
COMMENTARY

Relational Developmental Systems and Quantitative Behavior Genetics: Alternative or Parallel Methodologies?

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The two methodologies presented in this special issue—relational developmental systems and quantitative behavior genetics—are not competing alternatives in the exploration of genetic–environmental interplay, but parallel approaches having distinct focuses and distinct goals. This commentary explores several conceptual issues that can, and have in the past, result in confusions about the nature of each methodology, and their relation to each other.

It is most unlikely that there is a scientist or layperson alive who doubts that genetics specifically and biology generally constitute areas of inquiry that are fundamental to understanding and explaining the nature and development of the cognitive, emotional, and motivational dimensions of human behavior. But there have been, and continue to be, major issues about how science can best arrive at valid conclusions concerning the specific roles played by genetic, and more broadly biological, and environmental systems in this scientific understanding. Speaking of the “interplay” of genetic and environmental systems does not raise the discussion to the level of such issues, but once this notion of interplay introduces concepts *interaction*, *fusion*, and *coaction*, the issues arise full force.

The articles in the current special issue attempt to find common ground among disparate methodologies that have been applied to the study of interplay of the genetic and environmental systems. Finding a common ground is an admirable goal, but it should not be pursued at the sacrifice of conceptual clarity. And here I would like to briefly address some conceptual issues in what is generally referred to in the literature as $G \times E$ (gene–environment) research.

Broadly, the articles in this issue divide into two methodological approaches to the study of genetic–environmental interplay: the relational developmental systems approach taken by Greenberg and Partridge (and, as full disclosure that would be difficult to hide, me), and the population or quantitative behavior genetics approaches of Burk, Dick, and Turkheimer. Although these approaches have traditionally been viewed as competing alternatives, this is not a necessity, as each can be understood as having different foci and different goals. Relational developmental systems focus on the causal patterns of the development of the individual, on intraindividual variation, whereas population behavior genetics is concerned with interindividual variation (individual differences). Although not necessarily competing alternatives, the different approaches can become so when individual development is confused with population individual differences and/or when it is incorrectly assumed that findings based on the study of interindividual variation can be generalized to intraindividual situations. The error of this latter assumption is too complex to explore here, but the interested reader is referred to the work of Molenaar and his colleagues for elaboration (e.g., Molenaar, 2004; Molenaar & Campbell, 2009; Nesselrode & Molenaar, 2010).

With respect to goals of each approach, following the suggestion of Turkheimer (this issue) and others (Griffiths & Tabery, 2008), it is reasonable to say that the relational developmental systems approach aims at establishing the explanatory causal base of genetic–environmental interplay, whereas quantitative behavior genetics aims at predicting population differences. Within this framework, the concepts *coaction*, (Gottlieb, Wahlsten, & Lickliter, 2006), *fusion*, (Greenberg, this issue; Partridge, this issue) as well as *bidirectional* (\leftrightarrow) *causality* (Lerner, 2006), *relational causality* (Gottlieb, 2003), and *circular causality* (Witherington, 2011) are synonyms used in relational developmental systems models to indicate an understanding that genes and environment do not constitute discrete pure forms and, as a consequence, behavior cannot be considered to be the additive accumulation of a number of independent factors. Holistic, nonadditive, nonlinear, epigenetic self-organizing systems are, as discussed by Greenberg (this issue) and Partridge (this issue) fundamental to relational developmental systems methodology. Traditionally *transaction* has been synonymous with these relational causal concepts, but in this special issue it is presented in a more neutral fashion; more akin to $G \times E$ interplay. This usage can be conceptually confusing, especially to those who are familiar with, and/or work within, a transactional paradigm (see, e.g., Sameroff, 2009)

The concept *interaction* has had a more ambiguous position in the history of $G \times E$ interplay research. As Dick (this issue) notes, *interaction* has not infrequently been used loosely to mean that genes and environment act together in contributing to an outcome. Before the significance of reciprocal or relational causality became clear, *interaction* was also occasionally used by some taking a relational developmental systems approach. However, again as Dick (this issue) points out, it is the statistical concept of interaction that is fundamental to the program of quantitative behavior genetics. This concept implies a quantitative association among variables such that one varies as a function of the variability of another. This relation has, in fact, been the meaning of the \times in the published $G \times E$ literature.

For purposes of the present discussion the single most significant feature of the statistical meaning of the concept is that the interaction is completely decomposable into strictly additive elements. Partridge (this issue) and Turkheimer (this issue) accurately elaborate on the centrality of this assumption of additivity to quantitative behavior genetics in their descriptions of the nature and significance of the Fisher–Wright ANOVA model. Turkheimer refers to this additive model as “the foundation of modern quantitative genetics.” Partridge supports this judgment and quite correctly goes on to point out that advances in the model, such as extensions to multivariate and latent variable models and multilevel models, adhere to the same additive structure as the original Fisher model.

The nonadditivity and nonlinearity of relational developmental systems and the strict additivity of quantitative behavior genetics may again seem to place these approaches into conflict as competing alternatives. But, to repeat, this conflict is only the case if one needlessly casts the two approaches as methodologies which focus on identical content and maintain identical aims.

Although the methodologies are not necessarily in conflict there are two points of importance concerning the additivity/nonadditivity and broader statistical features of $G \times E$ interplay. The first concerns the historical fact that the relational developmental systems approach has lacked a toolbox of nonlinear analytic methods and, as a consequence, has often been in the unfortunate position of attempting to express nonadditivity effects in an additive context. For example, as Partridge (this issue) points out, whereas Burt (this issue) and Dick (this issue) incorporate measured genetic and environmental factors in their work, “the underlying assumptions of the original Fisher (1918) model and its latent variable model extension . . . are still deeply embedded” (Partridge, 2011, p. 246) in their analytic models. The fact that, as described by Greenberg and Partridge, nonlinear analytic methods have been emerging and are being employed with increasing frequency is refreshing and encouraging. Certainly the continuing development of nonlinear analytic methods will go a long way to avoiding conceptual confusions, exemplified by a presentation of the argument that “the field has now largely embraced a more *holistic* view of development, in which genetic and

environmental influences are viewed as *inexorably intertwined*" (Dick, 2011, p. 212) but, in the same context, going on to employ analytic tools designed to quantitatively isolate genetic and environmental elements.

The second point to be made concerning statistical features of $G \times E$ interplay involves the issue of causality. As suggested by Turkheimer (this issue), causality is an enormously complex topic. However, it simply cannot be too strongly emphasized that statistical models are inductive in nature and do not, in themselves, directly implicate causes. Statistical models describe quantitative associations. It is conceptually incoherent to claim "genetic influences" or "environmental influences" on the basis of statistical associations. In a related fashion, one might coherently argue on the basis of data that genes play an important role in the determination of height, but this is quite different than the statement that "height runs in families for genetic reasons" (Turkheimer, 2011, p. 232, emphasis added).

Closely related to the statistical issues is the problematic nature and status of "heritability" in the $G \times E$ quantitative population behavior genetics literature. The simple fact of the matter is that heritability (H^2) is a percentage generated by a statistical formula applied to differences in variances between individuals, and it should never be reified as a concept of biological inheritance. This fact has been described, explained, elaborated, and emphasized over and over again across the years by numerous investigators (e.g., Hirsh, 1992; Lerner & von Eye, 1992). In fact several investigators have described specifically nongenetic interpretations of heritability, for example, "A measure of how well the state of the parent predicts the state of the offspring" (Sterelny & Griffiths, 1999, p. 35; see also Weber & Depew, 2001). Despite this literature, and despite the extensive criticism raised concerning methodological and inferential features of family, twin, and adoption studies (e.g., Joseph, 2010) that form the database for the generation of heritability estimates, the concept continues to be used as a surrogate for "genetic influence." Turkheimer (this issue) concludes that "heritability is a distraction" and that is a conclusion that I find difficult to fault. But this conclusion needs to go further and recognize that what have been termed "genetically informed designs" involving families, twins, and adoptions that have yielded the heritability estimates are not themselves actually genetically informative (see Partridge, 2005) and represent as much of a distraction as does heritability itself. I would make the modest suggestions that these studies be interpreted as organism \times environment interactions rather than being used as a means of buttressing genetic explanations or as a means of getting closer to genetic causes. This usage would leave quantitative behavior genetics with the tasks of continuing its movement towards making predictions based on genes themselves or their close associated single nucleotide polymorphisms.

In conclusion, though there are a number of conceptual and methodological issues that require continued study there appears to be no gap and, hence, no

required bridge between relational developmental systems and quantitative behavior genetics. The two are not competing alternatives but two distinct modes of exploring the interplay of genes and environment.

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