Spinach and Ice Cream: Why Social Science Is So Difficult

Eric Turkheimer

Introduction

The nature–nurture debate has entered the postbiometric era. A generation ago, it seemed to behavioral geneticists and their opponents that the outcome of the nature–nurture debate depended on whether or not biometric research designs analyzing covariance among twins, adoptees, and other family members would reveal substantial genetic contributions to important domains of human behavior. That research has now been conducted, with nearly unanimous results: Variation in all behavior, including everything from schizophrenia (Gottesman, 1994) to marital status (McGue & Lykken, 1992; Turkheimer, Lovett, Robinette, & Gottesman, 1992), has a genetic component.

I have summarized the consistent outcome of a generation of behavioral genetic research as “The Three Laws of Behavioral Genetics,” stated simply as follows (Turkheimer, 2000):

1. Everything is heritable.
2. The environmental effect of being raised in the same family is substantially smaller than the genetic effect and is often close to zero.
3. Most behavioral variability remains in the error term after genetic effects and the effects of being raised in the same family have been accounted for.

These findings are no longer in serious dispute. What remains is to understand what they mean for the development of human behavior.

The unanimous outcome of biometric family research has led to a realignment of historical positions on both sides of the nature–nurture debate. On one side, traditional population-based behavioral genetics finds itself somewhat exhausted by its own success. For years, the bread and butter of behavioral genetic research consisted of demonstrations of heritability for one behavior after another, each appearing less likely than the last: schizophrenia (Gottesman &
Shields, 1972), intelligence (Cardon & Fulker, 1993), and personality (Loehlin,
1993) were followed by job satisfaction (Arvey, McCall, Bouchard, Taubman, &
Cavanaugh, 1994), religiosity (Waller, Kojten, Lykken, Tellegen, & Bouchard,
1990; Bouchard et al., this volume), and television watching (Plomin, Corley,
DeFries, & Fulker, 1990). As it has become apparent that some degree of heri-
tability is practically inevitable, behavioral geneticists have had to turn to other
frontiers, most notably multivariate biometric methods (Plomin, 1986) and
molecular genetics (Gottesman, 1997; McGuffin, Riley, & Plomin, 2001).
Although the prospects for multivariate behavioral genetics and molecular
behavioral genetics are both matters of intense and controversial theoretical
interest (Turkheimer, 2000; Turkheimer, Goldsmith, & Gottesman, 1995;
Turkheimer & Gottesman, 1991), this chapter will place greater emphasis on the
challenges faced by environmental social science in the postbiometric era.

On the environmental side, the idea that variability in almost all human
behavior has a detectable genetic component no longer evokes the controversy
it once did. Most environmentally oriented social scientists are willing to
accept the idea that human characteristics such as intelligence, personality,
and psychopathology are transmitted in part along genetic pathways (Bron-
fenbrenner & Ceci, 1994). The controversy that remains concerns the role of
the environment. Although modern behavioral geneticists contend that their
methods and results are environment-friendly, leaving plenty of variability
unexplained after genetic variation has been credited, environmentalists, as a
group, have remained dissatisfied (Maccoby, 2000). Why? If the simple fact of
genetic “influence” (to use the geneticists’ catchall term for the innumerable
and largely unknown ways in which gene products can exert causal effects on
complex human behavior) no longer troubles environmentalists and if the
behavioral-genetic models leave room for environmental influence on
behavior, what do contemporary environmentalists have to worry about? Why
not do as the behavioral geneticists suggest and render the geneticists what is
theirs, leaving environmentalism with its own domain, a domain no longer
confounded with genetic variability?

This chapter examines the remaining sources of environmentalist dissat-
sisfaction with behavioral genetic claims, seeks to understand them in terms of
some little-examined aspects of population genetic methodology, describes
simulations of complex gene–environment systems that reproduce some of the
more puzzling results of twin and adoption studies, and offers some recom-
endations for how the remaining differences between environmentalists and
behavioral geneticists might be resolved. I begin by characterizing some of the
new alignments of hereditarism and environmentalism that have been
necessitated by the three laws of behavioral genetics.

Nonshared Environmentalism

Behavioral geneticists are correct when they assert that biometric results have
not excluded environmental variability from the developmental picture, but
the environment has been included in a different form than had been antici-
pated. In its simplest form, the biometric model partitions phenotypic vari-
ability into three components: the additive effect of genes (A), the environment that is shared among family members (C), and the environment that is not shared among family members (E). The shared environment refers to environmental effects that serve to make children reared in the same family more similar; nonshared environment tends to make them different. Most environmentally oriented work in the social sciences—what Rowe (1994) has termed “socialization science”—has focused on the effects of families on children, that is, on the shared environment. If having a depressed mother has a discernible environmental effect on the personality of children, then multiple children reared by the same depressed mother should be similar along this dimension, and the similarity of siblings within such families would be reflected in the biometric variance component attributable to the shared environment.

During the last 15 years, Robert Plomin and his colleagues have argued that environmentalists’ historical focus on the shared environment is incorrect (Dunn & Plomin, 1990; Plomin & Daniels, 1987; Rowe & Plomin, 1981). As summarized by the second law of behavioral genetics, biometric components attributable to the shared environment are very difficult to find when appropriate genetic controls are employed. After genetic similarity has been accounted for, biological siblings often appear no more similar than individuals chosen at random from the population. For practically all behavioral traits, monozygotic (MZ) twins are nearly twice as similar as dizygotic (DZ) twins (if not more than twice as similar, which is an indication that something is awry in the twin model, a phenomenon that will be investigated later). Correlations between adoptive parents and children are usually much smaller than correlations between biological parents and children (Braungart, Fulker, & Plomin, 1992). The biometric implication is that little or no variability can be attributed to the shared environment. Nevertheless, genetically identical monozygotic twins usually show substantially less than perfect similarity for these same behavioral traits, so something must account for the residual dissimilarity.

Although the assertion that the residual variability in biometric models can be attributed to systematic effects of the nonshared environment is plausible, it must be noted that the evidence for the claim is still very incomplete. Traditional biometric models of twins and adoptees only demonstrate that a substantial proportion of phenotypic variability cannot be accounted for by the systematic effects of additive genotype or shared environment; they do not demonstrate conclusively that the remaining variability is attributable to the nonshared environment, unless the term “nonshared environment” is applied very loosely.

Direct evidence that nonshared environmental events are the cause of nonshared environmental variability must come from studies that include specific measures of the nonshared environment. Since the publication of Plomin and Daniels (1987), there has been a proliferation of studies of this type, which Mary Waldron and I (Turkheimer & Waldron, 2000) have reviewed. The conclusions to be drawn from this literature are crystal clear: Although the variance component called nonshared environment is indeed a substantial component of most behavioral variability, the specific effects of measured environmental variables cannot come close to accounting for it (Turkheimer & Waldron, 2000). Furthermore, when traditional biometric designs are generalized to the multivariate case, nonshared environment is
found to contribute to univariate variability but not to multivariate covariability among variables (Waldron & Turkheimer, 2000). Both genes and environment constitute the variability in behavioral measures; for the more compelling question of why variables are related to each other, only genes seem to matter.

Developmentalism

One important contemporary line of opposition to behavioral genetics finds its source not in environmentalist social science but in embryology and developmental biology. Historically, theoretical considerations in developmental biology have not focused on the sources of variation in phenotypic individual differences but rather on the viability of distinctions between inborn and acquired characters, or, in embryology, between preformationist and epigenetic accounts of ontogenesis (Oyama, 1985). The predominant modern view is that there is less than meets the eye to distinctions among biological, native, or instinctual characteristics on the one hand and learned or acquired characters on the other. All biological characteristics, according to this account, emerge out of a matrix of developmental interactions between biological and environmental elements, and because both are required for all development, neither can be more fundamental than the other for any particular phenotype.

Developmental biologists with this kind of outlook have always been suspicious of the variance partitioning methods of behavioral geneticists, which appear to violate their insistence on the unity of biology and environment in development and their preference for concrete ontological processes over statistical variance partitioning. Most recently, Gilbert Gottlieb has undertaken the task of extending these arguments from developmental biology to a full-fledged attack on the methodology of behavioral genetics. Replies to some of Gottlieb’s objections to behavioral genetics already appear in print (Turkheimer & Gottesman, 1991; Turkheimer, Goldsmith, & Gottesman, 1995), and I only outline the position in this chapter. (For a firsthand account, see Gottlieb, 1991, 1992, 1995, and, for another consideration of the developmental position, see Schaffner, 1998).

The basis of the developmentalists’ case against behavioral genetics is not to deny that genes play a significant role in the development of behavior, as past environmentalists have argued, but rather to contend that the intricate ontological interplay of genes and environments in development cannot be captured by the mathematics of population genetic research methods. According to the developmentalists, the goal of developmental science is to specify what Gottlieb calls the “developmental processes” that lead to behavioral phenotypes. Developmentalists can point to many carefully specified theories of behavioral development in animals (e.g., Gottlieb’s 1985 studies of ducklings) that they contrast unflatteringly with the variance partitioning of behavioral genetics. What do we learn about the development of personality, they might ask, by noting that 40% of phenotypic variability is “explained” by variation in the genotype (Bouchard & Loehlin, 2001)? They offer a radical answer: nothing at all.
Some skepticism about the causal or etiological content of behavioral genetic models may well be warranted, but it would be a significant overreaction to abandon behavioral genetics entirely, as developmentalists sometimes urge. Behavioral genetics has played an important role in demonstrating that radical environmentalism was incorrect, and it would be foolhardy to allow reasonable criticisms of population genetic research methods to lead us back to a time when correlations between biological parents and their children were naively accepted as unalloyed indicators of sociocultural transmission. Moreover, the successes of experimental animal psychology have been in the explanation of species-typical behavior in lower animals, whereas socialization science and behavioral genetics are more focused on individual differences in humans (i.e., personality, intelligence, and psychopathology; Scarr, 1995; Turkheimer & Gottesman, 1991), and it remains to be demonstrated how experimental methods from one domain can be applied to the other. Many, if not most, conclusive animal experiments cannot be performed with humans for obvious ethical reasons. It is still an open question whether multivariate behavioral genetics and molecular behavioral genetics will overcome Gottlieb’s objections by providing a quasi-experimental bridge between population-based variance partitioning and causally specified developmental models. Finally, it must be remembered that—thanks to all of these methodological difficulties—there are no solid developmentalist theories of human individual differences to contrast with behavioral-genetic models. The theories that do exist are socioenvironmental, and they are every bit as correlational as the genetic ones.

Antisocialization Theorists

Some theorists have taken the biometric results of twin and adoption studies at face value and concluded that the reason family environment explains little or no variance in twin or adoption studies is just what it seems: It is because parenting and other aspects of normal families have few if any long-term causal effects on children. The preeminent theorist of this kind is Sandra Scarr, whose 1992 presidential address to the Society for Research in Child Development was the apotheosis of decades of behavioral-genetic successes and galvanized a generation of environmentalists in opposition. Scarr (Scarr & McCartney, 1983) suggested that genes drive experience, by which she means that the most important aspects of the environment are those that are actively sought out by organisms and that organisms seek out environments according to their genotype. So the environments that families provide don’t much matter, as long as rearing conditions are “good enough” for the child to be able to seek out environments according to his or her genetic endowment. According to Scarr, environmental variability predominates within families because of the accrual of gene–environment correlation during development. As children, driven by their genotype, seek out suitable environments and evoke genotype-appropriate responses from their caregivers, environment and genotype become increasingly correlated within families, so genetic differences among siblings are magnified by the environmental differences they evoke.
More recently, somewhat similar theories have been advanced by behavior geneticist David Rowe and developmental psychologist Judith Rich Harris. Rowe (1994) documented the refusal of traditional socialization science to take seriously the methodological consequences of behavioral genetics, the ongoing failure to detect substantial shared environmental effects when genetic variance is appropriately controlled, and what he views as the disappointing results of environmental intervention programs. Harris’ (1998) theory focused less on genetics than on the transient and context-specific effects of the environment. Families matter as long as children remain in the context of the family, but as children grow, they are increasingly exposed to environments outside the family, environments that are to a large degree of their own choosing, as Scarr emphasized. This consideration led Harris to focus on the role of peer groups in the determination of childhood behavior.

**Where Things Stand**

The three positions outlined in this chapter each offer a more subtle view of the role of genes and environment in development than was implied by the genes versus environment face-off of the old nature–nurture controversy, but a great deal remains to be resolved. Everyone agrees that genes and environment must combine in some way to produce development, but consensus does not extend very far beyond this developmental cliché. The positions remain at odds because to one degree or another, they continue to cling to the old-fashioned contest between nature and nurture for supremacy in developmental models. Developmentalists make a plausible accounting of the shortcomings of population-based behavioral genetics for the explanation of the developmental mechanisms of behavior, but despite their proclamations of gene–environment integration in development, they give in to the environmentalist temptation to conclude that the consequences of genetic variability for behavior can safely be ignored. Antisocialization theorists, conversely, have used the surprisingly consistent, and consistently genetic, outcome of biometric family studies to argue that the shared effects of childrearing practices can safely be ignored. A domain of discourse in which it is seriously contended that either normal family environment or the methods of population genetics are of little relevance to the explanation of human development is in need of some new possibilities, and the goal of this chapter is to provide some.

**Methods**

The remainder of this chapter reports the results of a series of simulations of the interactions of genes and environment in development. (An earlier version of the method was described in Turkheimer & Gottesman, 1996; source code for the computer programs is available from the author.) The simulations take place in a two-dimensional space, which is illustrated in Figure 11.1A.
**Figure 11.1.** (A) Initial configuration of system. "G" = genes; "E" = environment; "P" = phenotype. (B) Phenotype is dynamically attracted to genes. (C) Phenotype is dynamically attracted to environment. (D) Environment is dynamically attracted to phenotype.
The dimensions of the space may be thought of as two independent dimensions of phenotype, such as intelligence and extraversion or sociability and conservatism. Three kinds of simulated entities are contained in the space: phenotype (P), genes (G), and environment (E). The locations of each are to be understood in terms of their relevance to the behavioral dimensions of the space, according to rules that will be specified more precisely here. The location of the phenotype at any moment of the simulation indicates the organism's current phenotypic values on the two dimensions. A phenotype in the lower left of the space would indicate that the organism currently has low scores on both phenotypic dimensions, and a phenotype in the upper right would indicate that the organism is currently high on both. The locations of the genes represent the direction of their influence on the phenotype of the organism. A gene in the upper left of the space will tend to pull the phenotype in that direction as it develops. The location of the environment has a similar interpretation: When the environment is in a particular region of the space, the phenotype will be pulled in that direction as it develops. If the environment is located in the lower right of the space, therefore, one can think of it as an environment that is favorable to the development of Dimension 1 but not Dimension 2.

The locations of the genes are fixed throughout the simulation. The locations of the environment and the phenotype change dynamically according to the following set of rules.

1. The phenotype is attracted to the genes. Each gene exerts an attractive force on the phenotype in an amount determined by Rules 2 and 4.

2. The relative attraction of the genes depends on the location of the environment. The attraction that each gene exerts on the phenotype is inversely related to the distance between that gene and the environment. As the location of the environment changes, therefore, it has the effect of “turning on” genes close to it. The attraction of phenotype to genotype is illustrated in Figure 11.1B.

3. The phenotype is attracted to the environment. The current location of the environment exerts an attractive force on the phenotype in an amount determined by Rule 4. The attraction of phenotype to environment is illustrated in Figure 11.1C.

4. At each moment in the simulation, the attraction of the phenotype to the genes and to the environment are stipulated to be equal. This rule is important because it eliminates traditional “How much?” questions from the analysis. At each moment during development, genes and environment exert precisely equal forces on phenotype.

5. The environment is attracted to the current location of the phenotype, as illustrated in Figure 11.1D. Note the contrast between Scarr’s model of G → E and the current model, which posits P → E. This distinction is elaborated in the Discussion section of this chapter. For the first quarter of the simulation, the location of the environment cannot change. This condition is referred to as “childhood.” (This rule is included for completeness, but it is not investigated in this chapter. See Turkheimer & Gottesman, 1996.)
For each simulated organism, the phenotype begins at the center of the space. The locations of the genes and the first location of the environment are under control of the experimenter or can be specified at random. When the simulation begins, genes and environment exert their attractions on the phenotype and on each other, and the locations of the phenotype and the environment begin to change. Phenotype and environment each trace a complex path that represents changes in the organism's environment and its phenotype in the course of its life, which consists of 300 iterations. Most of the time, the paths eventually settle down close to each other in a region of space close to one of the genes. An example of the paths followed by phenotype and environment for a particular configuration of genotype and environment is illustrated in Figures 11.2A and 11.2B.

Although many aspects of the simulation are under the control of the experimenter (e.g., the number of genes in the space, the relative force of the attraction of genes and environment, the rate at which the environment can change in response to changes in the phenotype), in this chapter, most will remain fixed at intermediate values. The simulated experiments that follow investigate the relationship among two predictors (the initial location of the environment and the average location of the genes) and one outcome, the final location of the phenotype, which is defined as the mean on each dimension of the last 50 iterations.

**Results**

**Experiment 1: Reaction Norm With Cloned Organisms**

The disagreement between scientists comfortable with linear and additive models of behavioral variability and developmentalists who insist on concrete models of actual developmental processes has crystallized in the discussion of one of the most fundamental tools of genetic analysis, the reaction norm.
(Gottlieb, 1991; Platt & Sanislow, 1988; Turkheimer & Gottesman, 1991). Although reaction norms are nothing more than tabulations or plots of variation in phenotypic outcome as a joint function of genotype and environment, different scientific disciplines have evolved very disparate methods for estimating them. To construct a reaction norm for a trait in lower animals, an animal experimentalist can use a “clones in cages” design: Obtain samples of cloned organisms representing several possible genotypes within a species and raise representatives of each strain in each of a controlled variety of environments. For the experimentalist to whom such research designs are available, no further statistical analysis would be required: One would simply tabulate the phenotypic outcome for each combination of genotype and environment with no expectation that the resulting surface would be linear or even continuous. The experimental developmentalist does not even have to assume that variation in genotypes or environments could be ordered along an axis from “worst” to “best.” The goal, after all, is to discover how genotypes and environments combine to produce a phenotype (and to make a point about variability of outcome within genotypes), not to generate statistically based models for predicting phenotype from genotype and environment.

Few of these methodological niceties are available to the social scientist, who must do without cloned organisms (except for MZ twins) or any possibility of “assigning,” much less randomly, genotypes to environments. The methodology of behavioral genetics encompasses the alternative quasi-experimental methods that have evolved. With such methods, phenotypic outcome can be plotted as a function of naturally occurring variation in genotype and environment. The regression methods employed are almost always linear and usually additive, so the reaction norms that result generally consist of straight parallel lines. Behavioral geneticists can point out that such linear models, despite their obvious etiological inadequacies, often do a pretty good job of predicting phenotypic outcome from its genetic and environmental antecedents. Developmentalists are appalled by the very smoothness of behavioral-genetic reaction norms, however: Bronfenbrenner and Ceci (1994, p. 571) described one of my own renderings of a reaction norm (Turkheimer & Gottesman, 1991) as resembling “a bent piece of chicken wire that quickly straightens out to become horizontal,” a criticism I will readily accept from any behavioral scientist who has never employed linear multiple regression in the analysis of data arising from a complex developmental process. The experimentalist Gottlieb probably qualifies; Bronfenbrenner and Ceci do not.

In the first investigation of the behavior of the simulated gene–environment system, reaction norms were estimated using techniques available to the experimental animal psychologist, that is, a single genotype was reared in a variety of environments. The genotype illustrated in Figure 11.1 was used in a series of 100 simulations, each with a different environmental starting point. The environmental starting points varied along Dimension 1 of the space. The first simulation was run with environmental starting point of (1, 50), the second had a starting point of (2, 50), and so on through (100, 50). A plot of final phenotype versus the starting point of the environment is the reaction norm for this genotype along Dimension 1.
Figure 11.3 shows the reaction norm that results. The line through the scatterplot is a smoothing spline. Because genotype was held constant across simulations, all variability in this reaction norm is "environmental." Although there is obviously a positive relationship between environmental starting location and phenotypic outcome ($r = .68$), several characteristics of the relationship suggest that it would be difficult to model using ordinary social scientific methods. The relation is clearly nonlinear and seems to include a discontinuity at an environmental starting point of about 50: For environmental starting locations below this value, phenotypic outcomes are usually in the low 40s, whereas for starting locations greater than this value, phenotypic outcomes suddenly jump to around 65. Moreover, throughout the relationship, there are many points that vary from the general trend line in apparently unsystematic ways.

Reaction norms are usually drawn for single genotypes as they vary across rearing environments, but it is just as appropriate to investigate phenotypic outcome for a variety of genotypes raised in a single environment (Turkheimer, Goldsmith, & Gottesman, 1995). For the next simulation, therefore, 100 randomly generated genotypes were reared using a single environmental starting location of (25, 25). A plot of the relation between the mean genotypic value on Dimension 1 and final phenotypic value, shown in Figure 11.4, is the "environmental reaction norm" for the starting location (25, 25). Although the correlation between mean genotypic value and final phenotypic
value ($r = .65$) is roughly the same as the correlation between environmental starting location and phenotypic outcome in the previous reaction norm, examination of the scatterplot reveals that it is much better suited to standard methods of statistical analysis in that it is roughly linear, continuous, and homoscedastic.

**Experiment 2: Analysis of Variance**

Experiment 1 has demonstrated that the simulated system comprises both environmental and genetic variability, that is, phenotypic outcome varies when genotype is held constant and environmental starting location is allowed to vary and when environmental starting location is held constant and genotype is allowed to vary. It will be useful to summarize this result in the form of an analysis of variance. For this experiment, 10 random genotypes were generated, and each genotype was reared using 10 randomly selected starting environmental locations, for a total of 100 simulations. Note that this design uses information that in the real world can only be obtained from clones: Each genotype is raised 10 different times in a variety of environments. The phenotypic variability resulting from this design can be partitioned into two parts, one attributable to variation between the genotypes and the other attributable to variation in environmental starting location within the geno-
Table 11.1. Genetic and Environmental Variance Produced by Experiment 2

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<tr>
<th>Dimension</th>
<th>Genetic Variance</th>
<th>Environmental Variance</th>
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<tbody>
<tr>
<td>1</td>
<td>21067 (50%)</td>
<td>21054 (50%)</td>
</tr>
<tr>
<td>2</td>
<td>18789 (66%)</td>
<td>14586 (44%)</td>
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types. Table 11.1 shows that variability was roughly evenly divided between these two sources, so the “heritability” of phenotype on Dimension 1 (the proportion of total phenotypic variability accounted for by genotype) can be taken to be about .5.

Experiment 3: Random Variation in Genotype and Environment

The experiments described so far would not be feasible in humans. We cannot know how variation in environment would affect the phenotypic outcome of a single human genotype or how genetic variation would affect phenotypic outcome in humans reared in identical environments (except, of course, for identical twins, which will be simulated in Experiment 5). Instead, we must infer the general shape of the relation among genotype, environment, and phenotypic outcome by studying variation in environment across different genotypes, each of which only appears once in the design. Because we cannot hold genotype constant experimentally, we do so statistically by using regression methods to isolate the effects of genotype and environment in populations in which both vary randomly.

To simulate this research design, 100 organisms were generated, each with a random genotype and a random environmental starting location. Once again, final phenotypic outcome was recorded for each outcome and regressed on mean genotype and environmental starting location. The partial regression of phenotypic outcome on environmental starting point is a linear estimate of the first, “genetic,” reaction norm from Experiment 1, and the partial regression of final phenotype on mean genotype is a linear estimate of the second, “environmental,” reaction norm. For genotype, the results of this experiment were similar to the previous two: Figure 11.5 is a scatterplot of the relation between mean genotype and final phenotypic outcome, and as before, it is roughly linear and generally smooth. Except for the environment, the results are very different. As can be seen in Figure 11.6, there is essentially no relationship between the starting location of the environment and phenotypic outcome when genotype is allowed to vary randomly. An analysis of variance showed that 39% of the variability in outcome was accounted for by the mean level of the genotype, whereas none of it was explained by variation in environmental starting location.

How can this be? In Experiments 1 and 2, environment explained as much variation as genotype; in Experiment 3, using precisely the same simulations under different sampling conditions, it explained none at all. The environmental effects in Experiments 1 and 2 were entirely within replicated genotypes, whereas each genotype only appeared once in Experiment 3. It appears that in this simulated system, the effect of the environment is all interaction,
Figure 11.5. Final phenotype plotted as a function of genotype for simulation in which both genotype and starting environment vary randomly.

Figure 11.6. Final phenotype plotted as a function of starting environment for simulation in which both genotype and starting environment vary randomly.
with no main effect. The effect of the environment can be detected within a single genotype, but the shape of the environmental effect depends crucially on the genotype with which it is interacting. Across varying genotypes, the effects of starting environmental location cancel each other out and become undetectable.

*Experiment 4: A Twin Study*

Monozygotic twins occupy a central place in behavioral genetic methodology precisely because they offer the only possibility to replicate the clones in cages design using humans. The replication is very incomplete: Only two “clones” of any particular genotype are usually available, and they cannot be “assigned” to random environments. Nevertheless, the classical twin design is arguably the most successful quasi-experimental research design in the behavioral sciences: Its elegance and the consistency of its results across an astonishing variety of behavioral phenotypes have contributed greatly to the increasing acceptance of behavioral genetics during the last generation.

Nevertheless, the results of twin studies are not without their mysteries. On the one hand, MZ twins are often too similar. Although the biometric model suggests that heritability approaches unity when the similarity between MZ twins is twice as great as that between DZ twins, MZ twins are often more than twice as similar (Lykken, McGue, Tellegen, & Bouchard, 1992). More complex family designs allow such excess similarity to be modeled as multiplicative effects among genes, but like the nonshared environment, genetic nonlinearity is easier to model than it is to specify. On the other hand, it sometimes seems as though identical twins are not similar enough. Schizophrenia, for example, is widely thought to be a genetic disorder of the brain (Liberman & Corrigan, 1992), and no plausible environmental component to its etiology has ever been identified. Yet identical twins are only about 50% concordant for the disorder (Gottesman, 1991). What is the source of the MZ twin discordance?

Stochastic processes in development may be one explanation. The simulations described so far contain no randomness: Phenotypic outcome has been completely determined by the starting configuration and the dynamic rules that describe the behavior of the system. Therefore, two “monozygotic twins reared together,” with identical genotypes and the same environmental starting location, would follow exactly the same course. In order to allow for differences between monozygotic twins, some randomness in development must be included. There are several points in the system at which randomness could be introduced, but the most plausible is in the effect of phenotype on the environment. In the system as it has been described so far, changes in the location of the environment are completely determined by the location of the phenotype. In real organisms, of course, phenotype can influence the environment but not determine it.

The following method was employed to determine the amount of randomness to introduce. A random component was added to the module determining changes in environmental location, so that 90% of the change depended on the location of phenotype and 10% depended on a random variate. One hundred pairs of monozygotic twins were generated with identical randomly chosen
genotypes and environmental starting locations within pairs, phenotypic outcomes were simulated using the new random component, and the correlation in phenotypic outcome between twins was computed. With the random component at 10%, the correlation was .95, suggesting that 10% randomness did not cause much divergence between the twins. The random component was then increased to 20%, and so on until changes in environmental location were 100% random. When changes in environmental location were 50% random, the correlation was reduced to .7, which seemed to be a reasonable representation of the similarity between MZ twins that is commonly observed, so this value was selected for the remainder of the twin study.

Another 100 pairs of MZ twins were generated using the parameters just described, along with 100 pairs of dizygotic (DZ) twins who shared a randomly selected 5 of their 10 genes. Phenotypic outcomes were generated for each twin pair, and correlations were computed for the MZ and DZ twins. Results are given in Table 11.2. For the MZ twins, as before, the correlations on Dimensions 1 and 2 were about .7, in keeping with the specification of the system. In DZ twins, however, the correlations were substantially smaller. Doubling the difference between the MZ and DZ twin correlations resulted in heritabilities on Dimensions 1 and 2 close to .8, whereas the shared environment once again accounted for no variability in phenotype.

Although Experiments 1 and 2 demonstrated that the simulations contain substantial environmental variability, the twin design, like the random family design in Experiment 3, failed to detect it. Why? It appears that the effect of adding randomness to the determination of changes in the environment had a disproportionate effect on the DZ twins. The exact replication of genotype in the MZ twins was sufficient to maintain the similarity of the twins despite dynamic variation arising from interaction with a partially random environment, whereas the 50% genetic dissimilarity of the DZ twins interacted with environmental randomness and resulted in substantially less similarity in phenotype.

**Experiment 5: Indeterminacy in Reaction Norms**

In Experiment 1, we saw that one of the difficulties in interpreting the reaction norms produced by the simulations was that they contained "outliers," or points that deviated from the general trend for no apparent reason. Attempts to understand such outliers represent a major portion of the

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<th>Dimension 1</th>
<th>Dimension 2</th>
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<tr>
<td>MZ</td>
<td>.75</td>
<td>.61</td>
</tr>
<tr>
<td>DZ</td>
<td>.35</td>
<td>.19</td>
</tr>
<tr>
<td>$r^2$</td>
<td>.80</td>
<td>.84</td>
</tr>
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activities of working social scientists. Why do some children raised in brutal poverty overcome their background and go on to successful lives? Why are some MZ twins of schizophrenic patients perfectly normal? A very reasonable scientific impulse is to want to investigate the environmental backgrounds of outlier cases in greater detail. Surely, if we study the upbringing of the successful child of poverty with enough care, we will discover the key factors that can “inoculate” children against the risks presented by chaotic environments.

In the simulations, we can model this process by “zooming in” on our view of the reaction norm and focus attention on the region in which the outliers occur. For example, in Figure 11.3, in the region along Dimension 1 from 10 to 20, most of the phenotypic outcomes are between 40 and 50, except for a few that are considerably lower, close to 10. Suppose the view of the x-axis of Figure 11.3 is expanded so that it runs from 10 to 20 in intervals of 0.1. Figure 11.7 shows the result. The expanded region from 10 to 20 looks very much like the original: Some organisms had a final phenotypic value between 40 and 50, others are close to 10 (and a few previously undetected points close to 25 appear!), but no pattern is discernible. We could then choose to zoom in again, perhaps to the region on Dimension 1 between 12 and 13 with an interval of .01. Figure 11.8 shows the result, which is again similar to the original reaction norm. This process of zooming in can continue indefinitely, as illustrated in Figure 11.9, which shows the reaction norm across a region on Dimension 1 extending only from 12.7 to 12.8 and yet contains as much variability in phenotypic outcome as the original figure.

![Figure 11.7](image)

**Figure 11.7.** Reaction norm from Figure 11.3, with expanded view of region between 10 and 20.
Figure 11.8. Reaction norm from Figure 11.3, with expanded view of region between 12 and 13.00.

Figure 11.9. Reaction norm from Figure 11.3, with expanded view of region between 12.7 and 12.8.
Suppose you were a social scientist assigned to understand the environmental circumstances that dispose this genotype to arrive at final phenotypic values near 10. How much scientific progress could be made, at least in the region of the environment between 10 and 20? No matter how detailed your environmental data became, you would still be unable to determine the exact environmental values producing the specific phenotypic outcome in question. Correlations between environmental variation and phenotypic outcome, as we have seen throughout this series of experiments, would be completely hopeless. It would be wrong, however, to conclude that the environment is impotent in the determination of phenotype, because all of the variability in phenotypic outcome in the reaction norm is environmental by definition. The reaction norms in Figures 11.7, 11.8, and 11.9, remember, illustrate environmental variability within the single genotype illustrated in Figure 11.3. This paradox is at the heart of the difficulty in developing a rigorous science of environmental effects on development: The environment is a necessary component of all developmental processes, and variation in the environment is demonstrably associated with phenotypic variation, but the nature of the covariation renders it invisible to typical social scientific methods.

Discussion

Spinach and Ice Cream

Gregory Bateson (1972) told a story about a mother who, in her eagerness to instill healthful eating habits in her son, reinforced him for eating a helping of spinach by rewarding him with a bowl of ice cream. Bateson asked, Will the boy grow up to love or hate spinach, love or hate ice cream, or love or hate his mother? If the topic of food preference management were ever taken up as a line of research by an enterprising assistant professor, I submit that we already know more or less what would result. If the research were undertaken in a convenience sample of singleton children and their biological mothers who happened to be spinach reinforcees (i.e., without random assignment of families to food behavior groups), modest but statistically significant linear effects of food preference reinforcement on later patterns of eating behavior would be found, leading to an initial wave of enthusiasm for the environmental potency of maternal food reinforcement behavior. When the skeptical behavioral geneticist at another university got wind of these findings and replicated the study in twins or adoptees, however, it would be discovered that most of the purported environmental effect was in fact attributable to genetic transmission of food preferences from mothers to children and that the familial environmental effects of food reinforcement were practically nil. This new finding would be greeted with stoically repressed glee in the behavioral-genetic community, whereas environmentalists would write angry replies about the limitations of twin and adoption methods for the study of behavioral development.
As the anger subsided over a period of years, positions on the matter might coalesce along the lines I have outlined. Nonshared environment theorists would contend that the environment acts to make siblings more different, not more similar, and would undertake the study of differential feeding practices within families, the effects of peer group standards, or birth order. Antisocialization theorists would declare victory, produce lists of reasons why one wouldn’t expect maternal behavior to be a strong determinant of eating behavior in the first place, generate theories of how genetically driven food preferences in children cause food management behavior in their caregivers, and begin linkage and association studies to find the genes responsible for food preference. Developmentalists would declare that the entire research program was meaningless, because the goal of studying the development of eating behavior is to establish the developmental sequence of gene–environment interactions that leads to phenotypic patterns of eating, a goal to which biometric exercises in variance partitioning have little or nothing to contribute.

I submit further that questions like these—they might be called “spinach and ice cream” questions—are paradigmatic of the difficult methodological and interpretive problems faced by behavioral scientists. The hallmark of spinach and ice cream questions is that they induce a conflict between environmentalists, clinicians, and our own intuitions on the one hand and biogenetically oriented scientists on the other. One does not have to be a radical environmentalist to believe that the peculiarities of your parent’s cooking had an effect on your adult food preferences. Right or wrong, introspection suggests that our families were important determinants of our adult selves. If taken seriously, however, the upshot of the last 20 years of biometric research is that the effects of early environmental events, at least to the extent they are shared among siblings, are largely illusory. We believe that our parents’ peculiarities shaped our adult attitudes and those of our siblings, but the antisocialization theorists tell us that we are mistaken. Instead, our adult phenotypes are the product of our genetic endowment, which is why they are generally similar to those of our parents and to environmental events that we did not share with our siblings, which is why children in the same family are as different as they are.

The simulations that I have described in this chapter suggest another possibility, which may offer a way to take behavioral genetics seriously without dismissing wholesale the causal effects of family environments. The simulations were designed so that at each moment, the genetic and environmental forces exerted on phenotype were precisely equal. When environmental variation was studied within a single genotype, unambiguous environmental variability in phenotypic outcome could be observed, but the shape of the relation between environment and phenotype was nonlinear, discontinuous, and not generalizable from one genotype to another. Because the relation between environment and phenotype was inconsistent across different genotypes, when genotype was allowed to vary randomly, it was no longer possible to detect any effect of the environment at all. When each genotype appears only once in the design, as is the case in most human research, the reaction norms relating environment to phenotype within each genotype average each other out, and nothing observable remains.
Why do the developmental dynamics of the simulations obscure the effects of the environment, whereas genetic effects are relatively unaffected? The genetic configuration is fixed at birth. Although developmental changes in the environment have the effect of turning genes on and off during development, the genotype nonetheless exerts a constant and steady force on phenotypic development. A genotype in which most of the genes are located in the upper left of the space will exert a constant pull on the phenotype in that direction, regardless of the other complex dynamics the system produces. The environment, in contrast, is in a constant state of flux induced by its reciprocal interactions with phenotype, which in turn is the joint product of genotype and environment. The starting location of the environment, therefore, is a source of variability in outcome but not a source of systematic variability.

Suppose a clinician interviewed a pair of siblings raised by a spinach-reinforcing mother. “Oh, yes,” one of them might say, crunching a carrot, “Mother always reminded me how important it is to eat well, and that is why I am a vegan today.” The other, with equal conviction and ice cream spoon in hand, might report, “Mother was always so annoying with her peculiar ideas about eating, I finally rebelled and gained 150 pounds.” If the direction of the effect of the mother’s behavior depended crucially on minute phenotypic details of siblings, they both might be correct. Woe to the social scientist who sets out to study the environmental effects of spinach-reinforcement on adult food preferences! The simulations suggest that an attempt to develop a systematic environmental theory would be a frustrating tangle of initial hopes, false leads, small effect sizes, and unreplicated findings, whereas the behavioral geneticist studying the same phenomenon could count on substantial and replicable heritabilities.

The Postbiometric Era Revisited

The possibility that environment is a potent but unsystematic source of variability in behavioral development offers a resolution of the dilemma posed by biometric studies of behavior. It is not that it doesn’t matter how humans are raised, but rather that the effects of variation in rearing practices are fiendishly difficult to specify. Behavioral scientists studying clones in cages have the advantage of wielding experimental control over both genotype and environment, and with these tools, it has proved possible to dampen developmental dynamics to an extent that allows inferences to be drawn about the consequences of specific environmental events. In free-ranging humans, for whom genotype and environment are essentially uncontrollable, not to mention correlated with each other, the dynamic complexities of development overwhelm any systematic consequences of environmental variation.

For nonshared environmentalists, this analysis suggests that a careful distinction must be maintained between the nonshared environmental events that have been the focus of empirical research so far and the nonshared effects of shared environmental events. These constructs are frequently discussed as if they were the same thing. Here, for example, are Plomin, DeFries, and McClearn (1990):
Environmental variance can be decomposed into two components, Ec and Ew. Common or shared environmental influences (Ec) are those that make members of a family similar to each other; the remainder of the environmental variance, the portion not shared by family members is called within, independent, or nonshared (Ew). . . . Given that environmental variance is important but shared environment is not, critical environmental influences must be of the nonshared variety, making children in the same family as different from one another as are pairs of children selected randomly from the population. The importance of this finding lies in the fact that much previous environmental research has been misguided: Environmental factors relevant to differences in behavioral development lie not between families but within families. (pp. 249–250, emphasis in original)

According to this definition, if parental divorce served to make children in the same family more different, would divorce count as shared or nonshared environment? In the first part of the paragraph, Plomin seems to want to count anything that makes children different as nonshared. In an important sense, however, divorce is shared among siblings regardless of whether it ends up making them more similar or more different, and researchers have generally taken Plomin's subsequent assertion that “critical environmental influences must be of the nonshared variety” (Plomin, DeFries, et al., 1990, p. 249) to mean that causal factors operating within families should be the focus of future research efforts. As a result, research undertaken under the banner of nonshared environment has focused on the systematic effects of nonshared causes, such as differential parental treatment and the effects of peer groups, to the exclusion of the nonshared effects of shared causes such as divorce, which are dismissed as a “misguided” focus of old-fashioned environmentalism.

Our meta-analyses (Turkheimer & Waldron, 2000) have offered little reason for optimism that nonshared environmental events will go very far toward answering Plomin and Daniels' (1987) seminal question about why children in the same family are so different. The range of possible explanations of within-family sibling differences will be greatly expanded if we can include the differential effects of the large domain of environmental events that are (usually) shared among siblings in a family, including everything from socioeconomic status to marital discord and divorce. The query, “What are the effects of divorce on children?” is a spinach and ice cream question. The effect of divorce on a child may depend nonlinearly on the phenotype of the child at the time of the divorce, leading to differential effects on siblings experiencing essentially the same environmental event. This leads to an empirical prediction: Children experiencing potent environmental events should show greater variability in developmental outcomes, and pairs of siblings should be more different from each other, even if no systematic effects on the mean can be observed.

The spinach and ice cream explanation provides some support for the developmentalists' insistence that partitioning variance is one thing and explaining the development of behavior another. It is hard to see how biometric family studies of the simulations would ever lead to an understanding of the dynamic rules that generated them. Indeed, unless they were interpreted very carefully, biometric studies might mislead us into concluding that the environment played no causal role in the simulations. The simulations also
remind us, high-level theorizing about development notwithstanding, that individual differences in genotype are associated with individual differences in behavior. Too often, developmentalists allow their legitimate concerns about the limited etiological consequences of population genetics to justify an unwarranted acceptance of old-fashioned environmentalism. In the simulations, as in the field, when the scientific goal is to predict behavior, as opposed to understanding it, knowledge about the genotype is still the best kind of information to have (Meehl, 1962).

The antisocialization theorists have taken the opposite tack, maintaining that because normal family environment is not a useful predictor of behavior, it is not a important cause of behavior. Plomin (1994) cited Grilo and Pogue-Geile:

Experiences that are shared among family members do not play an important role in determining individual differences in weight, fatness, and obesity... experiences that are not shared among family members comprise most of the environmental influence on weight and obesity.... The conclusion that experiences that are shared among family members count for little in determining individual difference in weight, and perhaps obesity, necessitates a drastic rethinking of many current environmental etiological theories of weight. (1991, p. 534)

The spinach and ice cream explanation suggests that this inference may not be correct. It may be that "experiences that are shared among family members" do count for something in determining individual differences in weight but nonetheless serve to make siblings different rather than similar. This is no mere semantic distinction, because the antisocialization theorists' conflation of small variance components with weak causal effects has provided the evidentiary basis for the theorists' dismissal of the causal efficacy of shared family environment and thus to environmentalists' impressions that they are being asked to swallow the contention that normal families have no effect on their children.

In this light, it is instructive to contrast Scarr's model (Scarr & McCartney, 1983) of the role of environment in development with the model that is instantiated in the simulations. Scarr stated simply that G -> E, and to the extent she means that genotype exerts some distal influence on the environment, she is certainly correct. The "genes," as the developmentalists remind us, cannot "influence" an organism's environment in any direct way. What they can do is interact with the environment in a way that sets off a complex chain of interactions that eventuates in environmental change. Although the simulations have demonstrated that the complex dynamics of such a system can preserve linear relations between genotype and outcome, it is hardly the case that G -> E directly, and indeed there is no direct G -> E attraction programmed into the simulation. In the simulations, genetically identical organisms born into different environments show variation in phenotypic outcome, but the unpredictability of environmental variation across genotypes obscures environmental effects when clones are not employed. By analogy to humans, our intuition that we would have turned out differently had we been born into a different family may be correct, even though effects of shared environments cannot be detected by biometric designs.
What does determine environmental change in a niche-picking organism? In a child with a genetic propensity for high intelligence, for example, it is not the child’s genes per se, but rather the child’s behavior, that is to say its phenotype, that evokes stimulating verbal productions by caregivers. Phenotype, not genotype, produces changes in the environment, and phenotype at any moment in development is the cumulative result of an organism’s developmental history, encompassing genotype, environment, and all the complexities of their epigenetic interactions. Replacing Scarr’s theory of $G \rightarrow E$ with a $P \rightarrow E$ theory of development would maintain Scarr’s concern with the role of organisms in the construction of their own environments while allowing for much greater indeterminacy and environmental variability in behavioral outcome.

**Why Environmental Social Science Is So Difficult**

By offering a solution to the problem posed by the second law of behavioral genetics, the spinach and ice cream explanation relieves the postbiometric positions on nature and nurture of their original reason for existence: the need to explain, or explain away, the mysterious failure of shared family environment to contribute to biometric analyses of behavior. The rejection of the claim that normal shared family environment plays no role in the development of behavioral variability, however, simply replaces a substantive implausibility with a methodological quandary. If the effects of the shared environment consist of complex developmental interactions with genotype and phenotype, interactions so complex as to be in principle unpredictable, how can they be studied? A domain of environmental causation that was theoretically undeniable but empirically intractable would be a poor consolation prize for environmentalists. If the meat and potatoes of socialization science—correlations between family characteristics and children’s development—is doomed to failure when appropriate genetic controls are employed, how is the empirical environmentalist to proceed?

A first step is to realize that spinach and ice cream questions are ill-specified and unanswerable, and moreover they concede a methodological advantage to behavioral genetic rivals. Divorce doesn’t have an effect on children: It has a multitude of effects, and the choice among them will be little clarified by correlation matrices of variables describing characteristics of parents and offspring (Wachs, 1993a). Population-based behavioral genetic models of marital status may not elucidate the developmental processes of marital discord and their various effects on children either, but they at least have the advantage of preserving reliable linear relations between the latent variable called “genotype” and behavioral outcome, which allows behavioral geneticists to assert with confidence the developmental banality that genes “influence” behavior.

Theories that attempt to predict behavior from environmental first principles will fail if they neglect the distal but reliable effects of genotype; genetic theories will remain etiologically empty unless they include the complex developmental dynamics of genotype and environment. The goal of an environmentalist theory of behavior, then, is first to catalog the range of outcomes
that are possible within a behavioral domain and then to specify the phenotype by environmental interactions that make some outcomes more likely than others for individual subjects. Of course, doing justice to phenotype by environment interactions would mean taking seriously the role of genotype in generating phenotypes, so the resulting theories would no longer be strictly "environmental," but that is precisely the point. Partitioning environmental and genetic variability has served a useful purpose, but that purpose has now been served.

**Nonlinear Models for Nonlinear Processes**

One expectation based on the outcome of the simulations is that environment may be a better predictor of behavior in some regions of the reaction norm than in others. In Figure 11.3, for example, although the reaction norm may not be well described by a linear model and may even be formally unpredictable in some regions, there are also regions where reasonable generalizations can be made. Below the environmental threshold at about 50 on Dimension 1, the mean phenotypic outcome is about 43; above the threshold, it is about 63. Note that this mean difference in outcome does not translate into a correlation between environment and outcome on either side of the threshold, as would be expected if the overall reaction norm were linear.

Exactly this phenomenon is observed in adoption studies of intelligence (Turkheimer, 1991). Correlations between the intelligence of adopted children and characteristics of their adoptive homes are typically close to zero, in accordance with the second law of behavioral genetics. Paradoxically, when the intelligence of children adopted into middle-class homes is compared with the intelligence of children adopted into poor homes or to their unadopted siblings, a substantial mean difference is generally observed. How can these findings be reconciled? Not with linear models, which require the relation between environment and outcome to be constant across all levels of environment. There appears to be a nonlinearity, even a threshold, in the reaction norm for intelligence, such that highly deprived—not good enough—environments exert an effect on intelligence compared with middle-class homes, whereas variation within middle-class homes does not. Many contemporary regression methods have completely abandoned the requirement of linearity (Hastie & Tibshirani, 1990) and would thus appear to be especially well suited for work in the softer social sciences, but they have yet to be widely applied. The lines through the reaction norms in this chapter were generated by such methods, and they generalize nicely to multivariate models.

Another class of modern regression methods, referred to as classification and regression trees (CART; Breiman, Friedman, Olshen, & Stone, 1984), capitalizes on the characteristic of the simulations that makes them most intractable for linear models: the fact that it is possible to predict outcome in some regions of the environmental prediction space but not in others. CART models work by making predictions sequentially rather than on the basis of weighted linear combinations of predictors. The estimation program seeks the cutoff score on a predictor variable that does the best job of classifying
observations according to outcome. It divides the sample into two groups on the basis of this cut score and then seeks the optimum cutoff within each of the two resulting groups, proceeding in this way until no further improvement in prediction can be made.

The Return of Interactionism

Similarities between the nature–nurture debate and the person–situation debate in social psychology have not been sufficiently explored since the publication of Rowe's (1987) proposal for cross-fertilization between fields. As Rowe observed, the two debates are remarkably similar in structure: In both, an internal mechanism to explain behavior (genotype or traits) is pitted against an external mechanism (environment or the situation). Rowe, however, whose primary concern involved the contributions behavioral genetics might make to the person–situation debate, did not emphasize a striking difference: The histories of the debates are reversed. In the nature–nurture debate, an environmental establishment was challenged by a new experimental paradigm (behavioral genetics) that succeeded in demonstrating powerful internal determinants of behavior. In the person–situation debate, a personological establishment was challenged by a new experimental paradigm (situationism) that succeeded in demonstrating potent external determinants of behavior.

It is worth pondering why the course of the debates have been so different. The person–situation debate has taken place largely in the laboratories of social and personality psychologists. In this setting, as in the animal studies of the developmentalists, the environment is under the control of the experimenter rather than the participant. Another characteristic of laboratory studies is that outcome is measured over a much shorter time span: Does the presumably “honest” subject pick up the $5 bill when she believes no one is watching? Human behavioral genetics, in contrast, usually examines uncontrolled behavior as it accrues over a lifetime. It appears that systematic effects of the environment are easier to detect in controlled settings and relatively short periods of time.

In any case, in both debates it became increasingly apparent that from a causal as opposed to a variance partitioning perspective, neither internal nor external determinants of behavior could be expected to prevail over the other and that from the point of view of explaining the genesis of behavior, the very attempt to separate them is a fool's errand. Both fields have thus been led to the conclusion that internal and external mechanisms of behavior necessarily interact etiologically and statistically in the determination of behavior. Interactionism was fashionable in social psychology in the 1970s (Endler & Magnusson, 1976), fell out of favor for a while, and has recently been resuscitated by a series of important theoretical and empirical papers by Mischel and Shoda (1995). These studies, which cannot be described in detail here, demonstrate that consistency in behavior is not explained by personality or by situation but by interaction between the two: Individual consistency in behavior comprises characteristic patterns of responding to variation in environments.
The reader is to be forgiven a groan as yet another nature–nurture discussion concludes with a call for the integration of genes and environment in the science of behavior. One cannot emphasize enough how difficult it is to specify the interaction of phenotypes and environments in the genesis of behavior. Indeed, I am not convinced that the usual well-intentioned endorsements of increased scientific collaboration between geneticists and environmentalists will bear fruit (Rowe & Waldman, 1993; Wachs, 1993b). The goal of the simulations presented in this chapter has been to offer a concrete explanation of why human developmental science is so difficult, that is, it is the result of the scientifically unfortunate but humanistically pleasing convergence of two factors: the complex dynamic genesis of behavior and the impossibility of exerting meaningful experimental control over the genes or environments of humans.

Traditional approaches to human development have circumvented this dilemma by simplifying the empirical domain in ways that made them easy targets for their opponents, who had of course simply chosen a different mode of simplification. Environmentalists, as behavioral geneticists never tire of pointing out, ignored genetic pathways between parents and children; behavioral geneticists, as developmentalists never tire of pointing out, ignored the interactive ontogenetics of behavior; and developmentalists (as traditional environmentalists might point out but have not, apparently content to encourage skepticism about behavioral genetics wherever it takes root) ignored the dynamic complexity of human behavior by limiting themselves to experimentally controlled work in lower animals. Thus is completed the perfect rhetorical circle that has kept the nature–nurture debate spinning for the past century.

References


