Eds.), *The general factor of intelligence: how general is it?* Mahwah, NJ: Erlbaum.
- Wahlsten, D., & Gottlieb, G. (1997). The invalid separation of effects of nature and nurture: lessons from animal experimentation. In: R. Sternberg, & E. Grigorenko (Eds.), *Intelligence, heredity and environment* (pp. 163–192). Cambridge, MA: Cambridge University Press.