In press, *Contemporary Psychology*.

"Environments" are genetic too

Environmental Effects on Cognitive Abilities

by Robert J. Sternberg and Elena L. Grigorenko (Eds.)

Mahwah, NJ: Erlbaum, 2001. 470 pp. ISBN 0-8058-3183-5. $99.95

Review by Linda S. Gottfredson

Sternberg and Grigorenko (2001, p. viii) offer *Environmental Effects on Cognitive Abilities* as "a handbook for those interested in the entire range of environmental effects" on cognitive development because it aims to "integrate what formerly have been very diverse literatures." This edited volume does, in fact, take psychologists a step forward in understanding non-genetic influences when five of its 14 chapters review evidence on various biological insults that depress cognitive development in childhood: malnutrition, environmental pollutants, prenatal exposure to drugs and alcohol, infectious diseases, and radiation.

But the book simultaneously encourages a step backward when it seems to repudiate an essential requirement for inferring environmental effects: research on environments must control for genetic influences. The editors first fail to include behavior genetic evidence in the book and then disparage that field's methods. The book therefore leaves to readers the unspoken job of culling the book's good evidence from its genetically-confounded results. There is enough of the former, however, to reward the effort.

The Step Backward

Environmental Effects never hints that behavior genetic techniques are among the most powerful for isolating and quantifying *environmental* influences. Ironically, the editors never

alert readers to the behavior genetic evidence on environments that they had showcased in their own 1997 edited volume, *Intelligence, Heredity, and Environment*, despite describing their new book as "supplement[ing] the chapters in that [earlier edited] book with respect to the specific nature and range of environmental effects" (p. vii). Chapters in the 1997 book had been valuable for illustrating how behavior genetic methods can be used to decipher the partnership between nature and nurture, for example, in the development of language (Pipp-Siegel, Robinson, Bridges, & Bartholomew) and infant social cognition (Reznick), and in creating both stability and change in cognitive development during childhood (Cherny, Fulker, & Hewitt). Another chapter (Jensen) had made progress in parsing behavior genetics' startling discovery that environments operate mostly to make family members *less* alike (so-called *nonshared* effects) rather than more alike (*shared* effects).

In contrast, the chapters in *Environmental Effects* tend to mention genetic influences only in passing (e.g., Fiese, p. 39), as non-existent (Ogbu & Stern, p. 6), or as "clearly" irrelevant to the issue at hand (secular rise in IQ; Fernandez-Ballesteros, Juan-Espinosa, & Abad, p. 388). Or they dismiss the nature-nurture debate as an "entertaining distraction" (Seifer, p. 78) and "psychology's bad penny" with regard to race (Ramey, Ramey, & Lanzi, p. 85). (The Schaie and Zuo chapter may be an exception in attending to genetic confounding, but it is too sketchy to follow.)

Next, Sternberg's Epilogue implicitly justifies the book's excluding behavior genetic research by explicitly disparaging it. His criticisms apply, however, to all methods of ferreting out environmental effects on cognitive abilities and, I suspect, to virtually all psychological research: for example, its techniques for estimating effects (i.e., heritability) have limits, and IQ measures have no zero point. Sternberg belittles a focus on individual differences themselves

(the raw material for behavior genetic analyses) as a "preoccupation" that may be due in large part to "an historical accident" (p. 427), even though many authors in *Environmental Effects* share this analytic focus. Ignoring genetic confounds in environmental research is self-defeating, however, because, as the lead chapter (Scarr) in the predecessor book had argued so forcefully, the results of such studies are causally uninterpretable.

Experiences Are Genetic Too

Why uninterpretable? Partly because both environments and genes affect cognitive development, as Sternberg and Grigorenko's preface points out, but also because "environments" themselves tend to be moderately heritable. People's genetic proclivities often affect their *exposure* to different physical and social environments, in particular, the experiences they seek out and construct (Scarr, 1996). Summarizing research on the heritability of environments, one textbook (Plomin, DeFries, McClearn, & Rutter, 1997, pp. 203-204) reports that "genetic research consistently shows that family environment, peer groups, social support, and life events often show as much genetic influence as do measures of personality," which is about 50%, on the average.

The environmental factors that the chapters in *Environmental Effects* invoke most frequently are parental rearing practices because, as several note, large differences in mental ability appear by age 2 and stabilize soon thereafter. Differences in parenting behavior, however, are partly a *response* to genetic differences among the children being reared (temperament, interests, abilities, and so on). Analyses of standard measures of early family environments, such as the Family Environment Scale (FES) and Home Observation for the Measurement of Environment (HOME), show that differences in children's rearing conditions (parental warmth, toys provided, and so on) are about 40% heritable. Consider, for example, that identical twins

reared *apart* rate their childhood environments as more similar than do fraternal twins reared *together*, and research that actually observes parent-child interactions provides similar evidence of genetic effects on family environment (Scarr, 1996, p. 219). Moreover, about half the phenotypic correlation between the HOME and children's IQ is genetic, that is, substantial variation in both HOME environments and IQ can be traced to the same genes (Plomin et al., 1997, p. 261). Only genetically-sensitive analyses of family data can isolate the non-genetic environmental effects in this tangled skein.

Behavior genetic research also reveals that social scientists have been looking mostly in the wrong places for enduring non-genetic effects. "Socialization theories" like those on display in the psychosocial-cultural chapters of *Environmental Effects* have wrongly presumed that environmental effects make family members more alike and thus magnify differences between families. The truth is actually quite the opposite. Behavior geneticists have now concluded, to their own surprise, that environmental effects "are relatively specific to each child rather than general for all children in a family" (Plomin, DeFries, McClearn, & McGuffin, 2001, p. 305). Moreover, the impact on intelligence of shared factors such as parents' education and income virtually disappears by adolescence while the heritability of intelligence rises to 60% by adolescence and 80% by late adulthood in typical Western populations. The story is similar for more specific cognitive skills and achievements (Plomin et al., 2001, pp. 190, 199-201, 298).

The biological insults discussed in *Environmental Effects* would seem to have primarily nonshared effects, judging from their specificity of effect depending on the affected individual's developmental age, current health, intensity of exposure, and perhaps genetic susceptibility. In fact, three of the chapters report studies using *siblings* as the control group. Jensen and Scarr had

speculated in the editors' 1997 book that most environmental effects on intelligence are, in fact, nonshared biological factors.

Biological Tortoises vs. Sociocultural Hares

The most informative chapters are therefore the five that review evidence on five classes of biological insults (Grantham-McGregor, Ani, & Fernald; Bellinger & Adams; Mayes & Fahy; Alcock & Bundy; Grigorenko). Although their authors do not discuss genetics as such, they are careful to consider confounding factors and to stress the importance of experimental or quasi-experimental data and the risks of inferring causal effects from non-experimental data. They also help provide an interpretive context for the environmental effects by giving prevalence or incidence rates for the various insults, distinguishing the relative magnitude of their effects, and considering the specific physiological and social mechanisms by which the insults probably cause their damage.

Compared to the biological chapters, the seven chapters on family, school, and social class or caste environments less often use experimental data, pay attention to potential confounds, note the often large differences in IQ and outcomes among siblings in the same family (e.g., Murray, 1997), or say anything about how common or how powerful the various putative social ailments and advantages are relative to each other or genetic effects. The psychosocial-cultural chapters nonetheless tend to draw strong conclusions about environmental effects, even when they report far more speculation than empirical data (e.g., Ogbu & Stern) or their data are completely confounded with genetic influences (e.g., Fiese; Seifer). The book's firmest evidence on psychosocial influences comes from experimental studies on preschool interventions (Ramey et al.) and quasi-experimental studies that compare the cognitive skills of

kindergartners and first-graders who just met or missed the age thresholds for entering those grades (Christian, Bachnan, & Morrison).

The Epilogue (p. 425) opens with Sternberg's conclusion that "Large numbers of variables have been shown to make a difference. The list seems almost endless and the case for their power irrefutable." The list is the unintegrated succession of results in the first 12 chapters, because the book lacks the incisive overview (Hunt) and "next steps" chapters (Waldman) that the 1997 volume had. The impression of irrefutable power is created mostly by the catastrophic effects (e.g., profound retardation and limb deformity) of some toxins and by the book's thirteenth chapter, which details the large secular increases in IQ scores (Fernandez-Ballesteros et al.). The placement of this otherwise informative chapter in the book's "Conclusion" section (Sternberg's Epilogue being the other entry), together with its authors' presumption that the secular increases must necessarily be entirely non-genetic, entices readers to leap to the false conclusion that these trends constitute self-evident proof that environmental effects are huge and genetic differences therefore pose few constraints on social engineers. In truth, the yet-unexplained secular increases pose as much of a challenge for nurture enthusiasts as they do for nature enthusiasts (Dickens & Flynn, 2001).

Moving Forward

Although the Epilogue's answer to such seeming paradoxes is to raise doubts about the merits of behavior genetic research, scrutiny of the book's good evidence—which concerns children only—reveals how the different bodies of data actually form a consistent pattern.

The biological insults seem to fall into seven categories in terms of magnitude and permanence of their effects: (1) sometimes catastrophic (e.g., cretinism, microcephaly, death) and not reversible—iodine deficiency, lead poisoning, AIDS, and prenatal exposure to high

levels of nuclear radiation, alcohol, and methyl mercury; (2) small to moderate irreversible effects—high levels of postnatal exposure to radiation (nuclear accidents, radiation therapy for leukemia) and infections of the central nervous system (encephalitis), although sometimes only or mostly when accompanied by complications such as seizures (respectively, bacterial meningitis, cerebral malaria); (3) small to moderate effects that are mostly reversible—parasitic infections (e.g., hookworm), short- or long-term severe protein-energy malnutrition (PEM), persistent ear infections, low-level prenatal exposure to PCBs, high postnatal exposure to methyl mercury, and iron deficiency; (4) mild and transient effects—colds, flu, short-term hunger; (5) no discernible effects—asymptomatic HIV, viral meningitis, febrile convulsions, and low levels of exposure to radiation or postnatally to PCBs; (6) unclear because effects are confounded with either genetic risk or exposure to other environmental toxins—prenatal exposure to opiates, cocaine, and high levels of PCBs; and (7) insufficient data—low levels of methyl mercury, zinc deficiency, pesticides, and prenatal exposure to marijuana.

All factors in the first set appear to act directly on the central nervous system (CNS) and to alter the architecture of the brain, in most cases during sensitive prenatal stages of brain development. In some cases (e.g., lead), the severity of effects is known to be dose related. The second set of usually milder but still irreversible effects also represent direct assaults to the CNS, but postnatally. Their major effects are often on attention and arousal or working memory.

Whereas the mostly man-made insults in the first two sets are probably more common in industrialized countries, the third set consists mostly of infections and forms of malnutrition that have long bedeviled humankind and which remain prevalent in poor countries. Their mode of action is less clear, but often involves a general stunting of physical growth. They also often have the effect of depressing energy and activity, leading affected children to be non-responsive and

thereby "functionally isolate" themselves from social stimulation and assistance. When the infections are treated and good nutrition provided, considerable catch-up growth occurs and normal behavior resumes. We might presume that humans have evolved physiological adaptations to these common biological assaults but not to the more man-made ones.

The particular cognitive processes affected and the degree to which they are affected often vary widely *within* each of the foregoing three sets biological insults, depending on the specific biological factor involved (species of pathogen) and the state of the affected individual (e.g., developmental age, nutrition, other infections and exposures), medical complications (convulsions), and sequelae that might affect caregiving (lassitude, irritability). As for the mild, transient, and ubiquitous maladies, they seem to more often afflict and more strongly affect individuals who are already malnourished or otherwise vulnerable. Their chief effect is apparently to impair attention or motor competence.

The book's discussions of biological insults say almost nothing about academic performance, but we might presume that they affect school performance indirectly by depressing cognitive abilities, reducing attention and arousal, or producing hyperactivity and conduct problems. In fact, the latter two effects—inducing too little or too much activity—appear to be the more commonly demonstrated ones.

Turning to the psychosocial chapters, they reveal nothing about parental effects, because all their data are confounded with genetic influences. The experimental/quasi-experimental data in two chapters (Christian et al.; Ramey et al.) do suggest, however, that *amount* of instruction raises mean levels of performance on a variety of ability and achievement tests. Effects are largest among children with the lowest average mental ages (lower IQ, lower social class, chronologically younger), but none may be permanent. Longitudinal data for genetically and

socially at-risk children show that the IQ gains relative to peers tend to disappear with advancing age and grade level. In this respect, they are probably most similar to the third set of biological effects, which are small to moderate and mostly reversible.

The schooling chapter reports no follow-up data for the increases in narrower skills and abilities from additional schooling it documented, but we might suspect that these increases are more sustainable. The reason is that other research shows them to be somewhat less heritable, more trainable, and more subject to shared effects. Narrower, more trainable skills tend to be less generalizable, however, and lack of generalizable effects is just what Christian et al. found: "The nature, timing, and magnitude of schooling effects are surprisingly domain specific and appear to depend, in part, on instructional emphases operating in the classroom" (p. 326). (Some of the specificity may be artifactual, owing to probable differences in tests' *g* loadings as well as sampling and measurement error.)

The environmental effects reported in *Environmental Effects* do not conflict with the behavior genetic evidence, even if we assume that they are substantially shared family effects, because genetic research shows sizeable shared as well as nonshared environmental effects on childhood IQ. The real puzzle is why, in longitudinal studies thus far, shared effects disappear with age, completely for IQ and mostly for specific abilities and academic achievement. Are large, enduring effects on cognitive performance rare and the smaller, more common ones usually reversed? Might recovery—or enhancement of abilities—be correlated with genotype, and hence differ among family members?

References

Dickens, W. T., & Flynn, J. R. (2001). Heritability estimates versus large environmental effects: The IQ paradox resolved. *Psychological Review, 108*(2), 346-369.

Murray, C. (1997). IQ and economic success. *The Public Interest*, No. 128, 21-35.

Plomin, R., DeFries, J. C., McClearn, G. E., & McGuffin, P. (2001). *Behavior genetics (4th ed.).* New York: Worth.

Plomin, R., DeFries, J. C., McClearn, G. E., & Rutter, M. (1997). *Behavior genetics (3rd ed.).* New York: Freeman.

Scarr, S. (1996). How people make their own environments: Implications for parents and policy makers. *Psychology, Public Policy, and Law, 2*(2), 204-228.

Sternberg, R. J., & Grigorenko, E. L. (Eds.) (1997). *Intelligence, heredity, and environment.* New York: Cambridge University Press.