How People Make Their Own Environments: A Theory of Genotype → Environment Effects

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SCARR, SANDRA, and MCCARTNEY, KATHLEEN. *How People Make Their Own Environments: A Theory of Genotype → Environment Effects.* CHILD DEVELOPMENT, 1983, **54**, 424–435. We propose a theory of development in which experience is directed by genotypes. Genotypic differences are proposed to affect phenotypic differences, both directly and through experience, via 3 kinds of genotype → environment effects: a passive kind, through environments provided by biologically related parents; an evocative kind, through responses elicited by individuals from others; and an active kind, through the selection of different environments by different people. The theory adapts the 3 kinds of genotype-environment correlations proposed by Plomin, DeFries, and Loehlin in a developmental model that is used to explain results from studies of deprivation, intervention, twins, and families.

Introduction

Theories of behavioral development have ranged from genetic determinism to naive environmentalism. Neither of these radical views nor interactionism has adequately explained the process of development or the role of experience in development. In this paper we propose a theory of environmental effects on human development that emphasizes the role of the genotype in determining not only which environments are experienced by individuals but also which environments individuals seek for themselves. To show how this theory addresses the process of development, the theory is used to account for seemingly anomalous findings for deprivation, adoption, twin, and intervention studies.

For the species, we claim that human experience and its effects on development depend primarily on the evolved nature of the human genome. In evolutionary theory the two essential concepts are selection and variation. Through selection the human genome has evolved to program human development. Phenotypic variation is the raw material on which selection works. Genetic variation must be associated with phenotypic variation, or there could be no evolution. It follows from evolutionary theory that individual differences depend in part on genotypic differences. We argue that genetic differences prompt differences in which environments are experienced and what effects they may have. In this view, the genotype, in both its species specificity and its individual variability, largely determines environmental effects on development, because the genotype determines the organism's responsiveness to environmental opportunities.

A theory of behavioral development must explain the origin of new psychological structures. Because there is no evidence that new adaptations can arise out of the environment without maturational changes in the organism, genotypes must be the source of new structures.

Maturational sequence is controlled primarily by the genetic program for development. As Gottlieb (1976) said, there is evidence for a role of environment in (1) maintaining existing structures and in (2) elaborating existing structures; however, there is no evidence that the environment has a role in (3) inducing new structures. In development, new adaptations or structures cannot arise out of experience per se.

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The most widely accepted theories of development are vague about how new structures arise; for example, Piaget (1980) fails to make the connection between organism and environment clear in his references to interaction. Nor is development well described by maturation alone (see Connolly & Prechtl, 1981). Neither Gesell and Ilg (1943) nor contemporary nativists (e.g., Chomsky, 1980) appreciate the inextricable links of nature and nurture in a hierachically organized system of development.

We suggest that the problem of new structures in development has been extraordinarily difficult because of a false parallel between genotype and environment, which, we argue, are not constructs at the same level of analysis. The dichotomy of nature and nurture has always been a bad one, not only for the oft-cited reasons that both are required for development, but because a false parallel arises between the two. We propose that development is indeed the result of nature *and* nurture but that genes drive experience. Genes are components in a system that organizes the organism to experience its world. The organism's abilities to experience the world change with development and are individually variable. A good theory of the environment can only be one in which experience is guided by genotypes that both push and restrain experiences.

Behavioral development depends on both a genetic program and a suitable environment for the expression of the human, species-typical program for development. Differences among people can arise from both genetic and environmental differences, but the process by which differences arise is better described as genotype → environment effects. Like Chomsky and Fodor (1980), we propose that the genotype is the driving force behind development, because, we argue, it is the discriminator of what environments are actually experienced. The genotype determines the *responsiveness* of the person to those environmental opportunities. Unlike Chomsky and Fodor, we do not think that development is precoded in the genes and merely emerges with maturation. Rather, we stress the role of the genotype in determining which environments are actually experienced and what effects they have on the developing person.

We distinguish here between environments to which a person is exposed and environments that are actively experienced or "grasped" by the person. As we all know, the relevance of environments changes with development. The toddler who has "caught on" to the idea that things have names and who demands the names for everything is experiencing a fundamentally different verbal environment from what she experienced before, even though her parents talked to her extensively in infancy. The young adolescent who played baseball with the boy next door and now finds herself hopelessly in love with him is experiencing her friend's companionship in a new way.

A model of genotypes and environments.—Figure 1 presents our model of behavioral development. In this model, the child's phenotype (P_c), or observable characteristics, is a function of both the child's genotype (G_c) and her rearing environment (E_c). There will be little disagreement on this. The parents' genotypes (G_p) determine the child's genotype, which in turn influences the child's phenotype. Again, there should be little controversy over this point. As in most developmental theories, transactions occur between the organism and the environment; here they are described by the correlation between phenotype and rearing environment. In most models, however, the source of this correlation is ambiguous. In this model, both the child's phenotype and rearing environment are influenced by the child's genotype. Because the child's genotype influences both the phenotype and the rearing environment, their correlation is a function of the genotype. The genotype is *conceptually prior* to both the phenotype and the rearing environment.

It is an unconventional shorthand to suggest that the child's genotype can directly affect the rearing environment. What we want

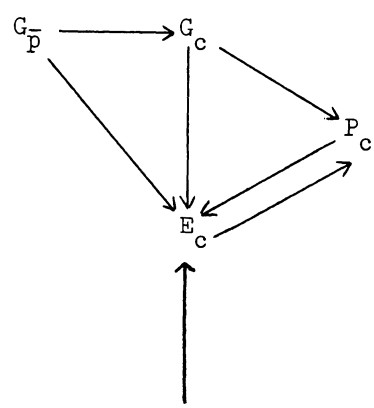

FIG. 1.—A model of behavioral development

to represent is developmental changes in the genetic program that prompt new experiences, before the full phenotype is developed. An example could be found in the development of productive speech; the child becomes attentive to the language environment receptively months before real words are produced. Our argument is that changes in what is "turned on" in the genotype affect an emerging phenotype both directly through maturation (G_c to P_c) and through prompting new experiences.

The model could just as well specify intermediate phenotypes, such as receptive language in the example of productive speech, but the *idea* that genetic differences (both developmental changes for an individual over time and differences among individuals) affect experiential differences could be lost in a web of path diagrams. The model is designed to present our ideas, not for analysis of variance.

Also clouded by an endless regress of intermediate phenotypes would be the idea that the correlation or transaction between phenotype and environment is determined by developmental changes in the genotype. We recognize that this is not a popular position, but we propose it to account for data to be discussed in the final sections of the paper.

Thus, we intend the path from G_c to E_c to represent the idea that developmental changes in phenotypes are prompted both by changes in the effective genotype and by changes in the salience of environments, which are then correlated.

The path from the G_c to P_c represents maturation, which is controlled primarily by the genetic program. New structures arise out of maturation, from genotype to phenotype. Behavioral development is elaborated and maintained, in Gottlieb's sense, by the transactions of phenotype and environment, but it cannot arise de novo from this interaction. Thus, in this model, the course of development is a function of genetically controlled maturational sequences, although the rate of maturation can be affected by some environmental circumstances, such as the effects of nutrition on physical growth (Watson & Lowrey, 1967). Behavioral examples include cultural differences in rates of development through the sequence of cognitive stages described by Piaget and other theoretical sequences (see Nerlove & Snipper, 1981).

Separation of genetic and environmental effects on development.—The major problem with attempts to separate environmental from genetic effects and their combinations is that people evoke and select their own environments to a great extent. There may appear to be arbitary events of fate, such as being hit by a truck (did you look carefully in both directions?), falling ill (genetic differences in susceptibility, or a life-style that lowers resistance to disease?), but even these may not be entirely divorced from personal characteristics that have some genetic variability. Please understand that we do not mean that one's environmental fate is *entirely* determined by one's genotype—only that some genotypes are more likely to receive and select certain environments than others. A theory that stresses either genetic or environmental differences per se cannot account for the processes by which people come to be the way they are. At any one point in time, behavioral differences may be analyzed into variances that can be attributed more or less to genetic and environmental sources (see Plomin, DeFries, & Loehlin, 1977; Scarr & Kidd, in press). A quantitative genetic approach to estimating variances, however, does not attempt to specify the processes by which individuals developed their phenotypes.

Genotype-environment correlations.—Plomin et al. (1977) have described a model of phenotype variation that estimates the amount of variance that arises from genetic and environmental differences. Genotype-environment correlation is a nonlinear component in the additive variance model, included to account for situations in which "genotypes are selectively exposed to different environments." They did not intend to describe developmental processes, as we are doing here. Rather, Plomin and his colleagues were responding to the question, How much of the variation in a phenotype is due to differences among genotypes, differences among environments, dominance effects, genotype-environment interactions, and genotype-environment correlations? Their model addresses sources of individual differences in a population of phenotypes at one point in time. By contrast, our use of the term, genotype → environment effects, is to describe developmental *processes* over time, not to estimate sources of variance in phenotypes. We seek to answer the questions, How do genotypes and environments *combine* to produce human development? and How do genetic and environmental differences *combine* to produce variation in development?

An Evolving Theory of Behavioral Development

Plomin et al. (1977) described three kinds of genotype-environment correlations that we believe form the basis for a developmental theory. The theory of genotype → environment effects we propose has three propositions:

1. The process by which children develop is best described by three kinds of genotype → environment effects: a *passive* kind, whereby the genetically related parents provide a rearing environment that is correlated with the genotype of the child (sometimes positively and sometimes negatively); an *evocative* kind, whereby the child receives responses from others that are influenced by his genotype; and an *active* kind that represents the child's selective attention to and learning from aspects of his environment that are influenced by his genotype and indirectly correlated with those of his biological relatives.

2. The relative importance of the three kinds of genotype → environment effects changes with development. The influence of the passive kind declines from infancy to adolescence, and the importance of the active kind increases over the same period.

3. The degree to which experience is influenced by individual genotypes increases with development and with the shift from passive to active genotype → environment effects, as individuals select their own experiences.

The first, *passive* genotype → environment effects arise in biologically related families and render all of the research literature on parent-child socialization uninterpretable. Because parents provide both genes and environments for their biological offspring, the child's environment is necessarily correlated with her genes, because her genes are correlated with her parents' genes, and the parents' genes are correlated with the rearing environment they provide. It is impossible to know what about the parents' rearing environment for the child determines what about the child's behavior, because of the confounding effect of genetic transmission of the same characteristics from parent to child. Not only can we not interpret the direction of effects in parent-child interaction, as Bell (1968) argued, we also cannot interpret the *cause* of those effects in biologically related families.

An example of a positive kind of passive genotype-environment correlation can be found in reading; parents who read well and enjoy reading are likely to provide their children with books; thus, the children are more likely to be skilled readers who enjoy reading, both for genetic and environmental reasons. The children's rearing environment is positively correlated with the parents' genotypes and therefore with the children's genotypes as well.

An example of a negative passive genotype-environment correlation can also be found in reading. Parents who are skilled readers, faced with a child who is not learning to read well, may provide a more enriched reading environment for that child than for another who acquires reading skills quickly. The more enriched environment for the less able child represents a negative genotype → environment effect (see also Plomin et al., 1977). There is, thus, an unreliable, but not random, connection between genotypes and environments when parents provide the opportunities for experience.

The second kind of genotype → environment effect is called evocative because it represents the different responses that different genotypes evoke from the social and physical environments. Responses to the person further shape development in ways that correlate with the genotype. Examples of such evocative effects can be found in the research of Lytton (1980), the theory of Escalona (1968), and the review of Maccoby (1980). It is quite likely that smiley, active babies receive more social stimulation than sober, passive infants. In the intellectual area, cooperative, attentive preschoolers receive more pleasant and instructional interactions from the adults around them than uncooperative, distractible children. Individual differences in responses evoked can also be found in the physical world; for example, people who are skillful at electronics receive feedback of a sort very different from those who fail consistently at such tasks.

The third kind of genotype → environment effect is the active, niche-picking or niche-building sort. People seek out environments they find compatible and stimulating. We all select from the surrounding environment some aspects to which to respond, learn about, or ignore. Our selections are correlated with motivational, personality, and intellectual aspects of our genotypes. The active genotype → environment effect, we argue, is the most powerful connection between people and their environments and the most direct expression of the genotype in experience.

Examples of active genotype → environment effects can be found in the selective efforts of individuals in sports, scholarship, relationships—in life. Once experiences occur, they naturally lead to further experiences. We agree that phenotypes are elaborated and maintained by environments, but the impetus for the experience comes, we argue, from the genotype.

Developmental changes in genotype → environment effects.—The second proposition is that the relative importance of the three kinds of genotype → environment effects changes over development from infancy to adolescence. In infancy much of the environment that reaches the child is provided by adults. When those adults are genetically related to the child, the environment they provide in general is positively related to their own characteristics and their own genotypes. Although infants are active in structuring their experiences by selectively attending to what is offered, they cannot do as much seeking out and niche-building as older children; thus, passive genotype → environment effects are more important for infants and young children than they are for older children, who can extend their experiences beyond the family's influences and create their own environments to a much greater extent. Thus, the effects of passive genotype → environment effects wane when the child has many extrafamilial opportunities.

In addition, parents can provide environments that are negatively related to the child's genotype, as illustrated earlier in teaching reading. Although parents' genotypes usually affect the environment they provide for their biological offspring, it is sometimes positive and sometimes negative and therefore not as direct a product of the young child's genotype as later environments will be. Thus, as stated in proposition 3, genotype → environment effects increase with development, as active replace passive forms. Genotype → environment effects of the evocative sort persist throughout life, as we elicit responses from others based on many personal, genotype-related characteristics from appearance to personality and intellect. Those responses from others reinforce and extend the directions our development has taken. High intelligence and adaptive skills in children from very disadvantaged backgrounds, for example, evoke approval and support from school personnel who might otherwise despair of the child's chances in life (Garmezy, Note 1). In adulthood, personality and intellectual differences evoke different responses in others. Similarities in personal characteristics evoke similar responses from others, as shown in the case of identical twins reared apart (Bouchard, Note 2). These findings are also consistent with the third proposition.

A probabilistic model.—The concept of genotype → environment effects is emphasized in this emerging theory for three major reasons: the model results in a testable set of hypotheses for which disconfirmation would come from random association between genotypes and environments, it describes a developmental process, and it implies a *probabilistic* connection between a person and the environment. It is more likely that people with certain genotypes will receive certain kinds of parenting, evoke certain responses from others, and select certain aspects from the available environments; but nothing is rigidly determined. The idea of genetic differences, on the other hand, has seemed to imply to many that the person's developmental fate was preordained without regard to experience. This is absurd. By invoking the idea of genotype → environment effects, we hope to emphasize a probabilistic connection between genotypes and their environments. Although mismatches between the behaviors of parents and children certainly exist (see Nelson, 1973), we argue that on the average there are correlations of parents' characteristics and the rearing environment they provide.

Waddington (1962) postulated a probable but not determinant connection between genotypes and phenotypes through an epigenetic space, in which environmental events deflect the course of the developing phenotype. Figure 2 illustrates Waddington's theory of the probable relationship between genotypic and phenotypic differences. Note that a correlation remains between genotype and phenotype, even though one cannot specify in advance what environmental events will affect phenotypic development. To this conception, we add that genotypes shape many of their own experiences through evocative and active genotype → environment correlations.

The Role of the Environment Revisited

If genotypes are the driving force behind development and the determinants of what environments are experienced, does this mean that environments themselves have no effects? Clearly, environments are necessary for development and have effects on the average levels of development, but they may or may

not cause variations among individuals (McCall, 1981). We argue like McCall that nature has not left essential human development at the mercy of experiences that may or may not be encountered; rather, the only necessary experiences are ones that are generally available to the species. Differences in experience per se, therefore, cannot be the major cause of variation among individuals. The major features of human development are programmed genetically and require experiences that are encountered by the vast majority of humankind in the course of living. Phenotypic variation among individuals relies on experiential differences that are determined by genetic differences rather than on differences among environmental effects that occur randomly.

Imposed environments.—In developmental studies, we usually think of environments provided for a child, such as parental interaction, school curricula, and various experimental manipulations. In some cases there are passive and evocative genotype-environment correlations that go unrecognized, as in parent-child interaction and the selection of children into school curricula. In a few cases there may be no correlation of the child's genotype with the treatment afforded an experimental group of which she is a member. On the other hand, it is impossible to ignore the attention and learning characteristics the child brings to the situation, so that the effects of environmental manipulations are never entirely free of individual differences in genotypes. Development is not necessarily constrained by genotype-environment correlations, although most often genotypes and environments are correlated in the real world, so that in fact, if not in principle, there are such constraints.

Sometimes, the influence of genotypes on environments is diminished through unusual positive or negative interventions, so that the environments experienced are less driven by genotypes and may even be negatively related to genotypes, as in the passive, familial situation. Examples of this effect can be found in studies of deprivation, adoption, and day care. Studies of children reared in isolation (Clarke & Clarke, 1976) and children reared in unstimulating institutions (Dennis & Najarian, 1951; Hunt, 1961, 1980) have demonstrated the adverse effects of deprived environments on many aspects of development. Such studies usually address average responses to these poor environments. In any case, studies of environments that are so extreme as to be outside of the normal range of rearing environments for the species have few implications for environmental variation that the vast majority of human children experience.

In contrast to the extremely poor environments in the deprivation literature, the adoption studies include only rearing environments in the range of adequate to very good. The evidence from studies of biologically related and adoptive families that vary in socioeconomic status from working to upper middle class is that most people experience what Scarr and Weinberg (1978) have called "functionally-equivalent" environments. That is, the large array of individual differences among children and late adolescents adopted in infancy were not related to differences among their family environments—the same array of environmental differences that were and usually are associated with behavioral differences among children born to such families (Scarr, 1981; Scarr & Kidd, in press; Scarr & Weinberg, 1976, 1977, 1978). On the average, however, adopted children profit from their enriched environments, and they score above average on IQ and school achievement tests and on measures of personal adjustment.

Negative genotype-environment correlations.—Environments provided to children that are negatively related to their genotypes can

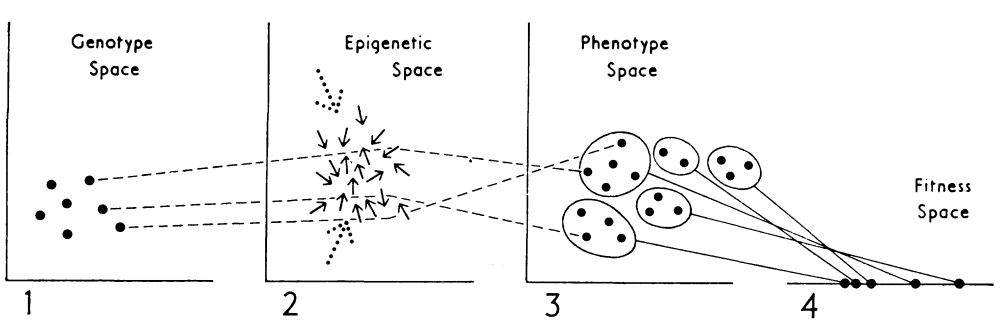

FIG. 2.—Waddington's epigenetic space

have dramatic effects on average levels of development. Extrafamilial interventions that provide unusual enrichments or deprivations can alter the developmental levels of children from those that would be predicted by their family backgrounds and estimated genotypes. Intervention theories predict these main effects (Caldwell & Richmond, 1968; Hunt, 1980).

Enriched day-care environments have been shown to enhance intellectual development of children from disadvantaged backgrounds (Ramey & Haskins, 1981; McCartney, Note 3). Similarly, less stimulating day-care environments can hamper children's intellectual and social development, even if they come from more advantaged families (McCartney, Scarr, Phillips, Grajek, & Schwarz, 1981; McCartney, Note 3).

These are, however, rather rare opportunities, or lack of same, providing negatively correlated experiences for genotypes. In the usual course of development beyond early childhood, individuals select and evoke experiences that are directly influenced by their genotypes and therefore positively correlated with their own phenotypic characteristics.

Environmental effects on averages versus individuals.—One must distinguish environmental events that on the average enhance or delay development for all children from those that account for *variation* among children. There can be "main effects" that account for variation among groups that are naturally or experimentally treated in different ways. Within the groups of children there still remain enormous individual differences, some of which arise in response to the treatment. It is rare that the variation *between* groups approaches the magnitude of differences *within* groups, as represented in the pervasive overlapping distributions of scores. In developmental psychology, we have usually been satisfied if the treatment observed or implemented produced a statistically reliable difference between groups, but we have rarely examined the sources of differential responsiveness within the groups.

Most often, the same treatments that alter the average performance of a group seem to have similar effects on most members of the group. Otherwise, we would find a great deal of variance in genotype-environment interactions; that is, what's sauce for the goose would be poison for the gander. For the kinds of deprivation or interventions studied most often in developmental psychology, the main effects seem not to change the rank orders of children affected. The main effects are real, but they are also small by comparison to the range of individual variation within groups so treated or not. Some children may be more responsive than others to the treatment, but we doubt that there are many situations in which disordinal interactions are the rule. Very few children lose developmental points by participating in Headstart or gain by being severely neglected in infancy. The search for aptitude-treatment interactions (Cronbach & Snow, 1977) and genotype-environment interactions (Erlenmeyer-Kimling, 1972) have not produced dramatic or reliable results.

In studies of adoptive and biologically related families, the correlation of children's IQ scores with the educational level of biological parents is about .35, whether or not the parents rear their children (Scarr & Weinberg, in this issue). Adopted children on the average have higher IQ scores than their biological parents as a result of the influence of their above-average adoptive parents. Taken together, these findings support the claim that treatments can have main effects without overcoming genetic differences in children's responsiveness to those environments. Adopted children have IQ scores above those of their biological parents, yet the *correlations* of adopted children are higher with their biological than adoptive parents (Scarr & Weinberg, 1977, 1978, in this issue). The average effects of treatments, such as adoption, seem to increase the mean IQ scores, but they do not seem to affect the rank order of the children's scores with respect to their biological parents, and it is on rank orders, not means, that correlations depend. These results imply that the effect of adoptive families is to increase the scores of adopted children above those which would be predicted by their biological parents, but not to alter radically the rank order of individual differences among children they rear. And so it is, we think, with most treatments.

Answering Questions from Previous Research on Twins and Families

Neither extreme genetic determinism nor naive environmentalism can account for seemingly anomalous findings from research on twins and families. Three puzzling questions remain, the first of which concerns the *process* by which monozygotic (MZ) twins come to be more similar than dizygotic (DZ) twins, and biological siblings more similar than

adopted siblings on all measurable characteristics, at least by the end of adolescence (Scarr & Weinberg, 1978). The second question concerns the declining similarities between DZ twins and adopted siblings from infancy to adolescence. The third question arises from the unexpected similarities between identical twins reared in different homes.

A theory of genotype-environment correlation can account for these findings by pointing to the degree of genetic resemblance and the degree of similarity in the environments that would be experienced by the co-twins and sibs.

Genetic resemblance determines environmental similarity.—The expected degree of environmental similarity for a pair of relatives can be thought of as the product of a person's own genotype → environment path and the genetic correlation of the pair. Figure 3 presents a model of the relationship between genotypes and environments for pairs of relatives who vary in genetic relatedness. G_1 and G_2 symbolize the two genotypes, E_1 and E_2 their respective environments. The similarity in the two environments (path a) is the product of the coefficient of each genotype with its own environment (path x) and the genetic correlation of the pair (path b). On the assumption that individuals' environments are equally influenced by their own genotypes, the similarity in the environments of two individuals becomes a function of their genetic correlation.

This model can be used to answer question 1 concerning the process by which MZ twins come to be more similar than DZ twins and biological siblings more similar than adopted siblings. For identical twins, for whom $b = 1.00$, the relationship of one twin's environment with the other's genotype is the same as the correlation of the twin's environment with her own genotype. Thus, one would certainly predict what is often observed: that the hobbies, food preferences, choices of friends, academic achievements, and so forth of the MZ twins are very similar (Scarr & Carter-Saltzman, 1980). Kamin (1974) proposed that all of this environmental similarity is imposed on MZ co-twins because they look so much alike. Theories of genetic resemblance do not speak to how close resemblances arise. We propose that the home environments provided by the parents, the responses that the co-twins evoke from others, and the active choices they make in their environments lead to striking similarities through genotypically determined correlations in their learning histories.

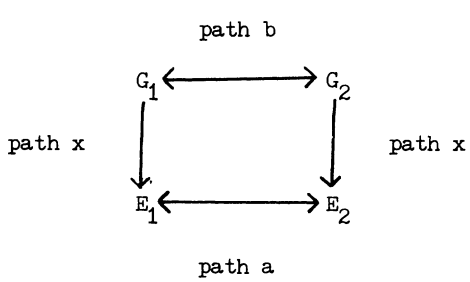

FIG. 3.—A model of environmental similarity based on genetic resemblance.

The same explanation applies, of course, to the greater resemblance of biological than adopted siblings. The environment of one biological sib is correlated to the genotype of the other as one-half the coefficient of the sibling's environment to her own genotype, because $b = 0.50$, as described in Figure 3. The same is true for DZ twins. There is a very small genetic correlation for intelligence between adopted siblings in most studies that arises from selective placement of the offspring of similar mothers in the same adoptive home. More important for this theory, however, is the selective placement of adopted children to match the intellectual characteristics of the adoptive parents. This practice allows adoptive parents to create a positive, passive genotype-environment correlation for their adopted children in early childhood, when the theory asserts that this kind of correlation is most important. In fact, the selective placement estimates from studies by Scarr and Weinberg (1977) can account for most of the resemblance between adoptive parents and their children. In addition, adoptive parents, like their biological counterparts, can provide negative genotype-environment correlations that assure that their several children will not differ too much on important skills, such as reading.

Changing similarities among siblings.—The second question left unanswered by previous research concerned the declining similarities of dizygotic twins and adopted siblings from infancy to adolescence. It is clear from Matheny, Wilson, Dolan, and Krantz's (1981) longitudinal study of MZ and DZ twins that the DZ correlations for intelligence of .60–.75 are higher than genetic theory would predict in infancy and early childhood. For school age and older twins, DZ correlations were the usual .55. Similarly, the intelligence correlations of a sample of

late adolescent adopted siblings were zero, compared to the .25–.39 correlations of the samples of adopted children in early to middle childhood (Scarr & Weinberg, 1978).

Neither environmental nor genetic theories can effectively address these data. How can it be that the longer you live with someone, the less like them you become? One could evoke some ad hoc environmental theory about sibling relationships becoming more competitive, or "deidentified," but that would not account for the continued, moderate intellectual resemblance of biological siblings. Genetic theory has, of course, nothing to say about decreasing twin resemblance or any resemblance among young adoptees.

The theory put forward here predicts that the relative importance of passive versus active genotype-environment correlations changes with age. Recall that passive genotype-environment correlations are created by parents who provide children with both genes and environments, which are then correlated. Certainly in the case of DZ twins, whose prenatal environment was shared and whose earliest years are spent being treated in most of the same ways at the same time by the same parents, the passive genotype → environment effect is greater than that for ordinary sibs. Biological and adopted siblings do not, of course, share the same developmental environments at the same time because they differ in age. The passive genotype-environment correlation still operates for siblings, because they have the same parents, but to a lesser extent than for twins. (See Table 1.)

Monozygotic twin correlations for intellectual competence do not decline when active genotype-environment correlations outweigh the importance of the passive ones, because MZ co-twins typically select highly correlated environments anyway. Dizygotic pairs, on the other hand, are no more genetically related than sibs, so that as the intense similarity of their early home environments gives way to their own choices, they select environments that are less similar than their previous environments and about as similar as those of ordinary sibs.

Adopted sibs, on the other hand, move from an early environment, in which mother may have produced similarity, to environments of their own choosing. Because their genotypes are hardly correlated at all, neither are their chosen environmental niches. Thus, by late adolescence, adopted siblings do not resemble each other in intelligence, personality, interests, or other phenotypic characteristics (Grotevant, Scarr, & Weinberg, 1977; Scarr, Webber, Weinberg, & Wittig, 1981; Scarr & Weinberg, 1978).

Biological siblings' early environments, like those of adopted children, lead to trait similarity as a result of passive genotype → environmental effects. As biological siblings move into the larger world and begin to make active choices, their niches remain moderately correlated because their genotypes remain moderately correlated. There is no marked shift in intellectual resemblance of biological sibs as the process of active genotype → environment influence replaces the passive one.

Identical twins reared apart.—The third question concerned the unexpected degree of resemblance between identical twins reared mostly apart. With the theory of genotype→ environment effects, their resemblance is not surprising. Given opportunities to attend selectively to and choose from varied opportunities, identical genotypes are expected to make similar choices. They are also expected to evoke similar responses from others and from their physical environments. The fact that they were reared in different homes and different communities is not important; differences in their development could arise only if the experiential opportunities of one or both were very restricted, so that similar choices could not have been made. According to previous

TABLE 1

THE SIMILARITY OF CO-TWIN'S AND SIBLING'S GENOTYPES AND ENVIRONMENTS DUE TO:

		CORRELATIONS IN THE ENVIRONMENTS OF RELATED PAIRS	
	GENETIC CORRELATION	Passive Genotype → Environment Effects in Early Development	Active Genotype → Environment Effects in Early Development
MZ twins	1.00	High	High
DZ twins	.52	High	Moderate
Biological siblings	.52	Moderate	Moderate
Adopted siblings	.01	Moderate	Low

studies (Juel-Nielsen, 1980; Newman, Freeman, & Holzinger, 1937; Shields, 1962) and the recent research of Bouchard and colleagues at the University of Minnesota (Bouchard, Note 2), the most dissimilar pairs of MZs reared apart are those in which one was severely restricted in environmental opportunity. Extreme deprivation or unusual enrichment can diminish the influence of genotype and environment and therefore lessen the resemblance of identical twins reared apart.

Research Strategies

The theory we propose can be tested in several ways and prove unable to account for results. First, studies of parental treatment of more than one child would be informative about passive genotype → environment effects. In general, we expect the rearing environment provided for the children in a family to differ in ways that are related to each child's characteristics. Do parents treat all of their children alike, as so many studies of one child per family seem to imply? Can parents be authoritative with one child and permissive with another? Our theory predicts that parents will respond to individual differences in their children, in keeping with Lytton's (1980) research on families with twins. If parent treatment of their children is not related to children's talents, interests, and personalities, the theory is wrong.

Second, studies of responses that individuals evoke from others would test our ideas about evocative genotype → environment effects. The social psychology literature on attractiveness (Bersheid & Walster, 1974; Mursteid, 1972), for example, would seem to support our view that some personal characteristics evoke differential responses from others. Similarly, teachers' responses to children with high versus low intelligence, hyperactivity versus acceptable levels of energy, and so forth provide some evidence for our theory. If others do not respond differentially to individual characteristics for which there is genetic variability, then the theory is wrong.

Third, active niche-building is being studied by the Laboratory of Comparative Human Cognition in their naturalistic observations of children's adaptations to problem-solving situations (Cole & The Laboratory of Comparative Human Cognition, Note 4). Our theory predicts that children select and build niches that are correlated with their talents, interests, and personality characteristics. If not, the theory is wrong.

Fourth, longitudinal studies of adopted children, such as the ongoing work of Plomin and colleagues, can provide valuable evidence of the changing influences of family environments on children. The theory predicts that children's characteristics will be more related to characteristics of the adoptive parents and other adopted siblings in earlier than later development. If adopted children are as similar to their adoptive parents and each other in late adolescence as they were in early childhood, that aspect of the theory is wrong.

Fifth, studies of older adolescents and adults who were adopted in infancy and others who were born into their families can provide evidence on the long-term effects of passive genotype → environment effects within families. Both evocative and active kinds of genotype → environmental effects can be traced through the similarities and dissimilarities of the two kinds of siblings.

In these ways, and others, the theory can be tested. It can fail to account for results obtained, or it can account for the diverse results more adequately than other theories. Given the various results of family studies presented in this paper, we believe that its predictions will be fulfilled. At least, we hope it will encourage more developmentalists to study more than one child per family, genetically unrelated families, and individual differences in experience.

Summary

In summary, the theory of genotype → environment correlations proposed here describes the usual course of human development in terms of three kinds of genotype-environment correlations that posit cooperative efforts of the nature-nurture team, directed by the genetic quarterback. Both genes and environments are constituents in the developmental system, but they have different roles. Genes direct the course of human experience, but experiential opportunities are also necessary for development to occur. Individual differences can arise from restrictions in environmental opportunities to experience what the genotype would find compatible. With a rich array of opportunities, however, most differences among people arise from genetically determined differences in the experiences to which they are attracted and which they evoke from their environments.

The theory also accounts for individual differences in responsiveness to environments —differences that are not primarily interac-

tions of genotypes and environments but roughly linear combinations that are better described as genotype-environment correlations. In addition, the theory accounts for seemingly anomalous results from previous research on twins and families.

Most important, the theory addresses the issue of process. Rather than presenting a static view of individual differences through variance allocation, this theory hypothesizes processes by which genotypes and environments combine across development to make us both human and unique.

Reference Notes

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