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Individual Differences and the Canalization of Human Behavior

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Gottlieb (1991) suggests that behavior geneticists and developmental psychologists have underestimated the importance of environmental regulation of species-typical behavior, outlines a theory of how environments and genes interact dynamically as behavior develops, and provides supporting examples from his own and others' laboratories. Although oversimplified explanations of development have occasionally gained currency, we contend that the complexities of genotype–environment relations have been addressed in psychology, with still greater difficulties arising from psychologists' interest in individual differences in behavior. The complexities of studying genetic and environmental determination of the development of individual differences are explored, using intelligence as an example. Finally, we outline a research program in the spirit of Gottlieb's contribution.

> Variety is said to be the spice of life. It is a staple of necessity to geneticists. (Dobzhansky, 1967, p. 42).
>
> Man is not just an overgrown *Drosophila*. (Dobzhansky, 1967, p. 47)

Gottlieb (1991) has provided a thoughtful and provocative set of ideas for developmentally minded psychologists and biologists. Before proceeding to some variations on his heuristic themes, however, we wish to demur on one point: the degree of attention that our developmentalist and behavior genetic colleagues in psychology have directed to the subtleties of the conjoint determination of phenotypes by genes and environments. It is possible to cite only a few of the many available references. The reciprocal necessity of genetic endowment and environmental stimulation (Gottlieb, 1991, pp. 5 and 6), far from being ignored, is perhaps the single most common theme in theoretical treatments of the genetics of development (Loevinger, 1943; McGue, 1989); the gulf between molecular genetics and phenotypic behavior (Gottlieb, 1991, pp. 5 and 7) is widely recognized (Gottesman, 1974; Scarr and Carter-Saltzman, 1982); the genetic limitations of phenotypic malleability have been extensively debated (Angoff, 1988; Jensen, 1973); the complexities of causal interrelations between genotype and environment have been worked out in considerable detail (Plomin, DeFries, & Loehlin, 1977; Scarr & McCartney, 1983); and, finally and most important, the hoary concept of genetic determination (Gottlieb, 1991, p. 6; attributed to "certain scientists" on p. 8) has once and for all been laid to rest (Fuller & Thompson, 1978). Much remains to be said about all of these topics, and we do not doubt that Gottlieb would rightfully find fault with much of what has been written. However, his impression that these issues "have not found their way into the psychological literature" (p. 5) is incorrect.

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Individual Differences and Species-Typical Regularities

The most striking difference between the experiments described by Gottlieb and those more familiar in the behavior genetic community is that the former have been concerned with similarities among individuals, whereas the latter have been concerned with differences among them (Goldsmith, 1988). In the wild, ducklings probably do not display a great deal of variability in responsiveness to the calls of other species: The goal of Gottlieb's experimental work is to explain why all ducklings end up paddling in the same canal. The familiar subjects of investigation in human behavior genetics—intelligence, personality, and psychopathology—are defined in terms of their variation among individuals. Why are some behaviors species-specific while others vary? An answer requires elaboration of a concept mentioned in passing by Gottlieb, the *reaction norm*.

It is true that the reaction norm has sometimes been a source of misplaced genetic primacy. The confusion is in part graphical. Traditionally, reaction norms have been drawn as a series of regressions of phenotype on a dimension of the environment, one regression for each of several genotypes (see Platt and Sanislow, 1988, for examples and criticisms of this presentation). Reaction norms are, properly, response surfaces: They depict the mean phenotypic value for all combinations of genotype and environment (Figure 1). The usual graphic presentation is a contour map of the response surface, with "isogenetic" lines marking the genetic contours of the surface. Reaction norms could just as validly be drawn in reverse, as a series of regressions of phenotype on genotype, one regression for each environment. Depicting them as surfaces emphasizes that reaction norms are simply graphs of the joint effect of genotype and environment. No causal order is implied.

In any case, because reaction norms depict variation in phenotype as a function of genotype and environment, they lead more naturally to an individual-differences perspective on development. A species-specific regularity in behavior has low variability among individuals exposed to the range of genes and environment under study, which is to say that the reaction norm is flat along both the genetic and environmental axes.

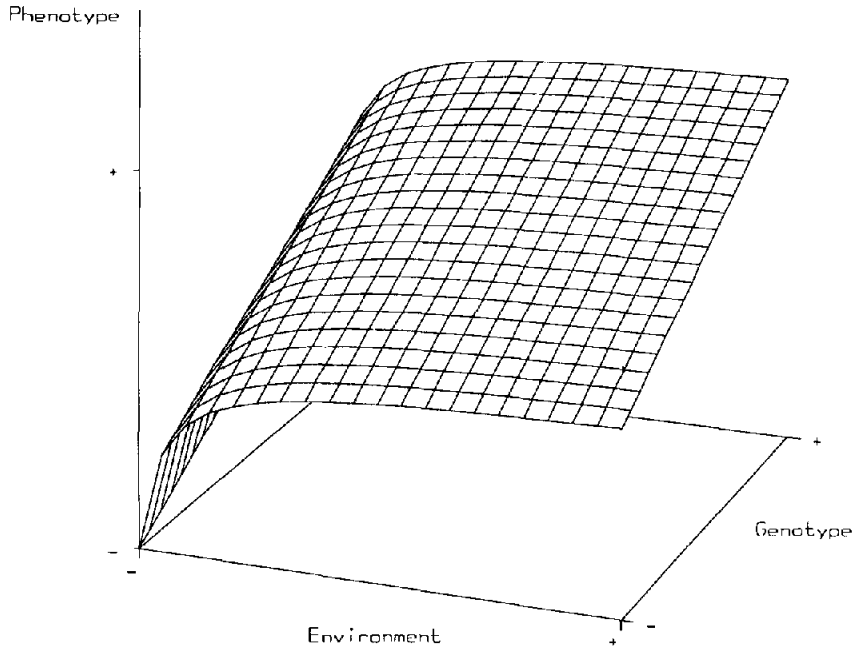

Figure 1. Hypothetical reaction surface for intelligence, showing a positive linear slope along the genetic axis and sharp decline in the range of very poor environments.

Consider the reaction norm for duckling sensitivity to maternal chicken calls. Across a broad range of genotypes and naturally occurring environments, it is relatively flat, as it must be to ensure survival of the ducklings. Gottlieb's (1991) experiments demonstrated that at one extreme of the environmental continuum, where ducklings are completely deprived of embryonic calls, the norm is not flat at all.

Now consider psychometric intelligence in humans. The facts about genetic and environmental influences on intelligence remain controversial, but three findings have been fairly well established: (a) a moderate linear relationship between parental genotype and offspring intelligence, (b) a powerful effect of severely deprived environments, and (c) very small environmental effects in the range of environments provided by intact families. These results suggest that along the environmental axis, the reaction norm for intelligence is flat across a wide range of normal family environments but drops off drastically at the very low end of the environmental continuum, as illustrated in Figure 1.

This description of the reaction norm of intelligence has been taken as evidence for apparently diametric points of view. To the variance-partitioning behavioral geneticist, it is evidence that genotype accounts for a substantial portion of the variance in IQ among children reared in reasonably intact families, whereas environmental differences among families account for almost no variance. From another perspective, however, one might note that along the environmental axis, the description of the reaction norm for intelligence is almost identical to the description of the reaction norm for duckling sensitivity to chicken calls, and one might conclude that "normal family environment" plays a crucial role: Without it, intelligence cannot develop at all, regardless of genotypic potential.

Therefore, the relative importance one attaches to genetic and environmental influences on behavior depends in large part on the universe of genes and environments to which one wishes to generalize. Variation in normal family environment may have little to do with differences in psychometric intelligence but at the same time may be crucial to its development. Both points of view are important. In some contexts, it is perfectly reasonable to ask how individuals in their natural environment come to vary as they do; in others, it is reasonable to ask how they might vary if the environment were to be altered radically.

The distinction between the explanation of variation as it occurs in nature and the explanation of results due to experimental manipulations has been one of the historical themes of the nature–nurture debate in developmental psychology, and it continues to be hotly (if somewhat unnecessarily) debated today. Behavioral geneticists, studying natural variation among twins and adoptive families, have identified few and small effects of environmental differences between families (Plomin & Daniels, 1987; Willerman, 1979); champions of the environment, studying unusual but potent events such as special education programs (Ramey & Haskins, 1981) and adoption out of extremely deprived circumstances (Capron & Duyme, 1989; Schiff & Lewontin, 1986) correctly argue that the environment, if properly controlled, can have an enormous impact (Gottesman, 1968).

Canalization: Stability and Diversity in Development

The notion of canalization extends the reaction norm into a fourth dimension, time. Because four dimensions are difficult to represent graphically, the traditional "landscape" illustration

collapses genetic and environmental effects into a single dimension, leading to much of the confusion that Gottlieb (1991) documents. In Waddington's (1957) often-reproduced illustration, the range of phenotypes is represented along the horizontal axis, with time extending along the length of the scoop. The third dimension, depth of contour, represents the susceptibility of the phenotype to genetic or environmental variation, which is a combination of the first partial derivatives of the reaction norm along the genetic and environmental axes.

Therefore, theories of canalization are about temporal effects on the slopes of reaction norms. Three types of hypotheses are implied by Waddington's illustration:

1. *Stability,* or changes in slope over time. Waddington's landscape is invariably drawn with contours increasing in depth as time passes. It is seldom noted that this represents an empirical hypothesis.

2. *Diversity,* or changes in variability of phenotype over time. Waddington's landscape is wider at the bottom than at the top. This represents another empirical hypothesis, even less widely discussed than stability: Variation between individuals increases with the passage of time.

3. *The causes of stability and diversity.* Gottlieb (1991) has ensured that it will no longer be possible to attribute them to genotype without empirical evidence.

The canalization model implies that individuals get stuck in diverging ruts as they age. Stating the matter baldly makes it clear why the model has always seemed so plausible.

The Canalization of Human Behavior: Intelligence

Whether human behavior is aptly described as canalized is a vast and complex issue, and in the interest of brevity we limit our discussion to intelligence, in part because it is one trait for which some good evidence is available.

Developmental increases in the stability of intelligence are well established, at least through childhood. Evidence comes from several domains. It has been known for some time that correlations between juvenile and adult IQ increase throughout childhood (Anderson, 1939; Honzik, McFarlane & Allen, 1948), as do correlations between child and parent IQ (Honzik, 1957) and between consecutive testings in childhood (Humphreys & Davey, 1988; Wilson, 1987). Whether increases in the stability of intelligence continue throughout adulthood has been studied less intensively, but the trend does seem to continue, albeit at a decelerated pace (Schuerger & Witt, 1989). Note that the stability of intelligence throughout the life span is conceptually separate from changes in its mean. Mean ability increases throughout childhood, remains stable in adulthood, and declines (to a controversial degree) in old age (Botwinick, 1977). The stability of intelligence depends on its predictability across time within individuals and is independent of changes in the mean.

The diversity of intelligence also appears to increase with development, in keeping with the canalization model. McArdle has used latent-growth models to demonstrate increasing interindividual variability in Wechsler Intelligence Scale for Children scores between ages 6 and 11 (McArdle, 1988) and in Wechsler Adult Intelligence Scale Verbal (but not Performance) scores throughout the life span (McArdle & Horn, cited in Horn, 1988).

The Causes of Canalization in Humans

Investigation into the causes of canalization is even more complex than documentation of its existence. Two orthogonal pairs of causal possibilities can be delineated. The first distinction, of course, is between genetic and environmental causation. As Gottlieb (1991) points out (and as has been widely recognized in psychology, under the rubric of *gene–environment interaction*), the choice between them is rarely, if ever, mutually exclusive, because almost any imaginable phenotype requires the coaction of both. This fact, of course, makes classification of particular instances of canalization as "genetic" or "experiential" a very ambiguous task.

The second distinction concerns the causal model within which genetic and environmental events produce canalization. One possibility is the *epigenetic* model, whereby, as an organism develops, each new phenotype exerts an influence on the next phenotype. Such a mechanism would serve to accumulate variance in the phenotype (diversity) and thus to diminish the marginal impact of each new perturbation to the system (stability). The other model, the *genotype–environment correlation* model, suggests that the development of the phenotype is associated with restrictions in the genetic and environmental variation to which it is subsequently exposed. Gene–environment correlation and its consequences have also been the subject of considerable discussion in psychology (Plomin et al., 1977).

These two sets of possibilities, neither of them mutually exclusive, can be combined to achieve degrees of complexity that would rapidly lead beyond the scope of our brief discussion. Gottlieb's (1991) examples of coaction and "nonlinear" causation are fascinating and, in this instance, outside of the usual confines of (at least our own) psychological discourse. There are, however, some data in the developmental behavior genetic literature that can be brought to bear on both matters.

Genetic and environmental influences on the longitudinal stability of intelligence (as opposed to its mean level at any particular point in time) have received considerable attention. The best-known investigations are those of Wilson (1977, 1978), who demonstrated that the increasing stability of abilities between 6 months and 6 years of age are primarily genetic in origin, thus providing an instance of an assertion of genetic canalization with good empirical support. At age 6 months, monozygotic (MZ; identical) and dizygotic (DZ; fraternal) twin pairs had identical ability correlations of 0.66; by age 6, the MZ correlation had increased to 0.85, whereas the DZ correlation remained essentially the same. These findings have recently been replicated and extended in a meta-analysis by McCartney, Harris, and Bernieri (1990), integrating intraclass correlations between MZ and DZ twins of different ages. Simple biometric modeling suggested that the heritable component of intelligence increases with development, whereas the components attributable to shared and nonshared environment decrease.

Adoption studies of intelligence have produced analogous findings. Children adopted from disadvantaged backgrounds into middle-class homes show increments in intelligence during early childhood that then decrease as they pass through

adolescence (Skodak & Skeels, 1949); correlations between the abilities of adoptees and measures of their adoptive environments decrease throughout childhood and are near zero by adolescence (Scarr & Weinberg, 1978). More sophisticated modeling of stability of intelligence in adoptees has shown that it, too, has a genetic component (Baker, DeFries, & Fulker, 1983; LaBuda, DeFries, Plomin, & Fulker, 1986; Loehlin, Horn, & Willerman, 1989).

How genetic and/or environmental influences on canalization are mediated by epigenetic and genotype–environment correlation models is a highly complex matter that has been addressed more often by theory than by data. Recent advances in developmental behavior genetic modeling (Hertzog & Schaie, 1986; McArdle, 1988) have promised to put such informed speculations on a stronger scientific foundation.

Conclusion

Canalization implies a risky empirical theory about genetic and environmental influences on the development of an organism. By "risky," we mean that for some behaviors in some organisms, it is certainly wrong, which is a good thing (Meehl, 1978). As Gottlieb (1991) pointed out, it has been all too easy, as theories have advanced from simple assertions of genetic or environmental primacy to specifications of their joint mechanism, to rely on empirically empty (but politically expedient) insistence on the necessity for both genes and environment in the determination of behavior. Everyone has always known this is true, even, we suspect, in the heyday of environmentalist–hereditarian acrimony. The question, now as always, is how genes and environment work together (Anastasi, 1958). The answer to this question will be discovered by diligent experimentation (where variation can be controlled) and modeling (where it cannot) across the behavioral and neurobiological spectrum.

We conclude by enumerating some steps in a research program inspired by Gottlieb's (1991) adumbration of the concept of *canalization.* Once again we confine ourselves (mostly) to intelligence in humans.

1. We prefer *experience,* rather than *environment,* as a description of nongenetic influences on behavior. Evidence of the relative unimportance of shared family differences in environment (in the normal range; see above) continues to mount, and "experience" seems a much better descriptor of what is left. The concept of experience has a respectable history in ethology (Uexkull, 1957).

2. Scientific investigation of experience is a daunting problem. Animals, whose experience is perhaps less variegated than that of *Homo sapiens* and on whom experimentation is possible, provide ample possibilities, as a quarter-century of developmental psychobiology has demonstrated. In behavior genetics, human experience is too often an error term (Wachs, 1983), although this state of affairs is easier to lament than to correct. There have recently been some interesting advances on the problem (Daniels, 1986), but much remains to be done.

3. The prototypical disagreement between geneticists and environmentalists—whether normal family environment is immaterial or crucial to the development of ability—will not be resolved until real progress is made in the empirical estimation of reaction norms or, for that matter, of developmental landscapes. Somewhere between barely supportive family environment and very poor institutions, the reaction norm for intelligence drops off drastically, as many studies, from Skodak and Skeels (1949) to the recent French adoption studies (Schiff & Lewontin, 1986), have demonstrated. The precise environmental dimensions that are responsible, and where along them the crucial events occur, are unknown. The gulf between the clarity of Gottlieb's (1991) experiments and the century-old confusion in the human realm speaks volumes about the difficulties of making strong inferences on the basis of nonexperimental research in humans.

4. As for canalization proper, greater specification of the predictions of the model are required, and in this case the need seems to be as great in the experimental animal realm. "Experiential canalization" means more than "experience affects behavior." Canalization implies specific types of effects on the development of behavior. We gather from Gottlieb (1991) that experience affects behavior in all sorts of interesting ways. Which of these are examples of canalization and which are not? Can experience affect behavior without canalizing it?

5. As we have already mentioned, canalization represents an extremely interesting subject for modelers (thirsty for higher-order interactions and epigenesis) to model. Genetic and environmental influences on the stability of development are already being studied (Loehlin et al., 1989), although these investigations are just beginning to proceed from quantifications of genetic and environmental contributions to specification of their mechanisms. Behavior can become more stable as a result of autoregressive effects from phenotype to phenotype, because of reductions in environmental and genetic variance that may themselves be related to phenotype, or because of some combination of the two. Developmental changes in the diversity of behavior present even greater challenges. If organisms seek out suitable experiences as they develop, and if genetic expression depends on prior experience, the correlation between genotype and environment will increase with development, with a concomitant increase in diversity. Only our belief in the possibilities for sophisticated modeling driven by carefully specified theory prevents our being daunted by the complexity of these problems.

6. Although we have not attempted to do so here, canalization may also be applied to the other traditional areas of investigation of human individual differences. A model predicting divergent, increasingly buffered phenotypes shows intuitive promise for personality development; models capitalizing on accumulating effects of gene–environment correlation seem well suited for psychopathology.

Theories about development, like development itself, are epigenetic in that each new theory is (or should be) influenced by those that have come before it. As a result, theories about development become more complex as they evolve, but only bad theories (e.g., environmentalism or hereditarianism) become canalized. One may hope that more sophisticated theories of development do not become stubbornly isolated from one another as they evolve, or, worse, increasingly buffered from change. Canalization, as it is confirmed for some organisms in some situations and refuted elsewhere, promises to provide researchers with a flexible, yet empirically rigorous, theoretical framework. Ideally, it could serve to resolve the conflicts

within theories of nature versus nurture, experimental versus correlational, and animal versus human, rather than selecting between them.

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