

## **A Note on the Scope of Developmental Behaviour Genetics**

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The behaviour genetic decomposition of individual differences has been presented as being irrelevant to the study of human behavioural ontogeny. This introduces two problems. First, the analysis of systematic differences constitutes the basis for most statistical models used in the social sciences. If, generally speaking, this type of analysis is uninformative regarding development, how then can one empirically investigate human development? Second, behaviour genetic analyses are the only way to arrive at meaningful statements regarding the contributions of heredity and environment to human development. If results thus obtained are irrelevant, it is impossible to say anything on the subject of heredity, environment, and human ontogeny that is both meaningful and informative.

It is argued that developmental behaviour genetics should not be viewed as a theory of development, but rather as a method of testing certain well-defined hypotheses regarding the contributions of genetic and environmental influences to human development.

Individual differences assessed at any point in time reflect developmental processes prior to that time—gene-environment models are in a very basic sense inherently developmental ... (Loehlin, 1975, p. 41).

Obviously the finding of innate differences in behaviour does not illuminate the development of that behaviour in any way (Johnston, 1988, p. 623).

What causes this striking contrast in the appreciation of the quantitative genetic analysis of human behaviour apparent in these quotations? Certainly not the common misunderstanding with regard to the model employed in quantitative genetics or the meaning of research results. Both Loehlin and Johnston are highly knowledgeable in this respect and this makes their disparate statements all the more interesting. The question concerning the relevance of quantitative genetic analyses to the study of

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human ontogeny is central to the nature-nurture debate. Yet it has not been given much attention, especially compared to the many discussions concerning the feasibility of quantitative genetic analyses of human behaviour. Discussions concerning the feasibility of these analyses (e.g. Roubertoux & Capron, 1990) focus on the basic tenets of statistical models and the many assumptions which are made in employing them. The question of relevance is of a higher order for even if the greatest possible agreement were reached regarding the acceptability of behaviour genetic models and their attendant assumptions, Loehlin and Johnston would presumably not alter their statements.

In the present paper, the question of the relevance of the quantitative genetic analyses to the study of human behavioural ontogeny is discussed. We argue that the rejection of the behaviour genetic decomposition of individual differences as irrelevant in the study of human development is, in a general sense, problematic. Social scientists have no other means to test their hypotheses than by studying differences either between or within groups. If the behaviour genetic decomposition of individual differences is deemed generally to be irrelevant, then how does one establish the relevance of any other such decomposition? Additionally, the quantitative genetic analysis of human behaviour is the only way to arrive at coherent and meaningful statements regarding the contributions of genetic and environmental influences on human behaviour. That is, there is no other way in which one can make quantitative, verifiable statements regarding these influences on human behaviour. If such statements are irrelevant it then is impossible to say anything that is simultaneously meaningful and relevant on the subject of nature, nurture, and human development. This would certainly resolve the so-called nature-nurture issue, but in a particularly unsatisfactory manner.

It is proposed here that behaviour genetics should not be viewed as a theory of development, but as a methodological tool that can be used to test certain well-defined hypotheses regarding the covariance between phenotypic, genotypic, and environmental individual differences. This perspective leads to a rejection of the behaviour genetic (or any other) decomposition of individual differences only in so far as results thus obtained cannot be placed in a theoretical or descriptive framework regarding the development of the phenotype under consideration.

### THE CHARGE OF IRRELEVANCE

The charge of irrelevance has been made by influential commentators on the nature-nurture issue. Johnston (1987, 1988) is among the most recent and most outspoken in this regard, for example, and has written (Johnston, 1987, pp. 177-178):

Population geneticists are concerned with the analysis of phenotypic variation in populations, not phenotypic development in individuals. They have devised statistical techniques that allow that variation to be partitioned among genetic variation, environmental variation, and the interaction between the two. But accounting for variation in a trait among individuals in a population is quite a different matter from accounting for its development in the individuals that make up that population... The analysis of development answers the question 'How does the phenotype of an individual in this population come to be the way that it is?'

A similar point of view has recently been advocated by Lerner and von Eye (1992). In a discussion of quantitative genetic decomposition of phenotypic variance, they state the following with respect to the utility of heritability estimates (p. 27):

In sum, then, heritability estimates describe only the characteristics of a distribution of scores; they describe only a feature of differences between people. Such estimates say nothing about the trait itself. Such estimates, in particular, say nothing about the genetic and/or environmental determination (or cause) of the trait within any person in a group.

Others have expressed reservations concerning the value of analysing human variation in the study of human development. For instance, Oyama (1982, p. 108) writes: "... heritability ... sheds little light on development because that is not what it is about." Lehrman, who has written two papers (1953, 1970) which are among the most influential in this context, also expresses reservations, although he is somewhat circumspect. He states that considerations relating to the sources of phenotypic differences do not really bear on the question of ontogenetic origin, which he judges to be (Lehrman 1970, pp. 25-26): "to some degree a question of a different kind." Similar remarks, although varying in explicitness, can be found in, for instance, Anastasi (1958, p. 197), Hebb (1953, p. 163), Michel and Moore (1978, p. 68), and Oyama (1985, p. 45).

We suspect that these reservations are at least partly motivated by the disparity between theoretical notions regarding the roles of the genetic and the environmental in behavioural ontogenesis on the one hand (referring to the epigenetic developmental process), and the model employed within the field of developmental behaviour genetics on the other (see Plomin, 1986; Plomin, DeFries, & Fulker, 1988). The latter is represented by an explicit linear regression model. In its most simple form a phenotypic deviation score is the linear contribution of a genetic deviation and an environmental deviation. This score is usually expressed for subject *i* by the equation:

$$dP_i = dG_i + dE_i$$

where  $d$  before each symbol reminds the reader that these variables are deviations from some fixed point (e.g. the mean values). The basic model has been extended to time- or age-dependent deviation scores yielding models that can accommodate longitudinal or time series data (Boomsma & Molenaar, 1987; Eaves, Long, & Heath, 1986; Hewitt 1990). Such approaches deal directly with developmental processes in terms of inter- and intra-individual differences in inter-individual change. These models, like all instances of the general linear model, have in common that (dependent) difference scores are related to other (independent) difference scores and that the results are intimately tied up with the question "How much?" Violations of the assumptions usually made in this model, such as nonorthogonality of the genetic and environmental variables, the presence of G×E interaction, assortative mating, etc., can be accommodated to varying degrees depending on the data available (see Neale & Cardon, 1992, and the references therein).

In theories of behavioural development addressing the nature-nurture issue, on the other hand, we generally do not have an explicit model, but a verbal description or theory regarding how genetic and environmental influences conjoin in the epigenetic process (e.g. Bateson, 1987; Lerner 1986, chapter 5). The theory concerns the causes of development, or ontogenetic causes which pertain to the ontogeny of the individual and can, for conceptual clarity, be distinguished from phylogenetic (distal, ultimate) and immediate (proximate) causes. According to Lehrman (1953), the study of ontogenetic causes involves addressing the problem of (p. 345): "the development of new structures and activity patterns from the resolution of the interaction of the existing structures and patterns, within the organism and its internal environment, and between the organism and its outer environment."

This contrast between what is essentially a statistical model with a particular choice of independent (genetic and environmental) variables and substantive developmental theory has found a number of analogous expressions. Anastasi (1958) argued that the study of behavioural ontogeny should address "how" and not "how much" heredity and environment contribute to development. "Causes of development" (or the "global analysis of causes") versus "causes of variation" is another analogous contrast (Lewontin, 1974). Lewontin, like Johnston (1987), rejects causes of variation as a worthwhile pursuit, if one's chief interest is causes of development. Both Anastasi and Lewontin are frequently referenced in comments on the nature-nurture issue. The point is usually made that the quantitative approach ("how much" or "analysis of variation") is not useful in the study of ontogeny.

A related phenomenon is the association of behaviour genetics with the mechanistic orientation within theoretical developmental psychology. For

example, this is expressed by Sameroff (1983, p. 249) in the *Handbook of child development*:

The issue of development, which might be thought to be central to the exploration of gene-behaviour relationships, is here ignored because in the mechanistic causal equation development is irrelevant.

The "here" in this quotation refers to the quantitative genetic decomposition of phenotypic individual differences. Overton and Reese (1973, pp. 80-81) express this as follows:

Anastasi's (1958) analysis implied that psychologists had come to agree that the question 'Which one?' and 'How much?' are meaningless with reference to the individual contributions of nature and nurture to individual development. But this was not true, because as long as a group maintains the mechanistic position and its corollaries of unidirectionality and linearity, at least the more sophisticated of these questions—'How much?'—will continue to constitute a meaningful issue ... In marked contrast, for a group that accepts the organismic position and its corollary of reciprocal causation ... the questions 'Which one?' and 'How much?' lose all meaning.

The tendency to associate behaviour genetic research with a particular theoretical orientation is based on the perception of behaviour genetics not as a methodology but rather as a theory of development. This perception, which leads to the proposition that developmental behaviour genetics can not be used to investigate causal theories of development, or that the adoption of the former implies a particular theoretical orientation, is problematic in two regards. These are considered next.

## TWO PROBLEMS

The first problem is that many hypotheses in developmental psychology are, in one way or another, stated in terms of differences between or within groups. It is hard to imagine any other way to proceed in empirically testing a theory of development, whether the theory is mechanistic, organismic, or contextualistic, or whether the research is correlational (differential) or experimental. A perusal of Wohlwill's programmatic view of the study of development (Wohlwill, 1973, chapter 3) reveals that theories of development which address behavioural ontogenesis invariably involve the study of differences (see also Baltes & Nesselroade, 1973; Buss & Royce, 1975). This is so even though very likely the prime objective of the developmental psychologist is the explanation or description of ontogeny and not the analysis of differences. So, if, in general, "the analysis of variation" is not suited to the study of ontogenetic causes or incompatible

with this study, then how should the developmental psychologist proceed in investigating such causes?

It has been argued that the proposed irrelevance of the behaviour genetic decomposition of individual differences is due to the global nature of the variables environment and genotype. For instance, the environment is often featured as an essentially negatively defined (i.e. non-genotype) aggregate encompassing many unidentified variables (Wachs, 1983). Although behaviour genetic studies have included measures of the home environment (see Plomin, DeFries, & Fulker, 1988) and can include specific knowledge concerning the genotype to detect single gene influences on metric traits (e.g. Fulker, Cardon, DeFries, & Kimberling (1991), this is a valid criticism. It cannot, however, justify the charge of irrelevance. Specifically, how does one determine what is a relevant independent variable and what is not? It appears that the answer to this question lies in the relationship between developmental hypothesis originating in developmental theory and statistical models. Decompositions of variance, regardless of the nature of the independent variables, are irrelevant when carried out in a theoretical vacuum. They gain meaningfulness against a theoretical or descriptive background which properly motivated them in the first place. On the basis of this reasoning, there is no reason to reject out of hand the behaviour genetic decomposition of individual differences, or to judge even the most meagre summary of such an analysis, the heritability (or environmentality) coefficient, as irrelevant.

Furthermore, the use of a linear model to test a particular hypothesis does not imply that the theory which generated the hypothesis is inherently linear. In this connection, it is interesting to note that Molenaar and Oppenheimer (1985) have shown that dynamic models of development, ranging from Newtonian mechanistic models to models based on nonequilibrium thermodynamics, are inherently neutral with respect to the mechanism-organicism controversy. Here we take a similar view that a statistical model, such as the regression model, should be viewed as a neutral tool, and not as embodying any particular theoretical world view such as organicism or mechanism. Of course, one may wish to reject statistical models based on linearity if they are deemed to be unsuitable, but it then becomes a problem how to empirically test one's hypotheses. This is apparent in Lerner, Skinner, and Sorell (1980), who reject the linear statistical models to investigate their developmental hypotheses, but are, unfortunately, subsequently forced to conclude that "appropriate models do not currently exist" (p. 231).

Let us consider, briefly, two examples to illustrate the present point. Gottlieb (1991, p. 9), in an article on the role of experiential influences on the canalisation of development, cites the following datum as being of interest to his thesis:

... it is significant that the amount of protein in the developing rodent and chick brain is influenced by two sorts of environmental input: nutrition and sensori-motor experience. Undernutrition and 'supernutrition' produce new-born rats and chicks with lower and higher quantities of cerebral protein, respectively.

To establish the linear (or curvilinear) relationship between nutrition and cerebral protein, one can regress protein on nutrition and statistically test the (null) hypothesis that the regression coefficient  $\beta$  is 0 against the alternative  $\beta > 0$ . This approach relates difference scores on one variable to those on the other according to a linear model. This decomposition of the variation in protein into a part that coincides with the variation in nutrition and a part that does not, is deemed by Gottlieb to be of interest given his theory. But can we conclude on this basis either that Gottlieb adheres to a mechanistic world view or that his theory concerning the role of experience in canalisation is of an additive, linear, or generally mechanistic nature? Or do we conclude with Lerner and von Eye (1992—see quotation above) that the demonstration of an environmental contribution (*viz.* nutrition) to the differences between new-born rats is of no value because it says nothing about the environmental determination (or cause) of the trait *per se* within the rat?

First, Gottlieb is, as a proponent of probabilistic epigenetic position, probably as far removed from the mechanistic view of development as one can get (see Gottlieb, 1983). Yet, if a theory concerning cognitive development predicts an increasingly large contribution of genetic influences to phenotypic differences in, say, IQ, can this be construed to mean that the theory or the researcher are of a mechanistic bent or that the researcher's single goal is to answer the question: How much ... etc? The only conclusion which can be drawn is the rather mundane one that the researcher subscribes to the prevailing methodology of empirical psychology of which the general linear model is an important aspect.

Second, it is facile to complain that an analysis of differences says nothing about the environmental and/or genetic determination of the trait within the organism. The question how the development of a given phenotype is genetically or environmentally determined within the organism, without comparison to a second (comparable) phenotype, is not answerable. As reiterated later, a question concerning the contribution of genotype and environment to phenotypic development, has to be stated in terms of phenotypic differences (*i.e.* difference scores) to be answerable.

As a second example, we merely mention the central role that is played by simple measures of association (*e.g.* Pearson product moment correlation coefficient) in the continuity-discontinuity debate. Although the correlation between, say, speed of habituation in infancy and measures of intelligence in

later life is based entirely on individual differences in the behaviours mentioned, this cannot be taken to imply that this correlation is not informative with respect to theories of development of the behaviour *per se*. We refer the reader to Volume 32 (1989) of the journal *Human Development* which is devoted to just this issue (see also Bornstein & Sigman, 1986).

The second problem associated with the rejection of the behaviour genetic analysis of individual differences is related to the first. It is universally accepted that it is only through the decomposition of individual differences that one can speak of both genetic and environmental influences on behaviour in a meaningful manner. This is an important theme in the Lehrman articles (1953, 1970) and is reiterated in many important discussions of the nature-nurture issue (e.g. Bateson, 1987; Hebb, 1953; Johnston, 1988; Medawar & Medawar, 1979; Oppenheim, 1982; Oyama, 1982; Scarr & Weinberg, 1980; Weiss, 1973; Wilson, 1978). Dobzhansky has stated this very clearly (in Plomin et al., 1990, p. 233):

The genotype and the environment are equally important, because both are indispensable ... The nature-nurture problem is nevertheless far from meaningless. Asking the right question is, in science, often a large step towards the right answer. The question about the roles of the genotype and the environment in human development must be posed as thus: To what extent are the differences observed among people conditioned by the differences of their genotypes and by the differences between the environments in which people were born, and were brought up in?

Discussions of the influence of nutrition and sensorimotor experience on the developing brain, necessarily include propositions in terms of differences. The statement "higher amounts of nutrition are associated with higher amounts of protein" implies an analysis of differences. Indeed it is hard to imagine any other way to state this relationship: the statement "nutrition causes protein" is as fallacious as "environment (heredity) causes phenotype X". Generally, statements regarding environmental and (or) genetic influences that are couched in terms other than those relating to the analysis of individual differences incoherent. Johnston (1987), Lehrman (1970), and Oyama (1982) discuss many examples of seemingly appealing, but flawed gene-environment models.

Now, if we agree that the behaviour genetic decomposition is irrelevant and the attendant analysis of variance unsuited to the study of development, we must accept the implication that no statement can be made regarding the contributions of genetic and environmental contributions to human development which is simultaneously meaningful and relevant. And, considering the former problem, we must also concede that in most empirical developmental studies of human behaviour, hypotheses are investigated using linear statistical models, (i.e. by studying differences in



phenotype and not the phenotype itself). By consistent rejection of the analysis of differences to investigate environmental (genetic) influences on behavioural development, one arrives at a point where it is in fact impossible to carry out relevant empirical developmental research.

The undesirable implications of the aforementioned can be avoided by viewing developmental behaviour genetics as a methodology that specialises in the time-dependent inter-relationship between genetic and environmental variables and their relation with phenotypic variables, and not as a developmental theory of human behaviour. Accordingly, the behaviour genetic decomposition of individual differences, or for that matter any other decomposition, cannot be regarded as irrelevant without considering the developmental hypotheses which informed the decomposition in the first place. As Freedman (1974, p. 3) notes: "It strikes me as a safe prediction that most correlations obtained with twin studies will dry and blow away with time, and only those that attain comprehensibility in the light of our evolved nature will remain." Similarly, we argue that behaviour genetic research gains relevance and comprehensibility in the light of our theoretical understanding regarding development.

### LIMITATIONS

It is important to realise the limitations to which behaviour genetic investigation of behavioural ontogeny is subject. Typically, no experimental manipulation of either the genotype or of the environment is possible. This lack of experimental control necessarily limits the scope of the developmental hypothesis to those genetic and environmental effects that: (1) occur naturally in the population studied; and (2) contribute to phenotypic differences between the members of that population. In view of these limitations, developmental behaviour genetics is more associated with differential psychological research than with experimental research and consequently involves more description of the ontogenetic process than testing of formal theories of development by means of a hypothetico-deductive model of scientific inference (McCall 1981; Wohlwill, 1973). Perhaps this position on the differential-experimental continuum underlies to an extent the rejection of individual differences as a tool in the investigation of development. For instance, we have had occasion to note Lehrman's (1970) reticence concerning the relevance of the behaviour genetic decomposition of individual differences. Nonetheless, Lehrman (1970, pp. 29 and 39) clearly acknowledges the relevance *vis-à-vis* the developmental process of experimental manipulation that causes a change in the behavioural outcome of that process. Yet, it seems to us that the developmental behaviour geneticist does exactly this by demonstrating that genetic or environmental differences—caused by naturally occurring

variation in genotypes and environments instead of experimental manipulation—make a difference to the behavioural outcome of a developmental process. It is in this sense that we read and agree with Loehlin's statement as quoted in the introduction to this paper.

Furthermore, reading Anastasi (1958) from the present perspective, it seems that Anastasi's message is not to banish posing the question: "How much?" but rather that theoretical considerations regarding the impact of the environment and heredity on human behaviour should inform the decomposition of phenotypic differences. For instance, at the end of her paper, Anastasi suggests seven promising methodological approaches to test such theoretical considerations. Although all seven imply an analysis of differences, we mention two briefly. The investigations into the hereditary conditions which underlie behavioural differences between selectively bred groups of animals and investigations into the psychological development of twins from infancy to maturity. That both these approaches are based on an analysis of differences can be established by reading, for example, Mather and Jinks (1982) (biometric analysis of inbred strains), Plomin (1986), and Plomin et al. (1988) (longitudinal twin research).

Speaking generally, it appears to us an undeniable fact that issues relating to *how* nature and nurture conjoin in psychological development are necessarily investigated empirically by posing some form or other of the question: *How much?*

## CONCLUSION

In the present paper we have tried to argue against: (1) the treatment of developmental behaviour genetics as a (mechanistic) theory of development; and (2) its presentations as useless in the study of human behavioural ontogeny because it is limited to the analysis of phenotypic differences, instead of phenotypes themselves. Rather we view this field as strongly methodological and as the only source of empirical, quantifiable information regarding genetic and environmental contributions to human behaviour. We have not concerned ourselves with the difficulties associated with this quantification (i.e. the tenability of the many assumptions in employing, for instance, the twin method or an adoption design). This is because we view this issue as beyond the scope of our present message; we do not wish to play down or discount these difficulties. We refer the reader to standard texts, such as Mather and Jinks (1977); Neale and Cardon (1992) or Plomin et al. (1990), for discussions of these difficulties.

The differential orientation of developmental behaviour genetics implies that it is a tool to describe the sources of individual differences during the developmental process, rather than an experimental tool. Central issues in developmental research are, however, formulated in terms of naturally

occurring individual differences such as the developmental implications of (in)stability of individual differences. Furthermore, the study of human development often requires evidence from diverse sources, none of which may in itself be conclusive given the specific hypotheses (McCall, 1981).

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