ment. This sets for the authors the task of partitioning the environment into "pure" environment and "genetic" environment. The people contributing to the "genetic" environment are also presumably contaminated by "environmental" contributions to *their* genetics, and so on for endless partitioning. (One might also inquire about the variance in the researchers who measure the environment, for even the so-called "completely objective measures such as videotape" are interpreted by people influenced by their genetics and environment.)

This predictment is similar to the one raised by Plomin and Daniels in their 1987 *BBS* paper so intriguingly titled, "Why Are Children in the Same Family so Different From One Another?" Under the assumptions of behavioral genetics, children in the same families, who share both genes and environment, ought to be more alike than they really are. Plomin and Daniels explained this inconsistency by the construction of *nonshared environment*, which consists of the systematic, but as yet elusive, environmental differences that affect even children in the same family and engender their unique constitutions. But, we ask, how can anything so hard to measure exert such a whopping influence?

The simple and seductive assumptions of behavioral genetics require these improvisations because they do not do justice to the biological realities of the developmental process, which is complex, nonlinear, and above all, constructive. As many have noted (e.g., Oyama 1985), the genes do not code behavior, but exert their influence through a long, contingent process. During development there are many opportunities for small random variations in organic and environmental conditions to be amplified and to cascade into individual differences that cannot be partitioned into two exclusives sources (Thelen 1989; 1990). These include the nongenetic and epigenetic events of early embryology that determine early morphology and the dynamic, stochastic, and selective nature of neurogenesis (Edelman 1987; 1988). Contemporary dynamic systems theories of development emphasize that the organism and its nuturing environment cannot be logically partitioned because behavior is never context-free; it is always an emergent property within that context. From the earliest days, the organism is constructed in interaction with its surrounds in a manner far more complex and nonlinear than the simple models of behavioral genetics. Plomin & Daniels's "nonshared environment" reflects, not poorly measured variance, but the very stuff of development: individual differences built by a nonlinear process, and, *in principle* unable to be "disentangled" into the "cause and effect" (Plomin & Daniels 1987, p. 21) models of behavioral genetics. Likewise, the environment as a phenotype is constructed by whole, intact people with developmental histories, and not by their genes.

A dynamic systems theory of development (DSTD) is a framework to answer the important questions posed by P&B without the genes-environment dichotomy (Thelen 1989; Thelen et al. 1987). DSTD offers an alternative to the linear and additive assumptions of the genetics approach with concepts from modern nonlinear dynamics, especially *synergetics* (Haken 1983), a theory of pattern formation in complex systems. In DSTD, behavior is an emergent property of the cooperation of the multiple organic subsystems in a particular physical and social context. Because the elements are free to combine in fluid and task-specific ways, causality cannot be assigned to any element in the organism or the environment alone. Behavior is truly constructed during development as certain cooperative interactions of elements coalesce and become stable, while others lose stability and disappear from the behavioral repertoire. Thus, the empirical task is not the statistical analysis of such endpoint traits as IQ, but the tracing of the developmental trajectories of variables that express the cooperation of organism and environment to identify periods of stability and change. A basic assumption is that the cooperation between organism and environment is dynamic, that is, it has a time-history, and is also highly nonlinear. The same behavior may therefore be assembled over time by different routes at the same time that very small differences in routes may be amplified to produce divergent end states. This means that a dynamic analysis is essential to begin to understand which processes and mechanisms are stable within the individual and between individuals, and which are the variables that engender developmental change.

Why do we want to know what part of behavior is "genetic" and what part is "environmental"? If the root question is one of the sources of individual differences (Thompson 1990), the purity of the analysis is already seriously compromised by the so-called genetic influence on environmental measures as well as the fundamental nonlinearity of developing organisms. If the goal is to plan interventions, we want to know about stability and the agents of change. The arbitrary partitioning into genes and environment does not help this task and imposes oversimplified assumptions, which must be strained and modified to fit the data. If understanding developmental process is the aim, P&B are not convincing about what the environment-genes partitioning adds to the conventional longitudinal studies.

Is $H^2 = 0$ a null hypothesis anymore?

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It was Robert Plomin (Plomin et al. 1977) who first brought to our attention the following assertion from Roberts (1967): "It matters not one whit whether the effects of the genes are mediated through the external environment or directly through, say, the ribosomes" (p. 218). To which Plomin et al. replied, "In practice it often matters quite a few whits, especially if one should be interested in intervening in the process. Changing behavior by changing parental attitudes is a decidedly different proposition than tinkering with the ribosomes" (p. 321).

It can hardly come as a great surprise that socioeconomic status, ratings of family characteristics, or self-reports of parenting styles have a genetic component. What doesn't? In recent years, genetic components have been demonstrated for social attitudes (Martin et al. 1986), divorce (Lykken et al. 1990), and indeed for practically every cognitive and personality variable imaginable. Our environment, especially to the extent we can measure it, consists of people, and almost all characteristics of people have turned out to be more or less heritable. When characteristics of people in the environment are measured through their own subjective impressions or those of others, there are two chances to detect genetic contributions, once in the subject and once in the rater.

Our concern is about where all this will lead. Behavior is influenced by genotype and environment. The environment provided by a parent is influenced by the parent's (not to mention the child's) genotype, and the parent's rearing environment, which had its own tangle of reciprocal genetic and environmental influences. Everything is intercorrelated; everything interacts. Where does this leave the columns of "model-fitting heritabilities," meticulously computed to two decimal places and starred for statistical significance on the basis of path models that cannot hope to keep pace with the reciprocal causal structures described in the target article?

Opponents of the behavior genetic agenda have always argued that the dynamic interaction of genotype and environment was too complicated to permit meaningful decomposition of phenotypic variance into independent genetic and environmental components (e.g., Taylor 1980). Many of these opponents, driven by an ideological aversion to *any* genetic influence on

behavior, proceeded to throw out the genetic baby with the bathwater of variance decomposition. The target article has demonstrated, yet again, the incontrovertible influence of genes on every aspect of human behavior, but in so doing it may weaken our already shaky confidence in the meaningfulness of traditional analyses of heritability – useful as they may have been in the earlier stages of our field. [See also Wahlsten: "Bias and Sampling Error in Sex Difference Research" *BBS* 11(2) 1988.]

Suppose, unknown to behavioral science, every 10 hours children spent watching *Jeopardy* added one point to their IQs. What would be the best way to go about discovering and understanding this phenomenon? First, consider the possibility that the tendency to watch *Jeopardy* may have – undoubtedly has (Plomin et al. 1990) – a genetic component. Children with a higher genetic loading for intelligence may find the show more interesting; intelligent parents may encourage their children to watch "educational" shows; childrens' inherited temperament may predispose them to watch one kind of show rather than another. Who cares? The important discovery that needs to be made is that an activity exists that can increase IQ, and the important quantification that needs to be accomplished is not to partition the variance of either *Jeopardy* or IQ, but rather to estimate the magnitude of the effect, that is, one IQ point per 10 hours watched.

The "Who cares?" in the above paragraph is a deliberate overstatement. It may, in some contexts for some populations, be interesting and useful to partition the variance of IQ. But consider that in our example, the heritability of IQ would be a function of the fundamental effect (one point per 10 hours), *in addition to* the variance of parental IQ (are these children unselected, or all from the same institution, or are they faculty kids?); the variance of *Jeopardy*-watching (if children do not vary much in number of hours watched, hours watched will account for little of the variance in IQ, and the effect will probably be missed entirely); and the variance of everything else that is influencing IQ. Add to this the myriad complexities stemming from the genetic-environmental cross-influences discussed in the target article, and the second decimals of those columns of heritabilities start to look pretty unimportant.

The point of Plomin et al.'s (1977) profound reply to Roberts (1967) is that variance partitioning can be deceptive, because variance can sometimes be manipulated. It may be that one of the ways nature has engineered for intelligent parents to transmit their intelligence to children is by predisposing such parents to provide facilitative environments, which are then, in some sense, genetic. But the provision of rearing environments is not a fixed aspect of nature: It is to some degree under our control. If social or educational programs enabled the less intelligent parent to provide the kind of rearing environment previously provided only by the genetically advantaged (a big *if*, itself a matter for empirical investigation), the genetic component of environment would be eliminated.

It is certainly important to remind behavioral scientists that genetic paths need to be included in all models of familial transmission, or for that matter in all models of behavioral development. Nonetheless, the very success of the behavior genetic agenda may have obviated the discipline's fundamental research paradigm. Having established beyond any reasonable doubt that *some* of the variance of *every* human characteristic is influenced by genes, it may be time to enshrine $H^2 \neq 0$ as the "first law of behavior genetics," and concede that $H^2 = 0$ is no longer an interesting null hypothesis.

NOTE

1. One might add that "genetic" measures also have environmental components. Biologic parent IQ, a common genetic measure in adoption studies, is clearly a measure of parental genotype *and* that parent's rearing environment.

Nature and nurture: A shaky alliance

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The major theme of Plomin and Bergeman's (P&B's) target article is that measures of the environment may contain genetic variance, which can be estimated with traditional quantitative genetic methods. Speaking as an environmental researcher, I have no quarrel with the possibility that genetic factors may be associated with measures of the environment. A paper looking at the interrelation of genetics and environment needs to pay as careful attention to environmental models and measures as it does to genetic models and measures, however. In addition, although the data presented by P&B suggest an *association* between genetic and environmental measures, it is doubtful whether they can be used to imply genetic *influences* upon environmental measures.

The need to consider environmental methods and models more closely. A prime example of the need to consider existing environmental models is seen in those sections where P&B discuss the SATSA study. Based on their assumption that the correlation for monozygotic twins who are reared apart "directly estimates heritability," they conclude that there is a genetic influence on certain dimensions of the Family Environment Scale. Such a conclusion would be valid *only* if we can assume that being reared in different families means being reared in totally different environments, *across all levels of the environment*. In making this assumption P&B ignore the contributions of ecological models of environmental action, which emphasize the need to consider multiple levels of the environment simultaneously. For example, as demonstrated by Bronfenbrenner (1986), when we take into account the larger ecological context, phenotypic similarities for separated monozygotic twins may reflect similar ecological contexts as much as similar genotype.

A similar problem is seen in P&B's treatment of environmental measures. The fact that a measure of the environment has been widely used in the literature does not necessarily guarantee its validity. (An excellent example of this point is seen in a recent review of parental rearing attitude measures by Holden & Edwards 1989.) Whereas it is possible to apply quantitative genetic methods to decompose any measure of the "environment," from an environmental standpoint it is important to ask what this environmental measure is actually assessing. For example, when discussing data from the Colorado Adoption Project, P&B present evidence on the possible association of genetic factors with "environment," as measured by brief videotaping of maternal behavior. What is not considered is the possibility that short-term videotaped measures of mothers may be assessing parental reactivity to being videotaped rather than representative parent behaviors (Wachs 1988), or that the environmental component of such brief measures is not likely to be stable across time (Wachs 1987). Given this, it is legitimate to ask whether the data reflect a genetic influence upon environment, parent reactivity or error.

With respect to other "environmental measures" decomposed by P&B, the problems with retrospective reports of the environment have been well documented for more than 20 years (e.g., Yarrow et al. 1970). Similarly, while *socioeconomic status* (SES) is often used as a measure of the environment, the adequacy of SES as a measure of the psychosocial environment is low, given that SES may also measure genetic variance, variance caused by biomedical factors, or variance resulting from nutritional status. Even with such widely used (objective) measures as the HOME, there are methodological factors that need to be considered – for example, whether the observer is in the home long enough to get an adequate sample of parent behavior, or whether there is sufficient environmental variance to produce meaningful HOME scores in middle class populations.