Published in final edited form as:

J Fam Theory Rev. 2014 March 1; 6(1): 18–30. doi:10.1111/jftr.12028.

Behavioral Genetic Approaches and Family Theory

Ginger A. Moore and Jenae M. Neiderhiser

Pennsylvania State University

Abstract

Family theories have been founded on research that cannot discriminate genetic and environmental influences and, consequently, most theories do not have highly developed models of gene–environment interplay in families. Behavioral genetic approaches, which can identify gene–environment interplay, have typically not been driven by family theories and have lacked adequate measurement of family processes. In this article, the authors describe behavioral genetic mechanisms and methods using representative examples of research on family processes, highlighting the advantages of an integrated approach. New directions in research and theory driven by an integrated approach are discussed.

Traditionally, family theories have emphasized the impact of the social environment on family processes (family relationships and the impact of families on children's development) without considering the role that family members' genetic characteristics may play (e.g., Baumrind, 1973). In the 1980s, however, researchers looking at samples of family members that varied in degree of genetic relatedness (e.g., identical- and fraternal-twin pairs, adopted children and their adoptive parents) presented a challenge to family theories (e.g., Plomin & Daniels, 1987; Rowe, 1981, 1983). They pointed to research that found consistent genetic influences on family processes and to findings that adult siblings are not typically alike in ways that family theories propose are influenced by the social environment (Plomin & Daniels, 1987). If family environment is so influential, they asked, then why do siblings end up so different from each other?

Nevertheless, behavioral genetic studies have consistently found that both genetic and environmental influences contribute to measures of family processes (e.g., Kendler & Baker, 2007; Reiss, 2000), with environmental influences explaining as much variance as, or more than, genetic influences. As research has moved beyond a focus on the independent contributions of genes and environment to investigate the interplay of genes and environments that influences family processes, the tension between traditional family theories and behavioral genetics has dissipated (McGue, 1994; Wachs, 1993). To date, however, most family theories are founded on an evidence base of research on biological families, which confounds genetic and environmental influences and masks interplay among those influences. However, behavioral genetic approaches, which can discriminate genetic and environmental sources of variability and identify gene–environment interplay, have typically not been driven by specific family theories and have lacked adequate measurement of the family environment and relationships. To advance family theories and to enhance the ability to examine the complexity of family processes, conceptual and methodological innovations based on an integration of behavioral genetic and family theories are needed.

In this article, we discuss the methods and advantages of an integrative approach. First, we define and describe methods used in behavioral genetic research and theoretical mechanisms

relevant to family theories, including genotype–environment correlation (r_{GE}), genotype $\times$ environment interaction ($G \times E$), and variations such as gene–environment correlation moderated by the environment ($r_{GE} \times E$). We present representative examples of recent research on family processes using behavioral genetic approaches to illustrate the advantages of these approaches. Then, we discuss ways in which theory-based measurement of family processes can be better utilized in genetically informed research designs, using the example of a prospective adoption study with innovative and detailed measurement of relational processes occurring within family interactions. Finally, we describe future directions in research and theory suggested by an integration of behavioral genetic approaches and family theories.

Behavioral Genetic Mechanisms and Methods

Main-Effects Models

In various behavioral genetic designs, genetic and environmental influences on individual differences in measures of family relationships or the impact of those relationships on individuals in families are estimated using quantitative techniques that analyze behavioral covariation among family members who vary in degree of genetic relatedness, such as twins (Loehlin, 1989). More recently, molecular genetic techniques have been used to analyze covariation between behaviors and specific genes (e.g., Moffitt, Caspi, & Rutter, 2005). Because family processes are likely to involve highly complex interactions among multiple genes, and because most behavioral genetic research to date that is relevant to family theories has used quantitative techniques, this article focuses primarily on quantitative research. We discuss molecular genetic approaches, particularly as they are being advanced and informed by family theories, primarily in terms of future directions.

Twin studies are commonly used to disentangle genetic and environmental influences because twins vary in regard to shared genes; identical (monozygotic, MZ) twins share 100% of their genes, and fraternal (dizygotic, DZ) twins share 50%, on average; and when reared together, twins also share environments. In general, genetic influence is indicated if MZ twins are more similar than DZ twins (Plomin, 1994). Adoption designs are also commonly used to differentiate genetic and environmental influences. For example, if adopted children are similar to their adoptive relatives, it must be due to environmental influences, because they do not share genes. However, if adopted children are similar to their biological relatives, it must be due to genetic influences, because they do not share an environment, except to the degree that any shared genetic influences elicit similar environments (see discussion of r_{GE} later). These findings indicate heritability, or variability in a particular trait that can be attributed to genetic factors.

Behavioral genetics makes a distinction between shared environment, which indicates environmental influences that contribute to similarity among relatives, and nonshared environment, which indicates environmental factors that contribute to differences among relatives (McGuire & Dunn, 1994). Model-fitting techniques are used to test the fit of a hypothesized model of genetic and shared and nonshared environmental influences against observed familial correlations. In general, when genetic influences on family processes are identified, it does not indicate a direct influence of the individual's genes. Instead, genes influence family processes through interplay between genetic and environmental influences, processes involving gene–environment correlation and gene–environment interaction.

Gene–Environment Correlation Within the Family

Gene–environment correlation (r_{GE}) refers to individuals' heritable characteristics being correlated with an aspect of family environment (e.g., parenting), with the nature of the r_{GE}

—passive, evocative, or active—indicating different effects on family processes (Plomin, DeFries, & Loehlin, 1977). Because most families consist of parents rearing their biological children, *r*GE is the norm. Passive *r*GE is the result of a parent and child sharing both genes and environments (e.g., Kendler, 1996) and suggests no effective influence. For example, a child may inherit a tendency toward negative emotionality from a parent and be treated negatively by that parent because of the parent's heritable negative emotionality. In this example of passive *r*GE, it is not parenting that explains the child's negative emotionality, although many family theories would interpret (and likely have interpreted) this type of association as reflecting parenting influence.

Evocative *r*GE occurs when a child's heritable characteristics evoke a particular response from the social environment (e.g., McGue, Elkins, Walden, & Iacono, 2005; Plomin, McClearn, Pederson, Nesselroade, & Bergeman, 1989). For example, a child who inherits a tendency toward negative emotionality may evoke more negative responses from his or her parent than would a child with a more easygoing temperament. Again, although some family theories may interpret the association between child emotionality and negative parenting as the influence of parenting, in this case the effect would be from child to parent.

Of note, passive and evocative *r*GE can co-occur, as can environmental influences and evocative *r*GE or all three. Because genes and family environment are typically correlated (most children are reared by biological parents), *r*GE often shapes development in the direction of an individual's genotype (Plomin, 1994). A child with an inherited negative emotionality is likely to elicit negative parenting, especially from a parent who also has genetically influenced negative emotionality, thus reinforcing the child's heritable negative emotionality through both *r*GE and environmental (parenting) effects. In addition, as a function of active *r*GE (Scarr & McCartney, 1983), which results when an individual seeks out environments that are associated with his or her heritable characteristics, the child may select peers who are more aggressive and behavior disordered, thereby further reinforcing the child's negative emotionality and increasing negative relationships in the family. Note that although these examples are based on parent-child relationships, the processes could function in sibling relationships as well.

The simultaneous effects of multiple genetic and environmental influences reflect the inherent complexity in family processes that presents an intractable challenge to family theories. Research designs that do not include behavioral genetic approaches are almost always unable to differentiate the nature of the multiple influences on family processes, although understanding the nature and direction of those influences is critical for family theorists, researchers, and interventionists (Leve, Harold et al., 2010a). A number of behavioral genetic approaches (discussed subsequently) have been developed that can help differentiate these processes and, thereby, enhance and advance family theories.

In a child-based twin design, children vary in genetic relatedness, and children's genes are the unit of measurement. For example, genetic influences on measures of parent-child relationships in a child-based design suggest that children's heritable characteristics influence the nature of that relationship (evocative *r*GE). In a parent-based twin design, parents vary in genetic relatedness, and parents' genes are the unit of measurement. Genetic influences on measures of parent-child relationships in a parent-based design suggest that parents' heritable characteristics, not child behavior, are influencing the nature of that relationship (passive *r*GE). Shared or nonshared environmental influences, or both, on the same parent-child relationship measure in a parent-based design also suggest that evocative *r*GE may be operating, because a child's heritable characteristics influence the types of behaviors evoked from the parenting environment, independent of the parents' genetic characteristics.

Examining *rGE* and distinguishing types of *rGE* are key to understanding family processes. Comparing child- and parent-based studies can elucidate whether evocative or passive *rGE* is operating or both, although a sample that integrates child- and parent-based designs affords stronger evidence (see the Extended Children of Twins model discussed later). For example, a set of twin studies examining relations between mothers' and adolescents' behaviors (Neiderhiser et al., 2004; Neiderhiser, Reiss, Lichtenstein, Spotts, & Ganiban, 2007) found maternal positivity toward their children to be a heritable characteristic of mothers (passive *rGE*) but maternal control and negativity to be in response to adolescents' heritable behaviors (evocative *rGE*). Some evidence indicated that both passive and evocative *rGE* were occurring simultaneously for mothers' negativity. To accommodate these findings, family theories need not only to account for gene–environment interplay but also to explain why the interplay might occur differently for different types of parenting behavior.

Bivariate models with child-based designs have been used to assess the extent to which genetic and environmental influences contributing to one variable also influence a second variable. Figure 1 illustrates a bivariate model examining parent–child conflict and child behavior in a child-based twin design. In longitudinal designs (the same variables measured at two different times), this type of analysis can help to specify direction of effects. For example, parent–child conflict and child externalizing behaviors have been found to independently predict each other 3 years later (Burt, Krueger, McGue, & Iacono, 2003), with parent–child conflict having a direct effect on children's externalizing behaviors (environmental effect), and externalizing behaviors having a genetic effect on parent–child conflict through evocative *rGE*. Results from adoption designs support these findings. Ge et al. (1996) found evocative *rGE* in a sample of adopted adolescents selected for genetic risk on the basis of biological parents' substance abuse and/or dependency or antisocial personality disorder. The *rGE* between biological parents' psychiatric disorders and adoptive parents' hostility and harsh, inconsistent discipline toward adopted children was mediated by child antisocial and/or hostile behaviors, which indicates that genetic characteristics of the children's behavior elicited increased negative parenting from their adoptive parents. Note that passive *rGE* is not possible in an adoption design because adopted children do not share genes with their adoptive parents.

In general, then, behavioral genetic approaches have shown that genetic influences explain a large proportion of the correlation between measures of the family environment and children's outcomes (e.g., Burt et al., 2003; Reiss, Neiderhiser, Hetherington, & Plomin, 2000), thus indicating the important role of evocative *rGE* in shaping the parent–child relationship and its impact on child development. Thus, although this research from a behavioral genetic perspective supports family theories such as Patterson's (1982) classic social learning model of coercive cycles, it also highlights the need for family theories to recognize a child's heritable characteristics when understanding how these cycles develop and to explain why these characteristics might evoke certain responses in some parents but not others.

Behavioral genetic designs also provide causal evidence for the negative effects of family environmental variables, independent of any heritable child characteristics. In a 2-year longitudinal study (Jaffee et al., 2004), parental physical maltreatment predicted child antisocial behavior and was not a function of genetic influences, which clearly indicates that physical maltreatment contributes to the development of children's problem behavior above and beyond heritable risk characteristics that children may have. The compelling evidence that behavioral genetic research such as this provides can not only enhance theory but also influence public policy regarding prevention of maltreatment.

A powerful approach for distinguishing passive and evocative *r*GE as mechanisms through which family environment is linked to measures of children's adjustment is the children-of-twins (CoT) design (Rutter, Pickles, Murray, & Eaves, 2001; Silberg & Eaves, 2004). The CoT design includes pairs of adult co-twins and their children. The children of co-twin parents are reared in different families, and therefore presumably do not share a common parenting environment. A child of an MZ twin shares 50% of his genes with his parent, 50% with his parent's co-twin (the child's aunt or uncle), and 25% with his cousin. Conversely, a child of a DZ twin shares 50% of genes with her parent, 25% with her parent's co-twin (like any niece or nephew), and 12% with her cousin. This design can distinguish direct environmental influences of family relationships, passive *r*GE, and those unmeasured shared environmental factors making the parent co-twins similar (D'Onofrio et al., 2003; Rutter et al., 2001). A higher correlation between children of MZ twins and their aunts and uncles than between DZ twins and their aunts and uncles would indicate genetic effects on the relationship between parenting and child behavior, but because aunts and uncles do not parent the nieces and nephews, the correlation between parenting and child behavior can be interpreted as passive, not evocative, *r*GE.

Research using a CoT design has been used to differentiate passive *r*GE from the environmental influence of marital conflict on child adjustment. Although most family theories about the effects of marital conflict propose that parenting mediates the relation between family conflict and children's negative outcomes and most empirical research supports this spillover effect (Erel & Burman, 1995), the bulk of this research has been conducted with biological families, which cannot rule out passive *r*GE. However, Harden et al. (2007) found that the children of MZ twins who were differentially exposed to varying levels of marital conflict had similar rates of antisocial behaviors, whereas the children of DZ twins who were differentially exposed to varying levels of marital conflict differed in level of antisocial behavior. These findings suggest that passive *r*GE may explain the commonly found association between marital conflict and antisocial behaviors in children, which is in contrast to most family theories of the impact of marital conflict on children's outcomes.

Behavioral genetic approaches emphasize the need to modify theory in this important line of research to include gene–environment interplay when conceptualizing family processes. Few family theories regarding the impact of marital conflict on children's development have incorporated parents' and children's heritable characteristics into their models. An exception is a recent study of the role of mothers' genotypes in moderating the spillover between marital conflict and parenting (Sturge-Apple, Cicchetti, Davies, & Suor, 2012), discussed in the section on gene–environment interaction.

A novel extension of the CoT design, the Extended Children of Twins (ECoT) design, combines samples of twins who are parents and their children and samples of children who are twins and their parent(s). This design combines the information provided by the traditional child-based twin design with that of the CoT design described earlier (see Figure 2). As such, reciprocal paths between parenting and child adjustment can be examined, and the genetic and environmental contributions to those reciprocal effects can be estimated.

Researchers applied this model in two investigations of the relations between parenting and child adjustment. In one study, maternal emotional overinvolvement was found to be a response that is evoked from children's heritable internalizing behaviors, evocative *r*GE, not passive *r*GE (Narusyte, Neiderhiser, D'Onofrio, Reiss, & Spotts, 2008). Similarly, in another study (Narusyte et al., 2011), mothers' criticism of their children was found to be primarily due to evocative *r*GE evoked by their adolescent's externalizing behaviors. Fathers' criticism, however, affected adolescent externalizing behaviors directly

(environmental influence). Again, these findings emphasize the need to incorporate the role of children's heritable characteristics into theories of parental influence and to generate theories that can explain differential processes for mothers' and fathers' parenting within families.

Gene–Environment Interaction Within the Family

Gene–environment interaction ($G \times E$) occurs when individuals with a particular genotype respond differently (in a systematic way) to the same environmental conditions than individuals who do not have the genotype (Plomin et al., 1977). The result is that children with different genotypes experience unique effects in the same environments. For example, a child with a heritable characteristic associated with externalizing behaviors may develop behavior problems when exposed to harsh, negative parenting, whereas a child who does not have that heritable characteristic may not be affected in the same way.

An important implication of findings to date from $G \times E$ studies is that family theories that propose a one-size-fits-all approach are obsolete and need to be modified to take into account children's heritable characteristics. For instance, most parenting theories propose that sensitive parenting is critical for children's overall development (e.g., Baumrind, 1973; De Wolff & van IJzendoorn, 1997). However, a study of biological mothers and their infants using a molecular genetics approach that measured specific genotypes using salivary DNA found that only children with a genotype associated with problems with self-control benefited from maternal sensitivity. Other children, even those with insensitive parenting, developed typically (Propper et al., 2008). Similarly, many family theories propose that structured parenting is optimal in the toddler years to help children develop appropriate self-regulatory skills (e.g., Denham et al., 2000). However, a longitudinal study of adoptive parents' structured parenting and adopted toddler's behavioral adjustment found that the effects of structured parenting on toddler behavior problems varied as a function of children's genetic risk for psychopathology (Leve et al., 2009). Specifically, structured parenting predicted fewer behavior problems for toddlers whose birth parents scored higher on measures of psychopathology (indexing high genetic risk) but was related to greater behavior problems for those toddlers at low genetic risk. The authors suggested that for children without genetic susceptibility, high levels of parenting structure could interfere with the development of self-control, thus encouraging children to rely on external regulation. For children who do have a genetic susceptibility, high levels of structure may scaffold their behavior so that they are able to attend to and engage their environments in more organized and productive ways. This is consistent with a differential susceptibility model, which states that some individuals are more susceptible to environmental influences in positive as well as negative directions as a result of so-called plasticity genes (Belsky & Pluess, 2009).

Parenting has also been found to moderate genetic influences on child adjustment at later ages. One behavioral genetic investigation assessed whether parental negativity and warmth moderated genetic and environmental influences on adolescent antisocial behavior and depressive symptoms (Feinberg, Button, Neiderhiser, Reiss, & Hetherington, 2007) and found that genetic influences on child antisocial behavior were enhanced when parenting behaviors were more negative or less warm. This indicates that genetic effects have a stronger influence on antisocial behavior in the presence of parental negativity and lack of warmth. In contrast, nonshared environmental effects on adolescents' depressive symptoms increased in the context of greater parental negativity. As such, experiences of parental negativity unique to each sibling may take on an increasingly important role as the degree of parent negativity increases. These findings indicate that parenting theories need to be modified to understand the unique mechanisms that may operate for the same parenting behavior on different children within families and different types of child outcomes.

There are many other examples of findings from twin and adoption studies focused on $G \times E$ effects that we briefly describe here. Natsuaki et al. (2010) reported that among children who had birth mothers with major depressive disorder, only those whose adoptive mothers were judged to be low in responsiveness increased in fussiness between 9 and 18 months old. This suggests a differential susceptibility to parenting responsiveness and is consistent with Propper et al.'s (2008) findings that only infants with a genetic susceptibility and insensitive mothers showed atypical patterns of physiological regulation during a stressful task. Leve, Kerr et al. (2010b) found that adopted infants with a genetic susceptibility for externalizing behaviors, based on their birth mothers' levels of externalizing behaviors, showed heightened attention to a frustration task (a known precursor to externalizing problems) only when they had adoptive mothers who were high in anxious and depressive symptoms. These findings suggest that mothers with high levels of anxious and depressive symptoms either parent in a way that only children with a genetic susceptibility are sensitive to or respond to children with a genetic susceptibility for externalizing behaviors in a way that reinforces those behaviors. Although the specific ways that children with a genetic susceptibility responded and the specific ways that adoptive parents with anxious and depressive symptoms responded to their children were unmeasured in these studies, additional work from our program of research suggests that both mechanisms may be involved (discussed later). Identifying the specific parenting mechanisms that contribute to children's externalizing behaviors is critical for designing effective interventions.

Most research on $G \times E$ has focused on children's differential sensitivity to parenting environments. However, parents' heritable characteristics may also result in differential sensitivity to their environments. As discussed already, a study on the spillover of marital conflict onto parenting using a molecular genetics approach found that mothers with a variation of the serotonin transporter (5-HTT) gene associated with risk for depression were differentially susceptible to the quality of marital relationships (Sturge-Apple et al., 2012). Mothers with the genotype and supportive marriages showed more sensitive parenting than other mothers, and mothers with the genotype and higher levels of marital conflict showed more harsh, insensitive parenting than other mothers. This research suggests that marital conflict does not affect all parents in a similar way and that the effects of marital conflict on parenting may be a function of parents' own heritable characteristics.

Despite the important new directions that are afforded by employing behavioral genetic approaches, more research is needed to identify the specific heritable and environmental factors that are involved in family processes. In addition, research that can model relational processes between and among individuals within families with a behavioral genetic design is critical to advancing family theories. In other words, findings to date have provided consistent evidence to suggest that family environment can moderate genetic influences on family relationships, and vice versa; research is needed now that can investigate precisely how. This is particularly important in order to translate the findings from basic research to practices that can help children and families. In the next section, we describe an innovative prospective adoption study that has incorporated detailed measurement of family interactions based on relational theories of family processes within a behavioral genetic design.

Integrating Behavioral Genetic and Family Theory Approaches

The Early Growth and Development Study (EGDS), which is composed of two cohorts, is a prospective adoption study examining gene–environment interplay from infancy through middle childhood (Leve, Neiderhiser, Scaramella, & Reiss, 2008). The EGDS design includes 561 linked sets of participants: children adopted within the first 3 months of life (mean 3 days), adoptive mothers (AMs) and adoptive fathers (AFs), and the children's birth

mothers (BMs) and/or birth father (BFs). The EGDS has the unique potential to identify differential and interactive effects of genetic, prenatal, and postnatal environmental influences on children's development because of its longitudinal adoption design; detailed assessment of birth parents and the prenatal environment; and extensive measures of family environment, including videotaped observations of adoptive families at child age 9, 18, and 27 months, and in Cohort 1 also at ages 4.5, 6, 7, and 8 years of age, and in Cohort 2 also at age 8 years.

The measurement of family environment in the EGDS is unique among behavioral genetic studies and was based on a theoretical model that emphasizes family systems principles and relational interactive processes. The theoretical model views parenting as an active relational process that is cocreated by the parent and child over time and is a function of mechanisms of evocative rGE and $G \times E$. Furthermore, the model proposes that the parent-child relationship is affected by and affects other emotional relational systems within the family, including the marital relationship. Using detailed microcoding of family interactions, the study measures family processes at a fine-grained level of analysis that allows for modeling of contingencies between parent and child behaviors at a real-time level (e.g., second by second across the course of an interaction). Although microcoding methods have been used profitably in family research based on parents rearing their biological children (e.g., Shaw et al., 1998), extending the use of microcoding methods to genetically informed designs is a powerful and innovative approach to understanding how parenting can modify biologically based influences on children's development.

An example of the many mechanisms that can be modeled by this approach is assessment of the degree of influence that individual family members have on one another's behaviors, with a very low degree of mutual influence suggesting emotional distance or cutoff and a high degree suggesting enmeshment, and an imbalance, by which one individual is influenced by the other to a much greater or lesser degree, thus suggesting a hierarchical relationship. Furthermore, we can assess whether these relational processes are moderated by aspects of the family environment (e.g., parent depression, marital conflict). Consistent with this, in the EGDS, we measured contingent responsiveness between parent and child positive and negative behaviors using dynamic factor models (see Molenaar, 1985). These models account for how one partner's behavior in the previous second influences the other partner's behavior in the next second. Analyses found a transactional relationship between the degree to which a parent's negative behavior predicted a child's negative behavior in the next second as a function of maternal depressive symptoms (Pemberton-Roben et al., 2012). Maternal depressive symptoms at 9 months predicted a greater likelihood of a child's immediate negative response to mothers' behavior at 18 months. Furthermore, higher rates of children's negative responses to mothers' behavior at 18 months predicted higher levels of maternal depressive symptoms at 27 months, thus indicating a mutual negative influence over time. This interactive process was not evident when using a global parent report of parenting (Arnold, O'Leary, Wolf, & Acker, 1993), which is not surprising, as global ratings do not describe parenting behavior at the level at which we can observe an influence on the child's behavior.

The importance of this finding should not be underestimated. Despite a large correlational literature based on biological families that links maternal depression with problematic outcomes for children, the field has been unable to support causal conclusions regarding the impact of depression on parenting and, consequently, on children. These and various other sophisticated analytic approaches have been developed for studying dyadic interaction (Kenny, Kashy, & Cook, 2006) and promise to inform current family theories. However, a significant advance occurs when integrating these analytical methods within a behavioral genetic design. Because the EGDS is a longitudinal adoption design that can control for

genetic influences, the relation between parent and child behaviors and maternal depressive symptoms over time indicates that the association is due to interactions in the environment rather than to passive rGE , something that cannot be distinguished in research with biological families. Our work continues to examine more complex models of family processes. For example, it is possible that only mothers with high levels of depressive symptoms respond to children's heritable characteristics (i.e., evocative $rGE \times E$) in a way that elicits negative cycles of behavior between them. This might occur in the same way that the parenting of only mothers with a genetic risk for depression was differentially susceptible to the quality of marital relationships (Sturge-Apple et al., 2012).

There are myriad ways that genetic and environmental influences could work together, and the various types of rGE can occur along with $G \times E$, and correlation between genes and environments may operate in different ways as a function of particular environments ($rGE \times E$) or as a function of particular genes ($rGE \times G$). New directions in research on these mechanisms is providing exciting empirical support for theoretical models and expanding the knowledge base to inform the next generation of family theories. An example of these new directions comes from the EGDS examination of adoptive parents' overreactive parenting (Hajal et al., 2012). Results indicated that adoptive fathers' (but not mothers') overreactive parenting responses to infants' difficult behavior was predicted by genetic influences on infant sociability (indexed by birth mothers' sociability), thus indicating evocative rGE . This was found only in families with high levels of marital warmth, which indicates an environmental moderation of evocative rGE ($rGE \times E$). This means that when marital relationships are warm and supportive and infants are being challenging (e.g., crying), fathers appear to respond to their infants in a way that is influenced by how positive and sociable their infants usually are. For adoptive mothers, overreactive parenting was linked to aspects of infant sociability that were unrelated to genetic influences (at least those indexed by birth mother personality), and the mother's parenting was not influenced by the quality of the marital relationship, which is consistent with previous research. This level of complexity can inform family theories and expand conceptualizations of interrelations among family members to model the family as an emotional system and to increase the field's understanding of how heritable characteristics of individual family members contribute to that emotional system.

New Directions in Theory and Research

As evidence builds from behavioral genetic research that family processes are a complex and dynamic interplay of genetic and environmental influences, family theories need to keep pace with the rapidly growing knowledge base. Whether a family theory is focused on the family as a system, the family in the context of society, how individual members develop in the context of the family, or relationships among individuals within families, it is reasonable and advantageous to integrate genetic influences into theoretical models (Reiss, 2010).

Theory building in family research has typically been more deductive than inductive. Because family theories have traditionally been tested in research with biological families, however, there has been inertia in the ability to accommodate to the growing empirical evidence from behavioral genetic research. This inertia may be exacerbated by the pervasive practice in social science research to not publish null findings (Ferguson & Heene, 2012). This can impede progress and contribute to a reification of theories, or what some researchers have called "undead theories that are ideologically popular but have little basis in fact" (Ferguson & Heene, 2012, p. 555).

In contrast, behavioral genetic research, which has tended to be more inductive in its approach, has profited from incorporating family theory and has become more deductive as

it actively tests existing theories using unique research designs. The empirical findings from this body of work present opportunities to revise family theories in significant, fresh, and exciting ways that may capture the complexity inherent in family processes. To respond to these challenges, family researchers do not necessarily have to adopt behavioral genetics research methods, but they may adapt design and data analytic methodologies to be more consistent with within-person approaches (e.g., Von Eye & Bergman, 2003) and to examine and distinguish relational and independent influences on behavior and biology (e.g., Slatcher, Mehta, & Josephs, 2011).

Thus, an integration of behavioral genetic approaches and family theory is strengthening and advancing both fields by the exchange of empirical findings and theory-building approaches, and by methodological innovations that have been developed in response to theoretical advances and that, in turn, have helped drive theoretical advances.

The integration is mutually profitable. The field of behavioral genetics has borrowed from family theories to progress from an emphasis on broad quantitative estimates of heritability and environmental influences to investigations of complex family processes to exciting new work examining the influence of environmental factors on genetic mechanisms. Whereas past research has conceptualized genetic effects as static influences in gene–environment interplay, research on epigenetics, which refers to functional modifications of gene expression caused by mechanisms other than changes in the underlying DNA, is investigating the environmental regulation of gene expression. A growing body of research has found that genes and environments operate in dynamic feedback systems to shape the expression of specific genes that govern health and behavior. For example, studies have found variations in the promoter regulating the serotonin transporter gene (5HTT), which results in buffering the effects of adverse family environments (Caspi et al., 2003; Champoux et al., 2002). Other work has found that older adults who reported feeling lonely showed different levels of gene expression in specific groups of genes related to immune system functioning, which suggests that perception of social support can modulate gene activity that supports physical health (Cole et al., 2007). Recent work in molecular genetics has found genetic commonalities in five major psychiatric disorders (i.e., major depressive disorder, bipolar disorder, schizophrenia, autism spectrum disorders, and attention-deficit/hyperactivity disorder) (Cross-Disorder Group of the Psychiatric Genomics Consortium, 2013), thus paving the way for family researchers to identify environmental mechanisms that may explain how the genetic commonalities develop into distinct disorders.

Advances in theory and research ideally translate to clinical practice and health policies. Reiss (2011) proposed key principles of change indicated by the findings of behavioral genetic research. Given broad and consistent evidence that genetic mechanisms are implicated in mental health disorders and family processes, Reiss (2011) proposed that practitioners consider the parent–child dyad the unit of assessment and intervention. Reiss proposed that if a child is found to have psychological and behavioral problems, then parents be assessed. Similarly, if parents are found to have psychological problems, practitioners should find ways to work collaboratively with parents to assess their children’s adjustment. Furthermore, individualized evidence-based interventions should be modified appropriately to take into account the need for integrated care within families.

Acceptance of these clinical practice recommendations would require shifts in public perception regarding the role of parenting in children’s development. Reiss (2011) based his proposals on the principle that disorders are likely to run in families because of gene–environment interplay, not because of “bad” parenting. However, general public beliefs about how parenting influences children’s development is oversimplified relative to current scientific knowledge, and this oversimplification is sometimes, unfortunately, promoted by

widespread family theories. Altering public perception of the complexities of parenting influence will be critical to change at the level of clinical practice and health policy. Academic researchers who study and develop family theories may play a key role in promoting this change by educating new generations of researchers and practitioners in the integration of behavioral genetic approaches and contributing to continued progress in family research.

Acknowledgments

The Early Growth and Development Study was supported by R01HD042608 (Eunice Kennedy Shriver National Institute of Child Health, National Institute on Drug Abuse, and Office of the Director) and R01DA020585 (National Institute on Drug Abuse, National Institute of Mental Health, and Office of the Director).

References

- Arnold DS, O'Leary SG, Wolf LS, Acker MM. The Parenting Scale: A measure of dysfunctional parenting in discipline situations. *Psychological Assessment*. 1993; 9:137–144.
- Baumrind, D. The development of instrumental competence through socialization. In: Pick, A., editor. *Minnesota symposia on child psychology*. Vol. 7. Minneapolis, MN: University of Minnesota Press; 1973. p. 3–46.
- Belsky J, Pluess M. Beyond diathesis stress: Differential susceptibility to environmental influences. *Psychological Bulletin*. 2009; 135:885–908. [PubMed: 19883141]
- Burt SA, Krueger RF, McGue M, Iacono W. Parent–child conflict and the comorbidity among childhood externalizing disorders. *Archives of General Psychiatry*. 2003; 60:505–513. [PubMed: 12742872]
- Caspi A, Sugden K, Moffitt TE, Taylor A, Craig IW, Harrington H, Poulton R. Influence of life stress on depression: Moderation by a polymorphism in the 5-HTT gene. *Science*. 2003; 301:386–389. [PubMed: 12869766]
- Champoux M, Bennett A, Shannon C, Higley JD, Lesch KP, Suomi SJ. Serotonin transporter gene polymorphism, differential early rearing, and behavior in rhesus monkey neonates. *Molecular Psychiatry*. 2002; 7:1058–1063. [PubMed: 12476320]
- Cole SW, Hawkey LC, Arevalo JM, Sung CY, Rose RM, Cacioppo JT. Social regulation of gene expression in human leukocytes. *Genome Biology*. 2007; 8:R189. [PubMed: 17854483]
- Cross-Disorder Group of the Psychiatric Genomics Consortium. Identification of risk loci with shared effects on five major psychiatric disorders: A genome-wide analysis. *Lancet*. 2013; 381:1371–1379. 10.1016/S0140-6736(12)62129-1 [PubMed: 23453885]
- Denham SA, Workman E, Cole PM, Weissbrod C, Kendziora KT, Zahn-Waxler C. Prediction of externalizing behavior problems from early to middle childhood: The role of parental socialization and emotion expression. *Developmental Psychopathology*. 2000; 12:23–45.
- De Wolff M, van IJzendoorn MH. Sensitivity and attachment: A meta-analysis on parental antecedents of infant attachment. *Child Development*. 1997; 68:571–591. [PubMed: 9306636]
- D'Onofrio BM, Turkheimer EN, Eaves LJ, Corey LA, Berg K, Solaas MH, Emery RE. The role of the children of twins design in elucidating causal relations between parent characteristics and child outcomes. *Journal of Child Psychology and Psychiatry*. 2003; 44:1130–1144. [PubMed: 14626455]
- Erel O, Burman B. Interrelatedness of marital relations and parent–child relations: A meta-analytic review. *Psychological Bulletin*. 1995; 118:108–132. [PubMed: 7644602]
- Feinberg ME, Button TMM, Neiderhiser JM, Reiss D, Hetherington EM. Parenting and adolescent antisocial behavior and depression: Evidence of genotype × parenting environment interaction. *Archives of General Psychiatry*. 2007; 64:457–465. [PubMed: 17404122]
- Ferguson CJ, Heene M. A vast graveyard of undead theories: Publication bias and psychological science's aversion to the null. *Perspectives on Psychological Science*. 2012; 7:555–561.

- Ge X, Conger RD, Cadoret RJ, Neiderhiser JM, Yates W, Troughton E, Stewart M. The developmental interface between nature and nurture: A mutual influence model of child antisocial behavior and parent behaviors. *Developmental Psychology*. 1996; 32:574–589.
- Hajal, NJ.; Neiderhiser, JM.; Moore, GA.; Leve, LD.; Shaw, DS.; Scaramella, LV.; Reiss, D. Angry responses to infant challenges: Parent, marital, and child genetic factors associated with harsh parenting. 2013. Manuscript submitted for publication
- Harden KP, Turkheimer E, Emery RE, D'Onofrio BM, Slutske WS, Heath AC, Martin NG. Marital conflict and conduct problems in children of twins. *Child Development*. 2007; 78:1–18. [PubMed: 17328690]
- Jaffee SR, Caspi A, Moffitt TE, Polo-Tomas M, Price TS, Taylor A. The limits of child effects: Evidence for genetically mediated child effects on corporal punishment but not on physical maltreatment. *Developmental Psychology*. 2004; 40:1047–1058. [PubMed: 15535755]
- Kendler KS. Parenting: A genetic-epidemiological perspective. *American Journal of Psychiatry*. 1996; 153:11–20. [PubMed: 8540566]
- Kendler KS, Baker JH. Genetic influences on measures of the environment: A systematic review. *Psychological Medicine*. 2007; 37:615–626. [PubMed: 17176502]
- Kenny, DA.; Kashy, DA.; Cook, WL. Dyadic data analysis. New York, NY: Guilford Press; 2006.
- Leve LD, Harold GT, Ge X, Neiderhiser JM, Patterson G. Refining intervention targets in family-based research: Lessons from quantitative behavioral genetics. *Perspectives on Psychological Science*. 2010a; 5:516–526. [PubMed: 21188273]
- Leve LD, Harold GT, Ge X, Neiderhiser JM, Shaw D, Scaramella LV, Reiss D. Structured parenting of toddlers at high versus low genetic risk: Two pathways to child problems. *Journal of the American Academy of Child and Adolescent Psychiatry*. 2009; 48:1102–1109. [PubMed: 19797981]
- Leve LD, Kerr DC, Shaw D, Ge X, Neiderhiser JM, Scaramella LV, Reiss D. Infant pathways to externalizing behavior: Evidence of genotype $\times$ environment interaction. *Child Development*. 2010b; 81:340–356. [PubMed: 20331671]
- Leve LD, Neiderhiser JM, Scaramella LV, Reiss D. The Early Growth and Development Study: Using the prospective adoption design to examine genotype–environment interplay. *Xin Li Xue Bao Acta Psychologica Sinica*. 2008; 40:1106–1115. [PubMed: 19458782]
- Loehlin JC. Partitioning environmental and genetic contributions to behavioral development. *American Psychologist*. 1989; 44:1285–1292. [PubMed: 2679255]
- McGue, M. Why developmental psychology should find room for behavioral genetics. In: Nelson, CA., editor. Minnesota symposia on child psychology: Threats to optimal development. Vol. 27. Hillsdale, NJ: Erlbaum; 1994. p. 105–119.
- McGue M, Elkins I, Walden B, Iacono WG. Perceptions of the parent–adolescent relationship: A longitudinal investigation. *Developmental Psychology*. 2005; 41:971–984. [PubMed: 16351340]
- McGuire, S.; Dunn, J. Nonshared environment in middle childhood. In: DeFries, JC.; Plomin, R.; Fulker, DW., editors. Nature and nurture during middle childhood. Oxford, UK: Blackwell; 1994. p. 201–213.
- Moffitt TE, Caspi A, Rutter M. Strategy for investigating interactions between measured genes and measured environments. *Archives of General Psychiatry*. 2005; 62:473–481. [PubMed: 15867100]
- Molenaar PC. A dynamic factor model for the analysis of multivariate time series. *Psychometrika*. 1985; 50:181–202.
- Narusyte J, Neiderhiser JM, Andershed A, D'Onofrio BM, Reiss D, Spotts E, Lichtenstein P. Parental criticism and externalizing behavior problems in adolescents: The role of environment and genotype–environment correlation. *Journal of Abnormal Psychology*. 2011; 120:365–376. [PubMed: 21280930]
- Narusyte J, Neiderhiser JM, D'Onofrio BM, Reiss D, Spotts E. Testing different types of genotype–environment correlation: An extended children-of-twins model. *Developmental Psychology*. 2008; 44:1591–1603. [PubMed: 18999324]
- Natsuaki MN, Ge X, Leve LD, Neiderhiser JM, Shaw DS, Conger RD, Reiss D. Genetic liability, environment, and the development of fussiness in toddlers: The roles of maternal depression and parental responsiveness. *Developmental Psychology*. 2010; 46:1147–1158. [PubMed: 20822229]

- Neiderhiser JM, Reiss D, Lichtenstein P, Spotts EL, Ganiban JM. Father–adolescent relationships and the role of genotype–environment correlation. *Journal of Family Psychology*. 2007; 21:560–571. [PubMed: 18179328]
- Neiderhiser JM, Reiss D, Pedersen NL, Lichtenstein P, Spotts EL, Hansson K, Ellhammer O. Genetic and environmental influences on mothering of adolescents: A comparison of two samples. *Developmental Psychology*. 2004; 40:335–351. [PubMed: 15122961]
- Patterson, GR. Coercive family processes. Eugene, OR: Castalia; 1982.
- Plomin, R. Genetics and experience: The interplay between nature and nurture. Vol. 6. Thousand Oaks, CA: Sage; 1994.
- Plomin R, Daniels D. Why are children in the same family so different from one another? *Behavioral and Brain Sciences*. 1987; 10:1–16.
- Plomin R, DeFries JC, Loehlin JC. Genotype–environment interaction and correlation in the analysis of human behavior. *Psychological Bulletin*. 1977; 84:309–322. [PubMed: 557211]
- Plomin R, McClearn GE, Pederson NL, Nesselroade JR, Bergeman CS. Genetic influence on childhood family environment perceived retrospectively from the last half of the lifespan. *Developmental Psychology*. 1989; 24:738–745.
- Propper C, Moore GA, Mills-Koonce WR, Halpern CT, Hill-Soderlund AL, Calkins SD, Cox M. Gene–environment contributions to the development of infant vagal reactivity: The interaction of dopamine and maternal sensitivity. *Child Development*. 2008; 79:1377–1394. [PubMed: 18826531]
- Reiss D. Genetic influence on family systems: Implications for development. *Journal of Marriage and Family*. 2000; 57:543–560.
- Reiss D. Genetic thinking in the study of social relationships: Five points of entry. *Perspectives on Psychological Science*. 2010; 5:502–515.
- Reiss D. Parents and children: Linked by psychopathology but not by clinical care. *Journal of the American Academy of Child and Adolescent Psychiatry*. 2011; 50:431–434. [PubMed: 21515190]
- Reiss, D.; Neiderhiser, JM.; Hetherington, E.; Plomin, R. The relationship code: Deciphering genetic and social influences on adolescent development. Cambridge, MA: Harvard University Press; 2000.
- Roben, CKP.; Moore, GA.; Cole, PM.; Molenaar, P.; Leve, LD.; Shaw, D.; Neiderhiser, J. Transactional patterns of maternal depressive symptoms and mother–child interactions in adoptive families: The added value of observational second-by-second coding. Paper presented at the 2013 Biennial Meeting of the Society for Research in Child Development; Seattle, WA. 2013 Apr.
- Rowe DC. Environmental and genetic influences on dimensions of perceived parenting: A twin study. *Developmental Psychology*. 1981; 17:203–208.
- Rowe DC. A biometrical analysis of perceptions of family environment: A study of twins and singleton sibling kinships. *Child Development*. 1983; 54:416–423. [PubMed: 6683621]
- Rutter M, Pickles A, Murray R, Eaves L. Testing hypotheses on specific environmental causal effects on behavior. *Psychological Bulletin*. 2001; 127:291–324. [PubMed: 11393298]
- Scarr S, McCartney K. How people make their own environments: A theory of genotype–environment effects. *Child Development*. 1983; 54:424–435. [PubMed: 6683622]
- Shaw DS, Winslow EB, Owens EB, Vondra JI, Cohn JF, Bell RQ. The development of early externalizing problems among children from low-income families: A transformational perspective. *Journal of Abnormal Child Psychology*. 1998; 26:95–107. [PubMed: 9634132]
- Silberg JL, Eaves LJ. Analysing the contributions of genes and parent–child interaction to childhood behavioural and emotional problems: A model for the children of twins. *Psychological Medicine*. 2004; 34:347–356. [PubMed: 14982140]
- Slatcher RB, Mehta PH, Josephs RA. Testosterone and self-reported dominance interact to influence human mating behavior. *Social Psychological and Personality Science*. 2011; 2:531–539.
- Sturge-Apple ML, Cicchetti D, Davies PT, Suor JH. Differential susceptibility in spillover between interparental conflict and maternal parenting practices: Evidence for OXTR and 5-HTT genes. *Journal of Family Psychology*. 2012; 26:431–442. [PubMed: 22563705]

- Von Eye A, Bergman LR. Research strategies in developmental psychopathology: Dimensional identity and the person-oriented approach. *Development and Psychopathology*. 2003; 15:553–580. [PubMed: 14582932]
- Wachs, TD. The nature-nurture gap: What we have here is a failure to collaborate. In: Plomin, R.; McClearn, GE., editors. *Nature, nurture, and psychology*. Washington, DC: American Psychological Association; 1993. p. 375-391.

Figure 1.

Bivariate model in a child-based twin design (one parent, two twin children).

Note. Paths for G estimate effects of genetic influences on variability in parent–twin conflict and child behavior, paths for Es estimate effects of shared environment (environmental influences that make twins similar to each other), and paths for En estimate effects of nonshared environment (environmental influences that make twins dissimilar).

Figure 2.
 Extended children-of-twins design (ECOT).
Note. Paths m = direct effect of parenting, n = evocative *rGE*, and s = passive *rGE*.