

.....

Commentary

Eric Turkheimer^a, H. Hill Goldsmith^b, Irving I. Gottesman^a

^a University of Virginia, Charlottesville, Va., and

^b University of Wisconsin, Madison, Wisc., USA

We find ourselves in an unusual position of rhetorical asymmetry regarding Gottlieb's assault on the validity of our chosen field of study – behavioral genetics. We admire Gottlieb's empirical work, feel no need to 'impugn' it, and do not see it as incompatible with our own. We agree with many of the theoretical premises advanced by Gottlieb and recognize them as long-established principles of behavior genetics. We acknowledge some of the limitations of the current state of our methods and findings but do not see how they would lead a reasonable critic to the conclusion that the study of behavior genetics is without value to the fields of developmental psychology or biology. Our discomfort is increased by the fact that we have already replied to many of the points Gottlieb has advanced here [Turkheimer and Gottesman, 1991; Goldsmith, 1994]. Evidently, our earlier efforts to find common ground were unsuccessful. At the risk of repeating ourselves, we make a further effort by examining Gottlieb's current expression of discontent.

Throughout Gottlieb's exposition, we are uncertain whether he wishes to impugn the very practice of behavior genetics, in the ideology-as-science manner of long-time critics [Hirsch, 1981; Lewontin et al., 1984], or to make a more limited argument about the extensibility of otherwise valuable population-genetic methods to questions concerning the causal mechanisms of behavioral development in individual organisms, in the spirit of his quotation from Bronfenbrenner and Ceci [1994]. In fact, Gottlieb often seems to wish to imply the former while arguing the latter. As we have little interest in resurrecting nature versus nurture cold wars [Plomin and McClearn, 1993], we focus on Gottlieb's most specific concerns, which are as follows:

(1) Findings of substantial heritability based on genetic epidemiological methods (twin, family, and adoption studies) do not necessarily imply knowledge of the causal mechanisms underlying development, because the gap between gene expression and phenotypic behavior is very wide, and the path across it stochastic and nonlinear.

(2) The concept of reaction range [Gottesman, 1963, 1974; Turkheimer and Gottesman, 1991; Gottesman and Goldsmith, 1994] is an oversimplification of the more fundamental concept of the reaction norm [Dobzhansky, 1955]. Reaction ranges imply genetic determinism; reaction norms are indeterminate.

(3) Hierarchical models from general systems theory provide a useful way to conceptualize the complexity of pathways between genotype and phenotype.

We are struck by the absence of fundamental disagreement between Gottlieb's theses and what we take to be the mainstream view of contemporary behavior genetics. As we will show, points 1 and 3 are uncontroversial, having been recognized since the inception of the field of behavior genetics [see, for example, table 1 in Goldsmith, 1988, and its antecedents – Gottesman, 1974; Goldsmith, 1984]. We are less in agreement with point 2, and as the essence of Gottlieb's concerns are to be found in his discussion of reaction norms and reaction ranges, we begin there, in an attempt to establish a framework for a broader discussion of the implications of behavior genetics for developmental psychology as well as biology.

From Reaction Norms to Reaction Ranges to Reaction Surfaces

The reaction norm and reaction range concepts are not isomorphic. To the extent we have suggested that they were so, we were incorrect. They are, however, intimately related, and the reaction range is not a clandestinely deterministic version of the reaction norm. An exploration of the structural relationships between them illuminates many of the disagreements between Gottlieb and ourselves.

As noted in a previous reply to Gottlieb [Turkheimer and Gottesman, 1991; and subsequently Gottesman and Goldsmith, 1994], a reaction norm is a theoretical entity that can be operationalized as a regression surface in which phenotype is fit to a joint function of genotype and environment, traditionally drawn as a set of isogenetic contours. A regression surface produces an estimated phenotype score for each combination of the predictor variables. Gottesman's [1963] concept of *reaction range* describes the response surface in a slightly different way, in terms of the difference between the minimum and maximum phenotypic values for a given genotype *across a specified range of environments*, and, we might add, at a particular point in time [Dobzhansky, 1955, pp. 156–157]. Reaction norms and reaction ranges can both change throughout development. The introduction of time to the illustration of reaction norms provides a bridge to Waddington's related graphical construction of 'epigenetic landscapes' [Turkheimer and Gottesman, 1991]. Although we do not attempt to do so here, Goldsmith and Gottesman [in press] have illustrated changes in reaction surfaces across development. A reaction range is thus an empirical characteristic of a reaction norm. Before exploring the legitimate complications introduced by this change in point of view, we need to dismiss the chimeras introduced by Gottlieb.

Gottlieb's first concern is that the reaction range is causally unidirectional in a sense derived from Waddington's concept of canalization. According to this view, genotype sets limits – narrow ones – for phenotypic expression, and environment determines where within the genotypic limits a phenotype is expressed. As noted in our reply to Gottlieb's 1991 article, neither the reaction norm nor the reaction range implies any such causal ordering. In fact, awareness of the bidirectionality of genetic and environmental canalization originated with Gottesman [1963] in the paragraph immediately following the one quoted by Gottlieb in his article in this issue:

A useful shibboleth for the reader is the following: Similar genotypes may have different phenotypes, and similar phenotypes may have different genotypes. This should mean that theorists who choose to focus on genetic factors in intelligence will treat stimulus conditions and life history variables

as boundary delimiters for the manifestation of intellectual levels and vice versa for theorists who focus on environmental factors [p. 256, emphasis added].

In his original formulation, Gottesman [1963] chose to describe reaction range as the difference between phenotypic extremes within a genotype, across environments. As is evident in the quoted paragraph, one could just as well compute ranges as the difference between the minimum and maximum values within an environment, across genotypes. We repeat, emphatically: *The reaction norm, and thus the reaction range, is a statistical description of the dependence of phenotype on both genotype and environment. No causal ordering of genotype and environment is implied.*

In our reply to Gottlieb's 1991 article, we suggested that some of the confusion surrounding reaction norms could be eliminated if they were depicted as surfaces rather than as isogenetic contour plots. The concept of a *reaction surface* also helps to clarify the relation between norms and ranges. Figure 1 is a hypothetical reaction surface. Three ranges are illustrated: (1) the difference between the minimum and maximum phenotypic values for a given genotype across environments – the original reaction range described by Gottesman [1963]; (2) the difference between the minimum and maximum values for a single environment across genotypes, to which one might apply the Gibsonian appellation, 'affordance range', and (3) the difference between the minimum and maximum values across both genotypes and environments, which is simply the range of phenotypic expression for the population.

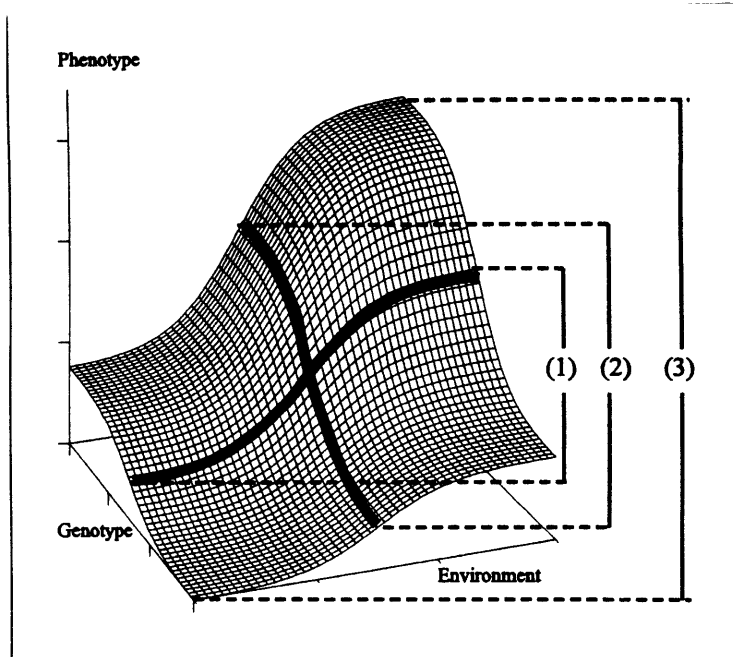
Gottlieb's second concern involves determinacy and indeterminacy in reaction norms and ranges. If, as we have suggested, a reaction range is simply an empirical characteristic of a reaction norm, it seems unlikely that reaction ranges would set 'strict and predictable upper and lower limits', as Gottlieb asserts, whereas reaction norms allow 'no prediction of the preservation of relative differences in phenotype when rearing circumstances are changed'. In fact, the distinction is specious.

Returning once again to Gottesman's [1963] original adumbration of the matter, it is clear that the entire exercise was intended to illustrate a principle rather than set 'strict upper and lower limits' on actual subjects. Reference is made, for example, to 'the possible distribution of IQs in a group of Mongoloids' [p. 255] and 'one of the curves that could be drawn' [p. 256]. It is also apparent that Gottesman was quite aware that his hypothetical curves awaited revision when new environmental circumstances arose: 'As soon as some biochemical intervention becomes feasible for this genetic defect, the curve will have to be modified in an upward direction; this would be analogous to the introduction of insulin as a treatment for diabetes mellitus' [p. 255].

Another way of looking at the determinacy issue is statistical. If reaction norms were estimated from real data, they would have standard errors around them, which would apply equally to their shape (the reaction norm) and to their range (the reaction range). So in point of fact, a reaction norm would never imply 'strict upper and lower limits'. Rather it would imply that subjects would be normally distributed around the regression surface, so that observed phenotypes greater than the maximum of the surface or less than its minimum would simply become less and less likely. It makes no more sense to say that the reaction range 'sets strict upper and lower limits' than it does to say that the reaction norm predicts that an individual with genotype x and environment y will have a phenotype of exactly z .

Gottlieb further asserts that the reaction range concept assumes additivity of genotype and environment, so that 'any phenotypic difference between genotypes would be

Fig. 1. A hypothetical reaction norm depicted as a reaction surface. Three ranges are illustrated: (1) the difference between the minimum and maximum values for a single genotype across environments, or reaction range; (2) the difference between the minimum and maximum values for a single environment across genotypes, for which we have proposed the term 'affordance range', and (3) the total phenotypic range of the organism. [Copyright 1994 by Turkheimer et al.; used with permission.]



preserved in the restricted and favorable environments'. It is not true that Gottesman's original exposition assumes additivity. The contour lines in Gottesman's original figure (fig. 1 in Gottlieb's article) may not intersect, but they are clearly not parallel, and with enough statistical power they would certainly give rise to a significant genotype \times environment interaction. This fact leads to an important point. A reaction range refers to a particular genotype underlying a phenotype, not to the phenotype as a whole. As Gottlieb notes, the ranges of reaction for different genotypes will be the same when the isogenetic contours are parallel. Note, however, that in Gottlieb's figure 1, the reaction range for genotype D is more than four times as wide as the reaction range for genotype A. How, then, does Gottlieb conclude that 'the essence of the conceptual difference between a *norm* of reaction and a *range* of reaction' is that 'the range of reaction predicts the occurrence of parallel phenotypic lines when the different genotypes are subsequently reared in different environments; i.e., the relative differences in phenotype are predicted to be maintained across a variety of novel rearing environments'? It is interesting to note that in Gottlieb's adaptation of Gottesman's figure [Gottlieb, 1992, reproduced as our fig. 2a], he has redrawn the isogenetic contours to be almost perfectly parallel.

By using the phrase 'relative differences', Gottlieb may have wanted to suggest that the reaction-range concept implied that the *ordering* of genotypes was preserved across environments, but that is not the case either. Figure 2b is an illustration of a reaction norm in which the isogenetic lines cross, but a meaningful reaction range can be measured for both of them. Additivity and interaction simply have nothing whatsoever to do with the difference between reaction norms and reaction ranges.

Gottlieb's misconstrual of the relation between reaction ranges and additivity becomes especially apparent in his discussion of the 1958 study by Cooper and Zubek. We find Gottlieb's assertion that this classic study is the 'only ... empirical study in the psychological literature that explicitly addresses the reaction-norm concept', very perplex-

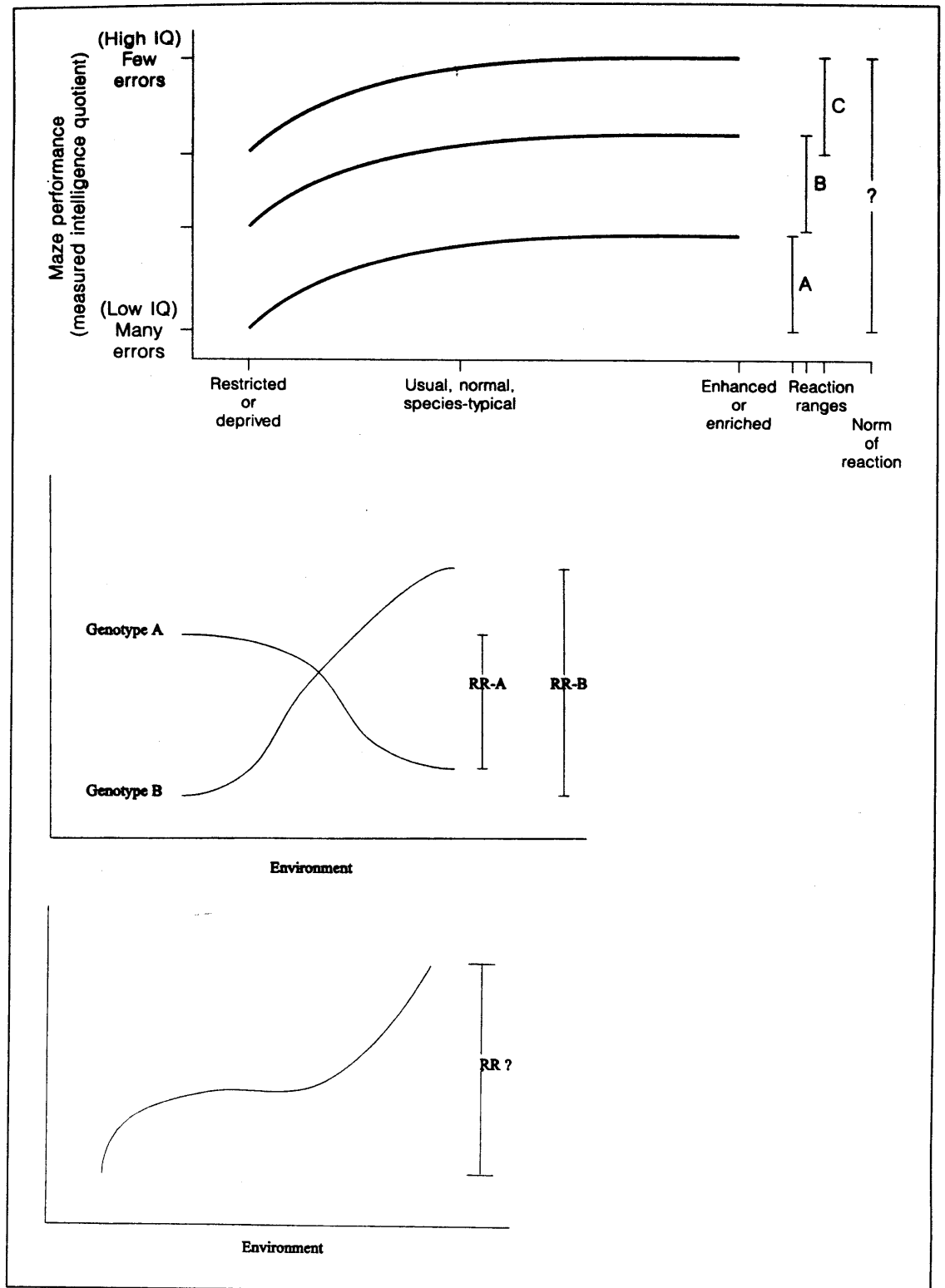


Fig. 2. Three varieties of reaction norms and ranges. **a** Gottlieb's [1992] adaptation of Gottesman's [1963] illustration of the reaction range, in which Gottlieb has redrawn the isogenetic contours to be more nearly parallel. **b** A reaction norm in which the isogenetic contours cross, but still imply reaction ranges. **c** A reaction norm that implies a reaction range that may be difficult to generalize because it is still diverging at the extremes of environment.

ing, and characteristic of his preference for animal work. As we have argued elsewhere [Turkheimer, 1991], all behavior-genetic studies address the reaction norm concept. Why, to cite only one very environmentally oriented example, do the French adoption studies of Schiff and colleagues [Schiff and Lewontin, 1986] not qualify as explorations of the reaction norm for human psychometric intelligence? In any case, the reaction range implied by the right panel of Gottlieb's figure 3 has nothing to do with the fact that the isogenetic lines are not parallel. For each of the illustrated genotypes, the reaction range is simply the difference between the minimum and maximum phenotypic values. So, once again, endorsement of the reaction-range concept does not commit one to preposterous notions like, 'genes coded for learning ability' that Gottlieb introduces, or to the idea that relative differences between genotypes will be preserved in all possible environments. The reaction range for a genotype is simply the observed difference between the phenotypic maximum and minimum in some specified range of environments.

The interpretation and generalizability of a reaction range as a description of a reaction norm will depend on the appearance of the reaction norm and the range of environments that were employed in estimating it. In figure 2c, although it is certainly possible to compute a reaction range as the difference between the minimum and maximum phenotype for each genotype, the shape of the graph warns that the minimum and maximum are still diverging at the extremes of environment under study, leading to the expectation that more extreme environments will increase the reaction range. Therefore, one must pay attention to the phrase, 'for a specified range of environments', when discussing reaction ranges. The same is true of reaction norms, as postulated by Dobzhansky (the first president of the Behavior Genetics Association) in the quote at the end of Gottlieb's article.

Nevertheless, for most phenotypes the reaction norm becomes asymptotic as the environment becomes more extreme. Consider height in humans [Gottesman, 1974]. Nutritional quality is positively correlated with height, and linear models can be fit to the relationship, but it seems obvious that the underlying relationship is nonlinear. Extremely deprived diets do not result in adults who are 18 inches tall, and all the nutrients in the world will not produce 12-foot gargantuans. Although we agree with Gottlieb that one cannot rule out the possibility that someone will one day concoct a nostrum capable of producing gigantism, for the time being it seems reasonable to conclude that the relation between height and nutrition is asymptotic at both ends.

Evolving Approximate Models of Human Behavior

Disputation aside, we are sympathetic with Gottlieb's fundamental concern that overly concrete interpretation of behavior-genetic results could lead a careless thinker into genetic determinism. It is wrongheaded to identify genetic determinism with reaction ranges as opposed to reaction norms, or with the beliefs and practices of mainstream behavior geneticists. Certainly, it is incorrect – and potentially dangerous – to state that 'the reaction range for IQ is x points', because the assertion presumes additivity of genotype and environment, neglects the statistical uncertainty that always surrounds such estimates, and does not make clear that the statement can only be generalized within the range of environments from which it was estimated. Yet simply translating the assertion into a statement about reaction norms does not help matters:

'the reaction norm for IQ has a slope of x IQ points for each y units of the environment', is just as wrong, and for exactly the same reasons.

Safe in the knowledge that Gottlieb will correct us if we interpret his motives incorrectly, we hazard the suggestion that his concern goes deeper than the technical issue of whether the reaction norm or the reaction range is the most appropriate means of representing the influence of genotype and environment on a phenotype. Gottlieb appears troubled, we think, by the scientific approach taken in behavior genetics, an approach that is commonplace in the human behavioral sciences but infrequent in the realms of animal behavior and developmental biology.

Ultimately, the relationship between genotype and phenotype cannot possibly be linear. The phenotypic expression of genes depends interactively on environments, the effects of genes depend interactively on each other (epistasis), the environments provided to offspring are in part a reflection of the offspring's genotype [Plomin and Bergeman, 1991], organisms seek out environments partly on the basis of their genetic endowment [Scarr and McCartney, 1983], and no one can ever explore the complete range of environments to which a particular genotype might be exposed. (Among other problems, to do so with humans would be criminal.) If we could ever know them completely, the relations among genotype, environment, and a high-level behavioral phenotype would be so complex as to daunt even the most gifted of a talented generation of genetic modelers. In the face of all of this complexity, and bearing in mind the intellectual dangers of genetic determinism, what is the point of estimating linear, and often (but not, contra Gottlieb, always), additive models of the distant relationship between genotype and human behavioral phenotypes?

Gottlieb appears to view reaction norms as latent entities that would be better off left that way. Each of an essentially infinite number of possible genotypes can be exposed to a literally infinite number of environments, and the phenotypic results are in some formal sense indeterminate each time. No amount of previous research can rule out the possibility that some new combination of genotype and environment will produce surprising results. Perhaps in animals, for which inbred strains can be produced and for which a limited set of experimentally controlled environments provided, some specification of the reaction norm is possible. But in humans, for whom genotype and environment are seldom quantifiable or controllable, the situation is hopeless. To even attempt to specify a norm of reaction or its corollary, a reaction range, is to deny humans the complex individuality they deserve.

Yet this kind of simplification is common in the many domains of social science in which the objects of study are exceedingly complex and controlled experimentation is not possible. If one is studying the effects of therapist behavior on outcome in psychotherapy, or of maternal expressiveness on the adult attachment of grown children, one chooses linear additive models not out of a belief that the underlying relationships are in fact linear, but simply because they work, albeit rather imperfectly, for some useful scientific and practical purposes. It's all well and good to insist that therapist-client interactions are infinitely more complex than allowed by a researcher's two-by-two analysis of variance, but one would not wish to forestall the development of new treatments while we figure out how to completely specify the psychotherapeutic process, in its practically infinite complexity and interactivity. We are reminded of Meehl's [1962] assertion that the best single predictor of whether an individual will develop schizophrenia is a finding that he or she has an identical twin with the disorder. This fact is useful scientifically, and must be reckoned with theoretically, whether or not we know anything about the devel-

opmental mechanisms that mediate the genetic effect. If knowledge of mechanism were required prior to investigation of relationships between predictor and outcome, how much of behavioral science would be disallowed?

This point suggests that many of the disagreements between Gottlieb and behavior geneticists may reflect the differing assumptions of theoretical biologists and social scientists. If one were running a classroom for economically deprived children who were doing poorly in school, one would want to know about the environmental interventions that are available now to improve classroom performance. A teacher in such a classroom would be nonplussed, we think, by a theoretical biologist who showed up to explain that the range of phenotypic expression of children's learning ability was formally undecidable because the relationships were sure to be nonlinear and one could never specify all the possible learning environments to which the children might be exposed. One might hope that the teacher would be equally unimpressed by a hereditarian who tried to explain that because environmental interventions hadn't worked very well so far, the children were clearly hopeless. The appropriate goal of behavior genetics is to try to find some solid ground between these two opposed, but ironically matched, nihilisms – radical insistence on the indeterminacy of reaction norms on the one hand and rigid genetic determinism on the other.

Gottlieb would prefer an approach based in general systems theory, in which attention would remain focused on the intervening mechanisms between genotype and environment, with bidirectional causal processes going up and down the hierarchy of complexity from the molecular to the molar. Gottlieb seems unaware that models of this type are almost universally endorsed as a methodological heuristic for the social sciences, including behavior genetics, usually based on illustrations similar to his figure 4 [Fuller and Thompson, 1960, fig. 10–2; Engel, 1980, fig. 1; Bateson, 1987, fig. 1; Sabelli and Carlson-Sabelli, 1989, fig. 1; Sing et al., 1992, fig. 1].

Gottlieb may also fail to appreciate how difficult it is to instantiate these ideals. To our knowledge, the most successful application in genetics of the kind of hierarchical causal models Gottlieb endorses are those that have been developed by Sing and his colleagues [1990, 1992, 1993] for the development of coronary heart disease (CHD). Gottesman [1994] has recently championed Sing's methods as a paradigm for work involving the genetics of behavior. In Sing's approach, one follows up identification of a genetic component in the variability of a complex phenotype with a search for the variables – involving lipid metabolism and blood pressure, in this case – that constitute two of the endophenotypic [Gottesman and Shields, 1972] nodes at the next level 'down' in the causal hierarchy. This 'top-down' approach is paired with a 'bottom-up' approach involving study of gene products. From the point of view of classical medical genetics, the complexity of CHD has been the mother of Sing's considerable inventiveness in developing models to study it. It thus does not diminish Sing's accomplishment to point out that human behavioral phenotypes are, if anything, several orders of magnitude more complex than heart disease, and therein lies a difficulty with the hierarchical systems model.

In figure 3a, we have merged Gottlieb's figure 4 with Sing's model of the development of CHD [Sing et al., 1992, fig. 1]. What makes Sing's approach successful is that the bottom-up and the top-down approaches meet in the middle. Looking 'up' from the genes, one finds precursors of lipid metabolism; looking 'down' from CHD, one finds fat accumulating in the arteries. But in the study of human behavior, things rarely work out so well. In figure 3b, we have sketched the nature of the problem for a complex pheno-

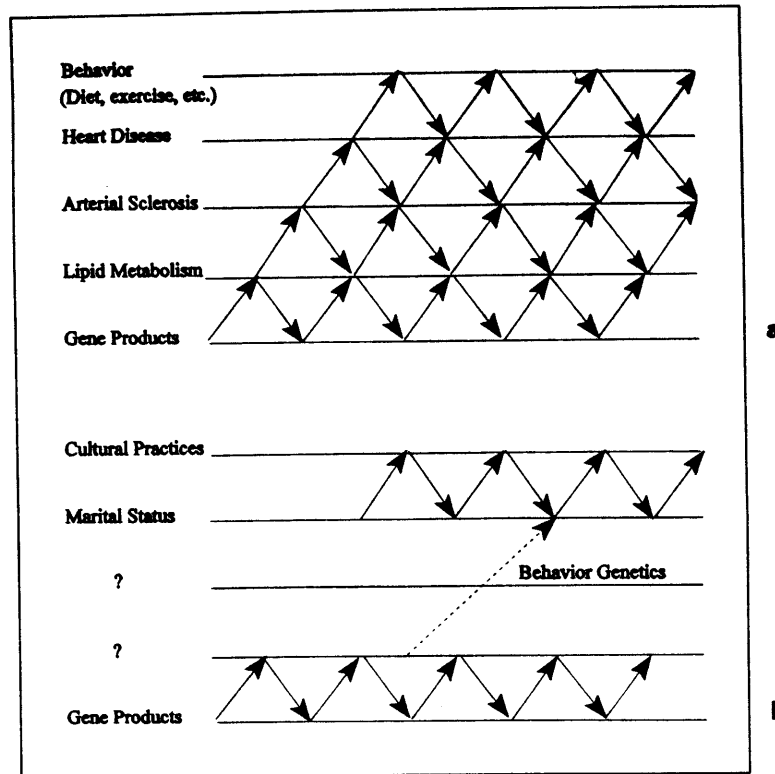


Fig. 3. An adaptation of Gottlieb's figure 4 (this issue), with headings adapted from Sing et al. [1992] (**a**), and another adaptation of Gottlieb's figure 4, illustrating the general ignorance of mechanisms intervening between gene products and marital status (**b**). Behavior genetics provides a means of bridging the gap.

type drawing some recent attention from behavioral geneticists, marital status [McGue and Lykken, 1992; Turkheimer et al., 1992]. What is the 'cholesterol' of divorce? What sort of gene products should we look for to point us in that direction? To say we don't know is to put it politely; we don't know where to look.

But the dotted line we have added to figure 3b represents what we do know as a result of behavior genetics. Twin studies have shown us that – however complex, interactive, and nonlinear the mechanisms may ultimately be – genotype bears some relationship to marital status, a relationship that can at least be modeled as linear. This finding is not without consequences. In light of behavior-genetic results, what would Gottlieb make of traditional divorce research, which observes differences between the children of continuously married couples and divorcees, and *assumes* them to be sociocultural consequences of marital discord and divorce?

This question returns us to the one we posed at the beginning of this commentary. Is Gottlieb arguing that behavior genetics in general does not lead anywhere important, or simply that behavior genetics does not necessarily lead to detailed specification of the developmental mechanisms underlying phenotypes? Presuming he intends the latter, we are perplexed about what to make of the charge that behavior genetics – or any scientific endeavor – is deficient or incomplete. Suppose it were true (and we argue this point shortly) that behavior genetics, while making 'important contributions' [Bronfenbrenner and Ceci, 1994, as quoted by Gottlieb], nevertheless does not lead to insight about mechanisms of individual development. Is it a requirement that scientific paradigms ex-

plain everything? Why not turn the charge around and complain that developmental psychobiology is incomplete because it doesn't have much to say about why some married people get divorced while others don't?

Behavior genetics is at a crossroads. Having established beyond a reasonable doubt that genetic variability is a major component of most of the important objects of study in the social sciences, the field is ready to move on to the next steps in Sing's paradigm – identification of the 'endophenotypes' that underlie behavior at the next lowest level of analysis, and (as the human genome project bears fruit) undertaking the daunting task of tracing gene effects upward toward behavior [McGuffin et al., 1994, Plomin et al., 1994]. A great many theoretical, statistical, and experimental advances will need to be made before this process is completed.

By conceding that the applicability of behavior genetics to questions of individual developmental mechanisms is an issue in need of searching exploration, we do not mean to vindicate the oversimplified and outdated version of the field that Gottlieb advances. Any recent issue of *Behavior Genetics* is sufficient to show it is not the case, as Gottlieb would have it, that 'Population geneticists identify two sources of variation, one described as heritable (genetic), the other nonheritable (environmental).' In fact, during the last decade vigorous methodological expansion of behavior genetics has occurred, motivated by a desire to elucidate more fundamental causal mechanisms and consisting precisely of a fragmentation of the traditional genetic and environmental variance components into a myriad of more specific ones.

We refer to multivariate behavior genetics [Neale and Cardon, 1992] and developmental behavior genetics [Hahn et al., 1990] more than a decade old and now the dominant paradigms in the field, although Gottlieb appears unaware of them. In multivariate and developmental behavioral genetics, the genetic and environmental sources of variation and covariation in multiple phenotypes at one or more points in time are analyzed simultaneously. Multivariate analysis can produce information about the relative degree of heritability among phenotypes, the magnitude of common and unique genetic and environmental variability among phenotypes, or the sources of genetic and environmental stability and change in single or multiple phenotypes across time. The last of these multivariate applications is, of course, of particular interest to developmentalists. The interested reader might wish to explore how multivariate behavior-genetic analyses have been used to investigate childhood traits that are genetically associated with adult psychopathology [Mendlewicz and Hippius, 1992; Kendler et al., 1993], the degree to which continuity of experience versus continuity of genetic effects mediates stability (and change) of individual differences in personality/temperament [McGue et al., 1993], and the degree to which intelligence or temperament is genetically or environmentally associated with educational achievement [Petrill and Thompson, 1993]. As to how effective these methods will eventually be in providing clues to the intervening causal mechanisms between gene products and behavioral phenotypes, the jury is still out. We would welcome a reasoned consideration of these problems, but to insist that behavior geneticists are doing nothing but doubling differences between monozygotic and dizygotic twin correlations is simply uninformed.

The most realistic basis for consensus between behavioral geneticists and their various critics may be found in an attitude of humble pessimism about the difficulty of formulating scientific models of complex human behaviors. Widely endorsed, we have noted, is a 'systems model' of the social sciences, focusing on the specification of intervening mechanisms in the hierarchy of complexity between molecular events and socio-

cultural processes. This is an easy position to take, but what do we expect our eventual model of something as complex as marital status to look like? Can one even imagine a causal model of divorce that respects the complexity and humanity of the phenomenon while preserving the scientific rigor of Sing's models of heart disease?

Behavioral scientists are typically faced with a Hobson's choice between reductionistic models that sacrifice complexity to scientific rigor, and models based in social or clinical science that do the reverse. General endorsements of systems theory notwithstanding, developing concrete methods to overcome this dilemma will require a major paradigm shift, and the difficulty of achieving it is unfairly laid at the doorstep of a single field like behavioral genetics.

Environmentally oriented social scientists, who have so far expended the greatest effort in the task of understanding the factors involved in marital status, have produced many interesting results, but have certainly not produced anything that resembles a general scientific theory. Behavioral geneticists have recently introduced some new clues, but Gottlieb, like earlier behavioral geneticists such as Hirsch [1967], is entirely correct in insisting that discovery of heritability is the beginning of a road, not the end. And although we are not entirely sanguine about the current enthusiasm for molecular genetic approaches to very complex human behaviors (based on our expectation that genetic influences on something like marital status will be radically polygenic), the genome project may one day have something to add, along with evolutionary psychology, psychoanalysis, neuroscience and a host of other specialities residing up and down Gottlieb's hierarchical model.

We conclude with a challenge. How would Gottlieb apply his methodological model to the study of marital status, and how would he include genetic information in the enterprise? Should developmental psychology go back to ignoring genetic effects? Should we adhere to a strictly bottom-up approach until reliable mechanisms can be traced all the way up? Might multivariate behavior genetics inform us about heterogeneous mechanisms of marital failure that could then be studied separately? Would anything be learned by putting divorcees in PET scanners? Or should we concede that something like marital status is too complex to be studied scientifically with available methods and abandon the effort?

Studying important human behaviors scientifically is vexing. The field of behavioral genetics represents an incomplete attempt. Similar limitations apply to psychobiology and to environmentally oriented social science. If everyone were frank about the limitations of their chosen field, we might all be more able to appreciate the valid contributions of others, enabling us to commence work on the prodigious task of formulating a solid scientific basis for the human sciences.

References

- Bateson P (1987). Biological approaches to the study of behavioral development. *International Journal of Behavioral Development*, 10, 1-22.
- Bronfenbrenner U & Ceci SJ (1994). Nature-nurture reconceptualized in a developmental perspective: A bio-ecological model. *Psychological Review*, 101, 568-586.
- Cooper RM & Zubek JP (1958). Effects of enriched and restricted early environments on the learning ability of bright and dull rats. *Canadian Journal of Psychology*, 12, 159-164.
- Dobzhansky T (1955). *Evolution, genetics and man*. New York: Wiley.
- Engel GL (1980). The clinical application of the biosychosocial model. *American Journal of Psychiatry*, 137, 535-544.
- Fuller JL & Thompson WR (1960). *Behavior genetics*. New York: Wiley.
- Goldsmith HH (1984). Continuity of personality: A genetic perspective. In Emde RN & Harmon RJ (Eds.), *The development of attachment and affiliative systems*. New York: Plenum.

- Goldsmith HH (1988). Human developmental behavior genetics: Mapping the effects of genes and environment. *Annals of Child Development*, 5, 187-227.
- Goldsmith HH (1994, winter). The behavior-genetic approach to development and experience: Contexts and constraints. *Newsletter of the Society for Research in Child Development*.
- Goldsmith HH & Gottesman II (in press). Heritable variability and variable heritability in developmental psychopathology. In Lenzenweger MF & Haugaard J (Eds.), *Frontiers of developmental psychopathology*. Oxford: Oxford University Press.
- Gottesman II (1963). Genetic aspects of intelligent behavior. In Ellis N (Ed.), *The handbook of mental deficiency: Psychological theory and research* (pp. 253-296). New York: McGraw-Hill.
- Gottesman II (1974). Developmental genetics and ontogenetic psychology: Overdue detente and propositions from a matchmaker. In Pick A (Ed.), *Minnesota symposium on child psychology* (Vol. 6, pp 55-80). Minneapolis: University of Minnesota Press.
- Gottesman II (1994). Schizophrenia epigenesis: Past, present, and future. *Acta Psychiatrica Scandinavia Supplement*, 90, 384, 26-33.
- Gottesman II & Goldsmith HH (1994). Developmental psychopathology of antisocial behavior: Inserting genes into its ontogenesis and epigenesis. In Nelson CA (Ed.), *Threats to optimal development: Integrating biological, psychological, and social risk factors* (pp. 69-104). Hillsdale NJ: Erlbaum.
- Gottesman II & Shields J (1972). *Schizophrenia and genetics. A twin study vantage point*. New York: Academic Press.
- Gottlieb G (1991). Experiential canalization of behavioral development: Theory. *Developmental Psychology*, 27, 4-13.
- Gottlieb G (1992). *Individual development and evolution: The genesis of novel behavior*. New York: Oxford University Press.
- Hahn ME, Hewitt JK, Henderson ND & Benno RH (Eds.) (1990). *Developmental behavior genetics: Neural, biometrical and evolutionary approaches*. New York: Oxford University Press.
- Hirsch J (Ed.) (1967). *Behavior-genetic analysis*. New York: McGraw-Hill.
- Hirsch J (1981). To unfrock the charlatans. *Sage Race Relations Abstracts*, 6, 1-65.
- Kendler KS, Kessler RC, Neale MC, Heath AC & Eaves LJ (1993). The prediction of major depression in women: Toward an integrated etiologic model. *American Journal of Psychiatry*, 150, 1139-1148.
- Lewontin RC, Rose S & Kamin LJ (1984). *Not in our genes: Biology, ideology, and human nature*. New York: Pantheon.
- McGue M, Bacon S & Lykken DT (1993). Personality stability and change in early adulthood: A behavioral genetic analysis. *Developmental Psychology*, 29, 96-109.
- McGue M & Lykken DT (1992). Genetic influence on risk of divorce. *Psychological Science*, 3, 368-373.
- McGuffin P, Owen MJ, O'Donovan MC, Thapar A & Gottesman II (1994). *Seminars in psychiatric genetics*. London: Gaskell Press.
- Meehl PE (1962). Schizotaxia, schizotypy, schizophrenia. *American Psychologist*, 17, 827-838.
- Mendlewicz J & Hippis H (1992). *Genetic research in psychiatry*. Berlin: Springer.
- Neale MC & Cardon LR (1992). *Methodology for genetic studies of twins and families*. Dordrecht: Kluwer.
- Petrill SA & Thompson LA (1993). The phenotypic and genetic relationships among measures of cognitive ability, temperament, and scholastic achievement. *Behavior Genetics*, 23, 511-518.
- Plomin R & Bergeman CS (1991). The nature of nurture: Genetic influence on environmental measures. *Behavioral and Brain Sciences*, 14, 373-427.
- Plomin R & McClearn GE (1993). *Nature, nurture and psychology*. Washington DC: American Psychological Association.
- Plomin R, Owen MJ & McGuffin P (1994). The genetic basis of complex human behaviors. *Science*, 264, 1733-1739.
- Sabelli HC & Carlson-Sabelli L (1989). Biological priority and psychological supremacy: A new integrative paradigm derived from process theory. *American Journal of Psychiatry*, 146, 1541-1551.
- Scarr S & McCartney K (1983). How people make their own environments: A theory of genotype → environment effects. *Developmental Psychology*, 54, 424-435.
- Schiff M & Lewontin R (1986). *Education and class*. Oxford: Clarendon.
- Sing CF, Haviland MB, Templeton AR, Zerba KE & Reilly SL (1992). Biological complexity and strategies for finding DNA variations responsible for inter-individual variation in risk of common chronic disease, coronary artery disease. *Annals of Medicine*, 24, 539-547.
- Sing CF & Moll PP (1990). Strategies for unravelling the genetic basis of coronary artery disease. In Berg K, Retterstol N & Refsum S (Eds.), *From phenotype to gene in common disorders* (pp. 37-59). Copenhagen: Munksgaard A/S International Publishers.
- Sing CF & Reilly SL (1993). Genetics of common diseases that aggregate, but do not segregate, in families. In Sing CF & Hanis CL (Eds.), *Genetics of cellular, individual, family and population variability* (pp. 140-161). New York: Oxford University Press.
- Turkheimer E (1991). Individual and group differences in adoption studies of IQ. *Psychological Bulletin*, 110, 392-405.
- Turkheimer E & Gottesman II (1991). Individual differences and the canalization of human behavior. *Developmental Psychology*, 27, 18-22.
- Turkheimer E, Lovett G, Robinette CD & Gottesman II (1992). The heritability of divorce: New data and theoretical implications. *Behavior Genetics*, 22, 757 (abstract).

Eric Turkheimer, Department of Psychology, Gilmer Hall, University of Virginia, Charlottesville, VA 22903 (USA).