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SPECIAL SECTION EDITORIAL

Gene–environment correlation in developmental psychopathology

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Abstract

Modern research acknowledges that psychopathology and individual differences in normal development are the joint products of both biological and social influences. Although there have been numerous publications on Gene × Environment interactions in the past decade, gene–environment correlation is another important form of gene–environment interplay that has received less attention. This Special Section demonstrates, using a range of methodological approaches, the importance of gene–environment correlation in developmental psychopathology. Several types of gene–environment correlation are described, including passive, evocative, and active. Other studies highlight the potential for gene–environment correlation to obscure associations between risk exposures and child psychopathology. Future directions for gene–environment correlation research are discussed.

A key principle in modern research on developmental psychopathology is that psychopathology and individual differences in normal development are the joint products of both biological and social influences (Kendler, 2011; Rutter, Moffitt, & Caspi, 2006). Interest in the study of Gene × Environment (G × E) interactions has soared in recent years, with scores of papers published on the interactive effects of measured genes and measured environments (Duncan & Keller, 2011). Although there are methodological and conceptual challenges to be overcome in the study of G × E (Duncan & Keller, 2011), these studies have offered proof of principle, generated new theories about gene–environment interplay (e.g., Belsky & Pluess, 2009), and generated new hypotheses about how genes and environments interact at the biological level to influence the development of normal and abnormal behavior (Caspi, Hariri, Holmes, Uher, & Moffitt, 2010).

A much less studied process by which genes and environments operate together in development concerns gene–environment correlation (*r*GE). This Special Section is concerned with the role of *r*GEs in developmental psychopathology. The term *r*GE describes the process by which an individual's genotype influences, or is associated with, his or her exposure to the environment (Kendler & Eaves, 1986; Plomin, DeFries, & Loehlin, 1977). Although the notion of *r*GE may be relatively unfamiliar to psychologists and psychiatrists, it builds on well-established theories of person–environment correlation (e.g., Elder, 1998) that describe how a person's behavior, personality, or cognitive abilities shape his or her environment. As noted by Kendler (2011), the study of *r*GE also has a long history in evolutionary biology, where there is ample evidence that animals are genetically programmed to modify their environments in ways that maximize genetic fitness.

Plomin et al. (1977) described three main *r*GE processes: passive, evocative (also called reactive), and active. In *passive rGE*, genetic relatedness between the parent and the child accounts for observed correlations between partially heritable traits, such as the child's behavior and the child's environment. For example, the reason children who are spanked or smacked are more aggressive than children who are not (Gershoff, 2002) may be that parents transmit a genetic risk for aggressive behavior that increases both the probability that their children will be aggressive and the likelihood that parents will use physical discipline rather than other disciplinary practices (DiLalla & Gottesman, 1991). In *evocative rGE*, partially heritable traits or behaviors evoke reactions from others in the environment. For example, children who are shy or withdrawn may appear aloof to peers who will, as a result, be less likely to make friendly overtures. In *active rGE*, individuals actively select or create environments that are associated with their genetic propensities. For example, youth who tend to follow rules and who adhere to social norms will be likely to seek out like-minded peers. The papers in the Special Section provide empirical demonstrations of all three kinds of *r*GE.

The Importance of Understanding *r*GE

For our understanding of the development of psychopathology, *r*GEs matter for at least two reasons, both of which are

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demonstrated in the papers included in the Special Section. The first reason is that they potentially obscure our understanding of the relationship between risk exposures and outcomes. The conventional understanding in much of the social and clinical sciences is that at least some of the things children experience in their day-to-day environments play a causal role in increasing their risk for psychopathology. These include exposure to marital conflict, harsh discipline, peer rejection, peer deviance, family and neighborhood poverty, and so on. This causal environmental model is represented in the top portion of Figure 1. However, the possibility that genes and environments are correlated calls this simple causal model into question. If the same genes that ultimately give rise to specific experiences also give rise to psychopathology, then the association between environmental exposures and child outcomes will be confounded. Of course, it is likely that the distinction between causal and noncausal associations is overly simplistic and that there may be reciprocal causal effects over time, where, for example, genetically influenced behaviors evoke responses from the environment that then reinforce those behaviors in a causal manner. Two models of such *r*GE are represented in the middle and bottom portions of Figure 1.

Understanding whether the relationship between risk exposures and outcomes is causal, noncausal, or indicative of more complex reciprocal processes matters for our understanding of the etiology and course of disorder and has important implications for how we develop interventions to prevent psychopathology. Special research designs are required to distinguish causal and noncausal hypotheses (e.g., Foster, 2010; Jaffee, Strait, & Odgers, 2012; Rutter, 2007). Such designs are described in greater detail below.

The second reason that *r*GEs matter is that they inform our understanding of how psychopathology develops over time.

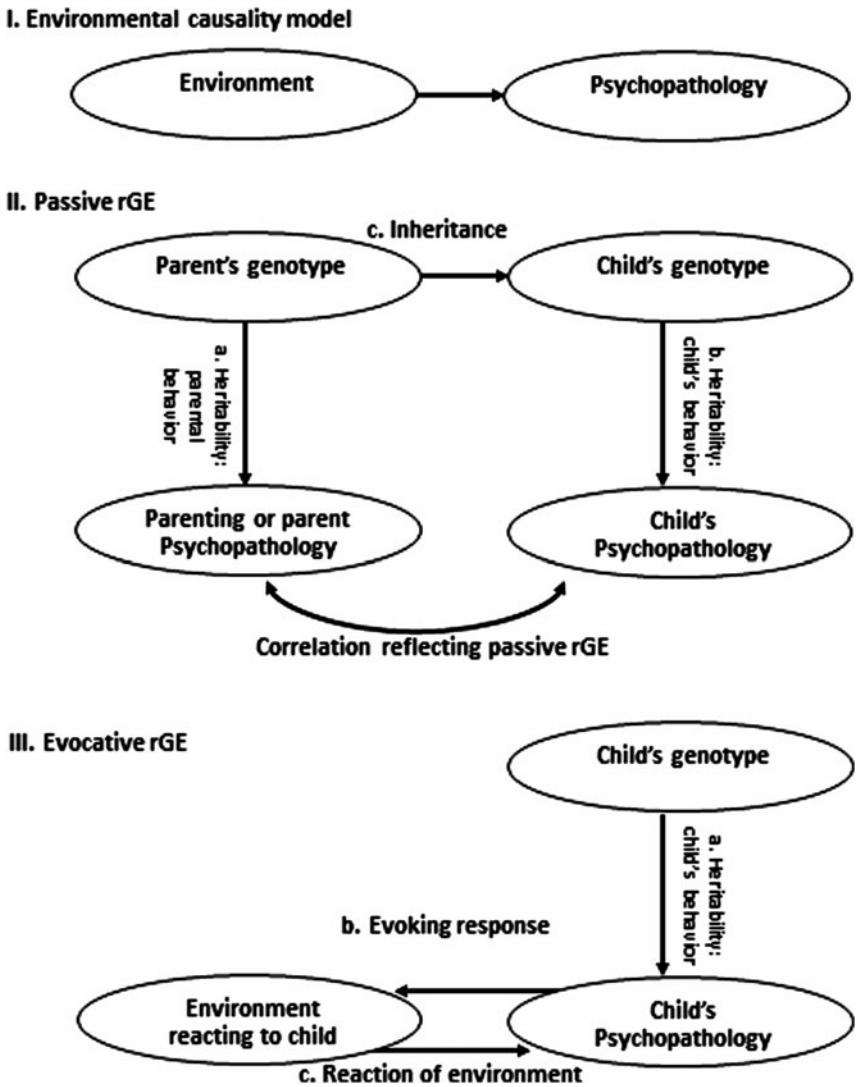

Figure 1. A simple environmental causality model and passive and evocative gene–environment correlations represented schematically. The top panel shows experiences in the environment play a causal role in increasing risk for psychopathology, and the middle and bottom panels show the association between the environment and psychopathology is confounded or initiated by genotype.

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From a life span developmental perspective, relatively small individual differences in temperament or abilities become much larger differences in personality, psychopathology, cognitive ability, musicality, or physical dexterity over time, with *r*GEs partially accounting for these changes (e.g., Caspi & Moffitt, 1995). Again, many of the papers included in this Special Section are concerned with developmental pathways from genetic risk to psychopathology via effects of the person on the kinds of experiences to which he or she is exposed. One of the contributions is specifically concerned with how *r*GEs undergird reciprocal effects of persons on environments over time (Beam & Turkheimer, 2013).

*r*GEs Potentially Confound Associations Between Risk Exposures and Outcomes

Traditionally, research in developmental psychology and psychopathology has relied heavily on research designs that comprise one child per family and in which parents and children are biologically related. Such studies have produced hundreds of findings documenting associations between parenting behavior and child outcome. However, these associations can be the combined result of three effects: (a) the effect of parents' genes on their own behavior, (b) the effect of children's genes on their own behavior, and (c) the overlap between the genetic influences on children's and parents' behavior (see middle panel of Figure 1). In biological families, parents and children share 50% of their genetic variance, and assuming that the same genetic processes influence parents and children, a positive correlation between parents' and children's behavior should be observed, regardless of any direct influence of parents on children. Thus parent–child correlations could be attributed in principle to passive *r*GEs.

Several of the studies included in the section addressed the possibility that genes common to parents and children could account for observed associations between parental behavior and child outcome. For example, although teenage childbirth is a robust risk factor for offspring antisocial behavior (Jaffee et al., 2012), this association could, in theory, reflect a passive *r*GE in which genes common to parents and children will increase both the likelihood that teenage girls will engage in behaviors that result in an early transition to motherhood and the probability that their children will engage in high levels of antisocial behavior. Three papers in this Special Section deal with passive *r*GE. Coyne, Långström, Rikert, Lichtenstein, and D'Onofrio (2013) use a longitudinal children of siblings and children of twins design to rule out the possibility that passive *r*GE accounts for observed associations between teen motherhood and offspring antisocial behavior and to provide support for a causal relationship. The study by Rice, Lewis, Harold, and Thapar (2013) includes parents and their children who were conceived through assisted reproductive technologies. Although the majority of parents were biologically related to the fetuses, some pregnancies involved sperm, egg, or embryo donation. Comparisons of the magnitude of the association between parent and child behavior in the genetically related versus unrelated pairs provides an estimate of passive *r*GE (e.g., Rice et al., 2009), with the current study focused on the link between parent and offspring depressive symptoms. Finally, Lemery-Chalfant, Kao, Swann, and Goldsmith (2013) use data from a sample of twins to estimate the magnitude of passive *r*GE in the relationship between chaos and physical disorder in the home and children's temperament.

Other forms of *r*GE also have the potential to confound observed associations between risk exposures and outcomes. Our genetic propensities to be impulsive or cautious, to react negatively or positively to new experiences, to be verbal or nonverbal, not only shape the way that others interact with us but also shape the choices we make about how and with whom we spend our time. Thus, the child who has difficulty managing his behavior may experience substantially more harsh verbal and physical discipline than the child who is more compliant (Jaffee et al., 2004). The possibility of such heritable child effects on the environment poses interpretive challenges for a unidirectional model in which children are shaped by the parenting they experience and not the reverse. Similarly, the possibility that youth who are aggressive and who break rules without compunction will seek out the company of other youth who will go along with these behaviors challenges the notion that affiliating with delinquent peers causes youth to become delinquent themselves (Kendler, Jacobson, Myers, & Eaves, 2008). These types of *r*GE are addressed in another paper in the Special Section that tests the hypothesis that marriage leads to reductions in men's antisocial behavior (Jaffee, Lombardi, & Coley, 2013). Using a variety of quasiexperimental and statistical matching methods in a longitudinal sample that included sibling pairs, this study rules out the possibility that evocative or active *r*GE could confound observed associations between men's marital status and their antisocial behavior.

*r*GEs Underlie Developmental Processes That Lead to Psychopathology

The *r*GE is not only a methodological challenge but also an important developmental phenomenon in its own right (Reiss & Leve, 2007). Consider the finding that heritability often increases with age (Haworth, Carnell, Meaburn, Davis, Plomin, & Wardle, 2008; Knafo & Plomin, 2006). One possible explanation (see bottom panel of Figure 1) is that children's behavior is genetically influenced and that it affects the way their environment treats them. For example, O'Connor, Deater-Deckard, Fulker, Rutter, and Plomin (1998) reported that adopted children received harsh parenting that was associated with their genotype. The reaction of environmental agents can in turn feed back to children and further affect their behavior. In the harsh parenting example, this kind of parenting can model aggressive or impatient behavior to children. Statistically speaking, these cycles of family influences end up in the heritability estimate, because they are initiated by children's genetic tendencies. However, these effects are not

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really purely heritable, because they also reflect the involvement of the environment. The increased heritability may attest to accumulating influences that involve the reaction of the environment to the child, over a long series of environment–child transactions. Beam and Turkheimer (2013) present simulation data to show how small phenotypic differences among individuals (e.g., the degree to which an individual is more physically dexterous than her sibling) lead to differences in experiences that, over time, reinforce and increase individual differences.

A great deal of research has been concerned with documenting the existence of *r*GEs. In quantitative behavioral genetics, this effort has typically involved estimating biometric models, in which differences in the genetic relatedness of monozygotic and dizygotic twins or adoptees and nonadoptees have been leveraged to decompose the variation in a phenotype into genetic and environmental components. Although psychologists and psychiatrists are often interested in phenotypes like behaviors, personality traits, or cognitive abilities, in biometric models that attempt to capture *r*GE, the phenotype is the putative environment itself. For example, monozygotic and dizygotic twins may report on their experiences of harsh discipline, or parents may report how often they experience conflict with their adoptive and nonadoptive children. Biometric models can be estimated to test how much of the variation in children's experiences is accounted for by genetic versus environmental factors. When putative environments (e.g., the disciplinary environment to which a child is exposed) are found to be at least moderately heritable, this is interpreted as evidence of *r*GE. Most of the risk exposures that psychologists and psychiatrists typically consider to be features of the environment have been shown to be at least moderately heritable (Kendler & Baker, 2007).

Several research groups noted that the pathway from genes to environments must involve behavior (Jaffee & Price, 2007; Rutter et al., 2006; Turkheimer & Gottesman, 1996). That is, there are no genes for environments per se; rather, genes give rise to behaviors and abilities, and these in turn shape a person's environment. Many of the papers included in this section contribute to our understanding of which behaviors account for the heritability of specific environments. For example, Boivin and colleagues (2013) show that genetic influences on children's disruptive behaviors in the early school years are substantially shared with genetic influences on their difficult relationships with peers. Similarly, Wilkinson, Trzaskowski, Haworth, and Eley (2013) identify genetic correlations between children's depressive symptoms and both child and parent reports of various features of the family environment. Using real-time observational data, Klahr, Thomas, Hopwood, Klump, and Burt (2013) show that mothers often respond to their children's bids for agency (in the context of a joint task) by relinquishing control and that evocative *r*GEs underlie these child effects on parental behavior. Finally, Hicks and colleagues (2013) show that personality characteristics, such as a willingness to follow rules, influence the degree to which adolescents are exposed to risky environments involving peers, family, and school, with this pathway resulting from active *r*GEs that ultimately give rise to risk for substance abuse in late adolescence. Similarly, Neiderhiser, Marceau, and Reiss (2013) identify a common genetic factor that accounts for the covariation of risk factors along a pathway to adolescent drug use.

Biometric models are powerful tools for estimating the genetic influences on putative environments and the degree to which genetic influences on behavior or abilities account for heritable variation in putative environments. However, such designs do not indicate which genes are involved in *r*GE. A more direct indication of *r*GE can come from molecular genetic studies using measured genes and measured environments. This relatively novel approach looks for associations between a genotype, a phenotype, and a measured environmental variable (e.g., Burt, 2008). In the current collection of papers, evidence emerges for specific genes that evoke the parenting that children receive, including serotonergic genes (Pener-Tessler et al., 2013), dopaminergic genes, and genes involved in dopamine signaling (Hayden et al., 2013; Oppenheimer, Hankin, Jenness, Young, & Smolen, 2013). Like the quantitative behavioral genetic studies included in the Special Section, these studies of measured genotype also identify behaviors that mediate the pathway from genes to environments, with children's negativity (Hayden et al., 2013) and lack of self-control (Pener-Tessler et al., 2013) partially accounting for the associations between child genotype and parenting behavior.

Conclusions and Future Directions

The current evidence for *r*GE leaves many unanswered questions. First, most studies in the Special Section showed meaningful environmentally mediated effects, as well as *r*GEs. Thus, although *r*GEs are pervasive, they do not preclude the possibility that environmental risk exposures have causal effects on risk for psychopathology. Research that uses genetically sensitive and quasiexperimental designs can be highly informative about the magnitude of associations between risk exposures and outcomes in the absence of familial (and other forms of) confounding. From an intervention standpoint, it is crucial to know whether exposures have causal or noncausal effects and how large those effects are likely to be.

These papers also allude to the complexity of modeling gene–environment interplay over time and across contexts. To the extent that "environments" are actually behaviors (e.g., harsh parenting), gene–"environment" correlations will be as difficult to detect as gene–disorder associations have been in the psychiatric genetics literature where main effects of genes have been notoriously elusive. Like gene–disorder relationships, the magnitude of *r*GEs may depend on other factors. This is demonstrated in the paper by Oppenheimer et al. (2013), where the significance of the association between genotype and parenting behavior varied as a function of parent personality traits.

Not only does the magnitude of *r*GEs vary across contexts, but it also varies across development, with reciprocal interac-

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tions between persons and environments generating ever-increasing differentiation of behavior, health, and abilities both within families and within populations (Beam & Turkheimer, 2013). At a bare minimum, longitudinal data are required to model the complexity of these reciprocal effects, but empirically based theory is needed to inform choices about measurement spacing and what, specifically, to measure. For example, during certain developmental periods when children are undergoing rapid changes, dense measurement of phenotypes and environments may be required to adequately capture reciprocal effects, whereas less frequent measurement may suffice in other developmental periods. Moreover, the knowledge that development is characterized by heterotypic and homotypic continuity poses challenges for models of reciprocal relations between phenotypes and environments. Imagine, for example, that a researcher is interested in how relatively small individual differences in early childhood temperament lead to much larger individual differences in adolescent antisocial behavior. Reciprocal interactions may involve different phenotypes at different points along the developmental pathway, such as temperamental traits like hyperactivity, anger, and fear in toddlerhood, oppositional behaviors like being argumentative or hot-tempered in the preschool years, and aggressive and rule-breaking behaviors in the school years. Underlying these diverse behaviors is arguably a dimension of behavioral undercontrol, but the measurement of this latent construct must change over time in order to remain developmentally sensitive. Similarly, the environments that shape and are shaped by these behaviors will also change across development. Although highly complex, efforts to understand how person–environment correlations unfold across time will advance our understanding of the development of psychopathology.

Taking a developmental perspective, Scarr and McCartney's (1983) important suggestion that active *r*GE is more likely to appear later in development as compared to evocative *r*GE has not been followed up by systematic investigation. It maps an important direction for future developmental research into *r*GE. For example, research could compare genetic contributions to children's, adolescents' and young adults' association with antisocial peers, or link individuals' genes to the personality and psychopathology of their romantic partners in different stages across the life span.

In summary, although most psychologists and psychiatrists reject false nature–nurture dichotomies, interest in gene–environment interplay has largely focused on G × E interaction as opposed to *r*GE. This Special Section was designed, in part, to draw attention to the pervasiveness of *r*GEs. The papers included in this Special Section use diverse methods and samples for modeling *r*GE, model all forms of *r*GE, and approach *r*GE from different perspectives: as a phenomenon in its own right and as a potential source of confounding in models of risk exposures and outcomes. In general, *r*GEs matter because they are pervasive in human experience and development. It is our hope that this collection of papers highlights their relevance for understanding the etiology and course of psychopathology.

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