Simulating the dynamics of genes and environment in development

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Abstract

Biometric analyses of variability in behavioral phenotypes have demonstrated that genotype plays a significant role in all behavioral development, but the developmental significance of the environment has remained obscure. Behavior genetic analyses typically show the effect of shared family environment to be very small, although considerable variability remains to be explained after genetic factors have been accounted for. Behavior geneticists have suggested, contrary to intuition, that almost all of the important effects of the environment serve to make family members more different from each other. Environmentalists of several persuasions have pointed to the crucial importance of the environment in the initiation and regulation of all developmental processes. In this article, we present a series of simulations to suggest that some of the difficulty of identifying environmental effects in biometric models is methodological. Adding simple dynamic parameters to models of development leads to systems in which environment produces substantial variability that can be detected in the context of a particular genotype, but vanishes when genotype is allowed to vary.

Introduction

The outcome of the nature–nurture debate

To the extent that the nature–nurture debate was about whether or not genotype influenced the development of behavioral characteristics in humans, it is over. Twin and adoption studies have been practically unanimous in demonstrating substantial genetic components of variation in complex human phenotypes (Plomin, Owen, & McGuffin, 1994) ranging from schizophrenia (Gottesman, 1991) to marital status (McGue & Lykken, 1992). The realization that no aspect of human behavioral development is free from genetic influence—a fact we have suggested deserves to be called the First Law of Behavioral Genetics (Turkheimer & Gottesman, 1991)—has profoundly changed the terms of the nature–nurture debate. Environmentalists can no longer make a plausible case that properly conducted twin or adoption studies would show genetic influence to be negligible, or naively assume that the transmission of behavioral phenotypes from parent to offspring is conducted exclusively along sociocultural pathways. But the First Law presents problems for behavior geneticists as well. The very ubiquity of genetic influence has reduced the staple of behavior genetic research—demonstration of significant heritability for a behavioral phenotype—to a commonplace. It has also focused attention on some peculiarities of prototypical behavior genetic results, which demonstrate the importance of genetic influence in the most general sense, but leave many unanswered questions about the developmental mechanisms through which genes might affect behavior.

The mystery of the biometric environment

Although it has become fashionable to advertise behavior genetics as a "window on the environment." (Reiss, Plomin, & Hetherington, 1991) the environment has not fared well in behavior genetic studies of development.

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Turkheimer (1995) has suggested that the First Law of Behavior Genetics, positing the universal importance of genetic variation, is not particularly surprising when it is understood correctly. But the Second Law—which holds that once genetic factors have been controlled, variation in shared family environment contributes practically nothing to biometric analyses of phenotypic variation—is deeply counterintuitive and controversial. It is worth reviewing the empirical basis of this claim, as a basis for then exploring its limitations.

In its simplest form, the biometric model partitions variability in a phenotype into three parts: the additive effect of genes, the effect of shared family environment, and the effect of nonshared environment (Mather & Jinks, 1971). The additive effect of genes is roughly estimated by twice the excess similarity in identical twins compared to fraternal twins, or by the parent–child correlation between biological parents and adopted-away offspring. The effect of shared environment is estimated by fraternal twin correlations in excess of one half of corresponding monozygotic (MZ) correlations, or by correlations between biologically unrelated individuals within adoptive families. The effect of nonshared environment is basically what is left after additive genetics and shared family effects have been accounted for, although this variability is confounded with errors of measurement and other residuals.

A corpus of behavior genetic research has established that within the context of biometric research designs, the additive genetic and nonshared environmental components are substantial, whereas the shared environmental component is usually close to nil. In twin studies of adults, MZ twins are often *more* than twice as similar as DZ twins, putting modern maximum likelihood estimation procedures in the uncomfortable numerical role of preventing the shared environmental variance from becoming negative. Correlations among genetically unrelated individuals in adoptive families are modest but promising among young children, but quickly approach zero as children pass adolescence (McCartney, Harris, & Bernieri, 1990).

Can it really be true that variation in shared family environment contributes little or nothing to variation in adult behavioral phenotypes? Not surprisingly, mainstream behavior geneticists have been most willing to take the Second Law at face value. It is generally accepted in behavior genetic circles that variation in "normal," or "good enough" (Scarr, 1992) environments has little to do with variation in behavioral phenotypes in adulthood, and that variation among the child-rearing methods of typical American families does not exert a strong causal influence on child development (Rowe, 1994). Behavior geneticists have observed (a) that organisms seek out environments in accordance with their genotype (Scarr & McCartney, 1984); (b) that survival of the species depends on a certain degree of robust resistance to variation in a broad range of normal environments (Scarr, 1992); (c) that many aspects of what we traditionally think of environment (parental rearing styles, for example) are themselves subject to significant genetic variation (Plomin & Bergeman, 1993); and (d) that the important actions of the environment cause family members to be dissimilar rather than similar (Plomin & Daniels, 1987). All of these considerations would serve to reduce the biometric contribution of normal family environment to the development of phenotypic variability.

Contemporary environmentalists are more cognizant of genetic transmission of variability in behavior than those of a generation ago, but remain hostile toward the strong behavior genetic contention that familial effects in the normal range are unimportant in behavioral development. The traditional environmentalist line of attack against strong genetic claims involved questioning the methodological or statistical validity of biometric studies (e.g., Taylor, 1980). Many of these criticisms may have some merit, but the relentless replication of the importance of genotypic variation (Plomin & McClearn, 1993) and the lesser importance of variation in shared family environment (Rowe, 1994) have made it increasingly difficult to make the case that changes to statistical assumptions of biometric analyses would radically alter their results.

More recently, some environmentalists

have conceded the importance of genetic variation in human phenotypes, and presented a new argument for the importance of the environment for development. Following a seminal paper titled, "The Analysis of Variance and The Analysis of Causes," (Lewontin, 1974) these environmentalists have suggested that developmental science was never about partitioning the variability of phenotypes in the first place; rather, that the goal is to specify the particular *causal processes* underlying the development of phenotypes in individual organisms. Causal development theories of this kind necessarily involve interactions between genes and environments, and not just in the statistical sense: real genes must interact with real environments before anything can develop at all. Heritabilities approaching unity at one extreme or zero at the other do not imply that genes or environment are unimportant for development, and the numerical value of heritability does not quantify the relative importance of genes and environment for development. Both are always crucial.

At the risk of rehashing an often-repeated example that has already been passed down from Mark Twain to Hebb (1970) to Lerner and von Eye (1992) to Bronfenbrenner and Ceci (1994), consider a biometric experiment based on Twain's recommendation that boys should be raised in barrels to the age of 12 and fed through the bunghole. Because the boys were raised identically, environmental variation would account for none of the variation in their behavioral phenotypes, which would therefore have heritabilities close to unity, but it would be incorrect to use such a study to conclude that being raised in a barrel has no *causal* influence on development.

In its strongest form, this new environmentalist argument implies that specification of individual developmental processes is the *only* legitimate goal of developmental science, and that population-based variance partitioning is pretty much irrelevant to the understanding of behavior (Gottlieb, 1991; Gottlieb, 1995). In a reply to the strongest extant statement of the genetic view—Scarr's (1992) Presidential Address to the Society for Research in Child Development—Baumrind (1993) concluded:

The proportion of genetic variation among individuals in a particular trait in a particular populations says nothing about how or why individuals differ in their development, how to nurture that development, or how genes and environment interact. . . . *How* heredity and environment interact to produce a phenotype, unlike *how much* of heredity and of environment produces a phenotype, is of considerable scientific interest. But the how question, unlike the how much question, requires knowledge of what actual environmental circumstances interact with which actual genetic processes in the development of a trait. (p. 1313)

Behavior geneticists have replied that analysis of variance is one thing and specification of causal processes in individuals is another, and that in fact, populations are *more* important than individuals when it comes to understanding development (Scarr, 1995).

We have suggested that individual- and population-based contributions are both necessary for a science of development, and moreover that the two views are not as diametrically opposed as they are often made out to be (Turkheimer & Gottesman, 1991; Turkheimer, Goldsmith, & Gottesman, 1995). Recently, compromise views have been offered by more environmentally oriented theorists as well (Bronfenbrenner & Ceci, 1994). The two approaches remain at odds, however, because no theory has been developed to connect individual causal processes with variation in populations, and thus to explain the apparent contradictions between them. If normal family environment is a crucial aspect of the development of individual humans, why exactly does it fail to contribute to biometric analyses of populations, which are, after all, composed of nothing but individuals and their causal processes (Turkheimer, 1991)?

The nature–nurture debate has evolved. The old question of whether genotype contributed substantial variability to behavioral outcomes has been decisively answered in the affirmative. The remaining question involves the role of the environment. On a microdevelopmental level, it is clear that gene–environment interactions are a crucial requirement for development, but in biometric analyses of populations the effects of families seem to evaporate. After genetic considerations have

been taken into account, why do some children do badly in school, become delinquents, or resist their impoverished beginnings and go on to positive outcomes? Even for the most heritable phenotypes, like schizophrenia, MZ twins reared together fall considerably short of perfect concordance, presumably because of some kind of environmental variation (cf. Roses, 1996), but generations of intense investigation have failed to discover specific environmental events predisposing to important behavioral outcomes. In this article, we introduce a set of simple simulations of dynamic interactions among genes and environment, with a goal of demonstrating that more dynamic models of behavior can reproduce some of the more perplexing aspects of environmental contributions to development.

Simulation as an alternative to experimentation

Psychology is not the only science confronting phenomena that resist direct experimentation and linear prediction. Meteorologists interested in the long-term prediction of weather cannot produce hurricanes in the laboratory or in nature, and the predictability of weather systems rapidly approaches zero over periods longer than a few months. Under such less than optimal scientific conditions, one empirical approach involves building simulations of the phenomena of interest, usually on computers. If a computer can use fairly simple parameters to simulate a hurricane, it becomes more plausible that something like those parameters might be involved in the production of actual hurricanes. And if the simulation becomes good enough, it may eventually be useful in the prediction of real phenomena, as is currently the case with the weather.

One way to understand the statistical assumptions of population-based methods that are used in traditional behavior genetics—independent and additive genes and environments, and additive combination of multiple genes—is as a set of simple parameters for simulating the similarity of genetically and environmentally related individuals. Although these methods have the advantage of allowing numerical estimation of relevant parameters

(an advantage the simulations described below will not share), and have been very successful in identifying genetic components of behavioral variation, they have not done as well in formulating useful theories of environmental action. In the remainder of this article, we propose a somewhat more complex method of simulating the dynamics of genes and environment in development.

Methods

The simulations to be described in this article are conducted in a two-dimensional space, as illustrated in Figure 1a. The two dimensions of the space can be thought of as hypothetical independent dimensions of phenotype (e.g., neuroticism and extraversion). Located in this space are a number of "genes" that affect the development of the simulated organism. The genes, which might be more accurately referred to as "alleles," are intended only as units of genetic influence; we are not attempting to simulate the actual mechanics of genetic transmission at this time. Ten genes are denoted as "G" in Figure 1. Also in the two-dimensional space is the simulated phenotype of an organism, denoted as "P" in Figure 1, and the initial location of the organism in the environment, denoted as "E." The location of the phenotype at each point in time represents the organism's current values on the two trait dimensions. The location of the environment represents the favorableness of the organism's environment for the development of the two traits.

As the simulation proceeds, the organism's genotype (i.e., the location of the Gs in the two-dimensional space) remains fixed. The organism's phenotype and location in the environment change dynamically, however, according to the following rules:

1. The phenotype is attracted to the genes in the space. That is, it will tend to move in the direction of the Gs.

2. The relative strength with which each gene attracts the phenotype depends on the location of *environment*. When the environment is located close to a gene, the attraction of
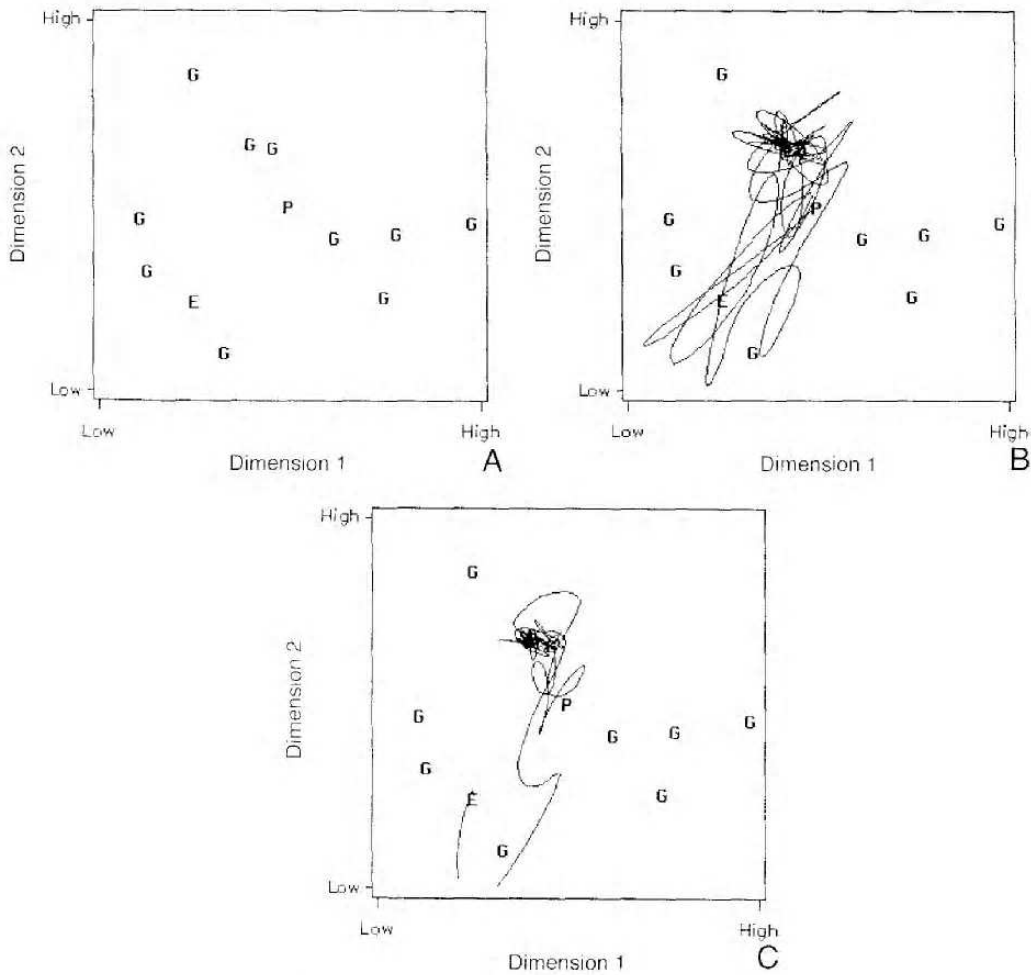
Figure 1. Two-dimensional space simulations.

the phenotype to that gene becomes stronger.

3. The phenotype is attracted to the environment.

4. The total amount of attraction exerted on the phenotype by the genes and the attraction of the phenotype to the environment are fixed to be precisely equal. This rule is important because it eliminates the traditional nature–nurture question from the simulation. At each "moment" in the organism's "life" the relative influences exerted by genes and environment are equal.

5. The environment is attracted to the current location of the phenotype. That is, over time the organisms environment will tend

to be a reflection of its phenotype (which in turn depends in part on the environment).

6. Both phenotype and environment have momentum. They will tend to keep changing in the direction in which they have been moving, unless genes or environment cause them to do otherwise.

7. For the first quarter of its "life," the organism's environment cannot be influenced by phenotype. We refer to this condition as "childhood."

At each iteration in the simulation, the program computes the forces acting on the phenotype and the environment, moves them accordingly, then recomputes the forces. The

organism's "life" comprises 200 such iterations. The simulations are programmed in APL2 for OS/2, which runs on PC-compatible computers. (Source code for the program is available from the first author.)

The final result is a plot of the complex curvilinear path followed by the organism and the environment, as illustrated in panels b and c of Figure 1. Figure 1b traces the path followed by the phenotype, and Figure 1c traces the path followed by the environment. As is the case in Figure 1, most configurations of genes and environment eventually become "canalized" in a circumscribed region of the space as both the phenotype and the environment converge on a region of the genotype. We compute the mean location of the phenotype on dimensions 1 and 2 of the space during the final 50 iterations as a measure of the final phenotype achieved by the organism.

The remainder of this article will describe some preliminary investigations of the effects of variation in genotype and starting environment on the final phenotypes of simulated organisms. We will conduct these investigations in the form of simulated "experiments," in which samples of organisms are allowed to develop under different genetic and environmental conditions. We hope to show that many features of behavior genetics can be reproduced by the addition of more dynamic elements to the interplay of genes and environment.

Experiment 1: Genotypic reaction norms

Building on concepts introduced to genetics by Schmalhausen (1949/1986) and Dobzhansky (1955), Gottesman (1963) proposed that the *reaction norm* and its close cousin, the *reaction range*, are the most informative methods for representing the outcomes of studies of genes and environment. A reaction norm is a graphical representation of the outcome of phenotypic development under different combinations of genes and environment. A reaction range is the difference between the upper and lower extremes of phenotypic development for a given genotype across a specified range of environments (Turkheimer & Gottesman, 1991). Although reaction norms are the most useful method for representing the outcome of behavior genetic studies, they are notoriously difficult to estimate in humans because the crucial experimental designs, in which cloned organisms are reared in a variety of contrasting environments, can only be distantly approximated by available twin and adoption methods (Platt & Sanislow, 1988; Gottlieb, 1991). Our simulated model of genes and environment allows us to explore what happens when a single simulated genotype develops under a variety of environmental conditions. We selected at random the 10-gene system illustrated in Figure 1. In each of 100 simulations, the phenotype started at the center of the space, and the environment started at points in which the location on the x axis was fixed at 50, and the location on the y axis varied from 1 to 100. For each starting point, we recorded the means of the final 50 (out of 200) locations of the phenotype on x and y.

The results of the simulation are illustrated in Figure 2, in which final mean phenotype is plotted on the y axis against the starting point of the environment on the x. The line through the points is a smoothing spline, which approximates the reaction norm for the one genotype as the environmental starting point is varied. Several aspects of the results should be noted. First, varying the starting point of the environment obviously had an effect on the final phenotype, because starting environment was the only factor varying across simulations. The variance of the final mean phenotypes, 173 units, is therefore entirely "environmental" in origin. Other aspects of the relationship suggest some of the difficulties that will arise when we study environmental effects under less controlled conditions. The relationship appears highly nonlinear, with regions in which environmental variation produces major changes in phenotypic outcome, and other regions where it is relatively unimportant. There also appear to be sudden threshold changes in the relationship. For example, when the environmental starting point is about 50, there is a sudden shift from final phenotypes that are mostly around 40 to phenotypes mostly around 60. Finally, across all parts of the reaction norm, one can see individual points that vary consid-
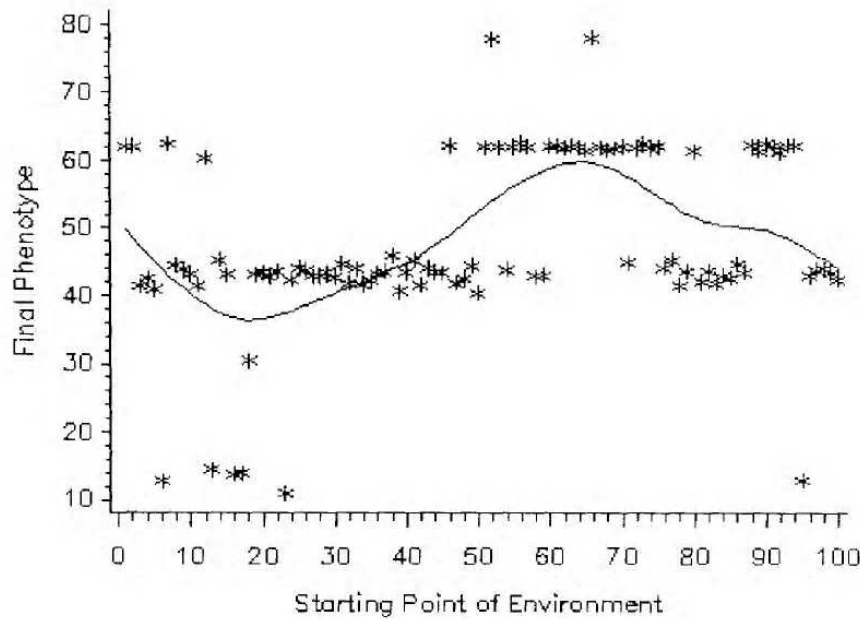
Figure 2. Results of Experiment 1.

erably from those in the immediate neighbor-hood. For example, a starting environmental value of 94 results in a final phenotype of about 12, whereas starting points of 93 or 95 both result in final phenotypes over 40.

Experiment 2: An environmental reaction norm

Traditionally, reaction norms have been drawn as contour graphs, with each contour representing the phenotypic outcome of a single genotype across a range of environments. More recently, we have shown that reaction norms can be also represented as regression surfaces (Turkheimer & Gottesman, 1991), and that the reaction range concept can be extended to describe the range of phenotypic development for a given environment across a range of genotypes (Turkheimer, Gottesman, & Goldsmith, 1995). We conducted a simulation of such an "environmental" reaction norm in Experiment 2. For a fixed environmental starting point of (25, 25) we generated 100 random genotypes and computed the final mean phenotypes on dimensions 1 and 2. Results for Dimension 1 (Dimension 2 is very similar) are illustrated in Figure 3.

There was a strong positive relationship

between mean genotype and final mean phenotype on both dimensions, and the nature of the relationship appears to be more systematic than was the case in the previous experiment. The relationship appears to be essentially linear ($r = .65$ for both dimensions) without evident thresholds or drastic deviations from the general trend. The phenotypic variation of 417 units on Dimension 1 and 408 units on Dimension 2 is entirely genetic in origin.

Experiment 3: Random genes and random environment

In the third experiment, we simulated a situation that is more typical of actual studies of human behavior by studying phenotypic variation in genotype and environment simultaneously. That is, for each of 100 trials, we generated a random set of 10 genes and a random starting place for the environment. We noted once again the mean of the final 50 locations of the phenotype on the two dimensions of the space. Results are illustrated in Figure 4. Whereas the covariation between genotype and phenotype was unaffected by the inclusion of environmental variation ($rs = .63$ and .73 on Dimensions 1 and 2), there is no longer any discernable relation between environmen-
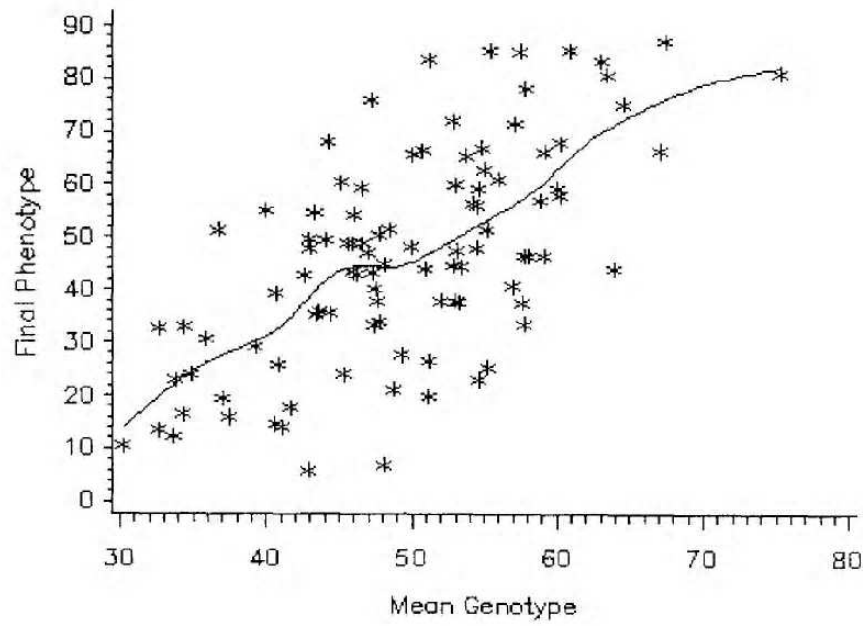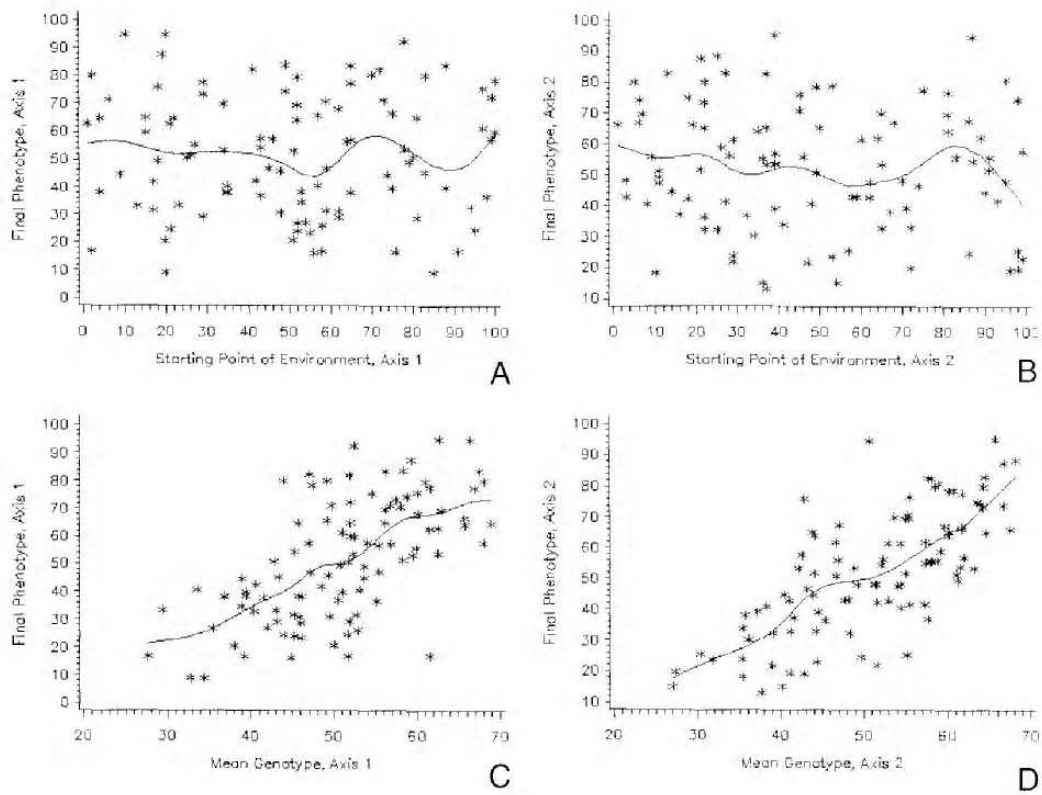
Figure 3. Results of Experiment 2.

Figure 4. Results of Experiment 3.

tal starting point and final mean phenotype. We then conducted an analysis of variance in which we predicted final mean phenotype from mean genotype, environmental starting point, and their interaction. Only genotype was a significant predictor, accounting for 40% of phenotypic variability on Dimension 1 and 49% on Dimension 2. These percentages represent the percent of phenotypic variance accounted for by genotype when environmental variation is controlled, so they may be thought of as the heritabilities of the simulated trait. On both Dimensions 1 and 2, variation in environmental starting point accounted for less than 1% of the variance. The total phenotypic variation, 471 units on Dimension 1 and 405 units on Dimension 2, is essentially the same as in Experiment 2, which included no environmental variation at all.

Discussion

We have described a simulation of the dynamic interaction of genotype and environment, which reproduces some of the more perplexing observations of the genetics of complex human behavior. The influence of genotype and environment are fixed to be co-equal at each moment in the development of a simulated organism. We then observed the cumulative effects of genotype and environment over a simulated lifetime. Although variation in simulated environmental starting point produces variation in phenotypic outcome for a single fixed genotype, the environmental variation is only detectable when genotype is held constant. If genotype is allowed to vary, no covariation with environmental starting point can be detected. It appears that environmental variation in this simulation can only be detected in the context of a *particular* genotype.

In the simulations, therefore, variation in genotype is associated with equifinality in development, in the sense that individuals with similar genotypes tend to reach similar developmental outcomes. The relationship between environmental variation and developmental outcome appears to be more complex. As the reaction norm illustrated in Figure 2 illustrates, environmental variation is associated with equifinality in development *within a single organism, or a sample of cloned organisms.* This suggests why the effects of environment on development have been easiest to detect in the context of comparative studies of lower animals, where the dynamic interactions of genotypic and environmental variation are easier to keep under control. But in the human realm, where equivalent genetic and experimental controls are rarely possible, the exquisite sensitivity of environmental effects to uncontrolled variation in genotype means that environmental variation is more often associated with multifinality in developmental outcome. Over a lifetime, tiny differences in environmental conditions can produce substantial and unpredictable variation in development.

We recall a story about a diligent mother who rewarded her child for eating spinach by giving him a bowl of ice cream.[1] Will the child grow up loving or hating spinach, loving or hating ice cream, or loving or hating mother? A generation of behavior genetic research has demonstrated that a study of such child-rearing practices with proper genetic controls would in all likelihood show small environmental effects or none at all. But should such findings lead us to the conclusion that variation in child-rearing practices are unimportant to phenotypic outcome across a broad range of "good enough" environments? Our results suggest that such negative conclusions may be premature. Instead, it may be that the effects of ice cream reinforcement are substantial, but depend for their direction on individual configurations of genotype (and, probably, other environmental variables as well). If interviewed, a sample of subjects reinforced with ice cream as children might each tell of the important consequences of their mothers' unusual culinary discipline, although some had become vegans and others sweet-crazed gourmands.

The simulation suggests some other difficulties of studying the environment in a genetic context. Whereas phenotypic variation associated with genotype was well fit by lin-

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1. We are uncertain of the origins of this example, but it is not ours.

ear models, environmental variation was non-linear and even discontinuous. Small changes in environment resulted in large and sudden changes in phenotypic outcome that would be very difficult to fit with traditional linear models. Suppose, for example, that the two extreme outcomes illustrated in Figure 2 (with phenotypic values around 78) represent a form of pathological developmental outcome. Attempts to discover the relationship between environmental starting point and such outcomes would appear to be virtually impossible. The extreme phenotypic outcomes are associated with intermediate values of environment, so linear models would not fit the relationship; moreover, other individuals starting in environments very close to the two extreme points do not show the extreme outcome.

Finally, the simulations may offer some insight into the relationship between shared and nonshared aspects of the environment. Although we did not create simulated siblings in this set of experiments (we are currently doing so), the environmental variation in these studies, based on the starting location in the environment, simulated the *shared* environment. Two simulated siblings in the same family would share an environmental starting point. But if the effects of the environment depend on genotype, the effect of the shared environment might be to make the siblings dissimilar rather than similar. This suggests that we need to make a distinction between shared environmental *events* and shared environmental *effects*. Environmental events shared by children may affect them differentially.

A fundamental question underlies any interpretation of these simulations in terms of the roles of genotype and environment in the development of actual phenotypes in real organisms: Can one take seriously the possibility that the mechanics of behavioral development resembles the dynamic motion of bodies in space? The answer to this question is clearly no. But it is important to remember that traditional models of genes and environment, with their assumptions of additive genetic effects that combine additively with uncorrelated environments, do not provide a very realistic model of actual genes and environments either. Indeed, unhappiness with the developmental oversimplification of behavior genetic models has motivated much of the opposition to behavior genetics that has dogged it since its inception. We hope these simple dynamic simulations may offer a first step in the direction of a more complex synthesis of behavioral genetics and environmentally informed developmental psychology.

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