

Blackwell Handbook of Early Childhood Development

Edited by

Kathleen McCartney and Deborah Phillips

Blackwell Handbook of Early Childhood Development

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Preface

In the first two years of life, infants develop amazing competencies across development, from controlled movements to representational thought to goal-directed attachment relationships. Our story picks up from this point through about age 7, the early childhood years. Early childhood, like infancy, represents a time of emerging skills – skills that make the 7-year-old seem more like an adult than an infant. In early childhood, children exchange magical and egocentric thinking for a theory of mind, the ability to execute a plan of action, and a rudimentary logic. Over time, “terrible” 2-year-olds become young children who can exhibit self-control by delaying gratification and inhibiting inappropriate responses. In a stunning feat, by the end of early childhood, children master most of the grammatical rules that adults use. Parents and teachers respond to these kinds of noticeable changes by using reasoning, encouraging independence, arranging play dates, and providing explicit opportunities for learning in their dealings with young children. Soon, children are reading, counting, developing friendships, choosing to engage in favorite activities, and more.

There are two main ways to study development at any age. The first is to chart the milestones that reflect changes observed in the average child, while the second is to assess individual differences among children. Both perspectives inform the knowledge base on early childhood development. Individual differences are of particular interest to those concerned with applied problems. For this reason, an understanding of early childhood necessitates an investigation of the contexts of development, what we refer to here as the social ecology of early development, including the family, peers, poverty, child care, and the media. To some extent, research on early childhood has informed policy issues, as reflected in work on assessment as well as evaluations of early interventions for children and parents. Comparative studies reveal vast differences across cultures in policies for children and families, which are more likely to reflect public will than research *per se*.

We began our editing task by reflecting on the emerging competencies in early childhood, the approaches social scientists use to study it, and methodological issues in the

field. Then we constructed topics that represent our view of the landscape in early childhood development. We recruited leading scholars in developmental science to write relatively short, albeit comprehensive, reviews of the literature from both a theoretical and a conceptual perspective. To give the volume a unified voice, we asked authors to consider four organizing themes in their reviews: the role of early experiences as they shape the course of development; contributions of the cultural contexts within which children grow up; individual differences in developmental trajectories; and applications of developmental science to issues of practice and policy.

The result is a Handbook that we have organized into seven parts: Part I, Conceptual Frameworks; Part II, Early Biological and Physiological Development; Part III, Cognitive Development; Part IV, Language and Communicative Development; Part V, Social, Emotional, and Regulatory Development; Part VI, The Social Ecology of Early Development; and Part VII, Policy Issues. The four conceptual frameworks that open the Handbook provide a foundation for the field through their discussions of how to describe the interplay of genes and environments, how research on children's vulnerability and resilience informs our understanding of individual differences, how the study of normal and atypical development can enhance our understanding of developmental processes, and how domains of development intersect.

Our aim, like that of other editors in this Blackwell series, is to provide a Handbook that is accessible to a broad audience, from students to researchers to practitioners. Each chapter offers an independent overview of a topic, which can be read as a stand-alone piece. Note, however, that the authors liberally reference other chapters in the volume to help readers make important connections across the field. Indeed, several authors read one another's first drafts to discover not only common ground, but also points of diversion. One can easily imagine organizing an early childhood seminar around cross-chapter discussions of key developmental issues. In the final analysis, our aim has been to assemble a Handbook that will be useful to all within our field who seek to understand the developing child, to move the knowledge base forward, and apply this knowledge toward constructive policies and programs for all children.

Kathleen McCartney
Deborah Phillips

PART I

Conceptual Frameworks

1

Nature and Nurture in Early Childhood

Kirby Deater-Deckard and Katherine Cahill

Human development is shaped by dynamic transactions between genes and environments – genetic and environmental influences that can be independent or correlated, and additive or interactive in their effects. These effects cannot be elucidated without understanding how these transactions may be operating throughout the lifespan. The focus in developmental science has shifted toward testing models of how genes and environments work together to create human variability, as part of a much broader trend toward investigating biological and environmental factors in brain growth, functioning, and plasticity.

In the current chapter, we present research investigating the interplay between nature and nurture in early childhood development. We begin with an overview of the techniques used to ascertain genetic and environmental influences, and then turn to a description of what we know about the etiology of individual differences. We concentrate on the domains of physical development, cognitive and language skills, temperament, and the early signs of developing psychopathology. In addition, we consider recent developments in the study of gene–environment processes and molecular genetics, as they apply to early childhood.

Methods in Research on Gene–Environment Processes

Human genes and environments share remarkable similarities across populations. Indeed, humans share much of their genotype with many other species. However, there also is awesome variability in the form and function of genes and environments that give rise to equally remarkable variability across individuals, and it is the examination of the etiology of these individual differences that is at the root of contemporary quantitative and molecular genetics research (Plomin, DeFries, McClearn, & McGuffin, 2001). With few exceptions, behavioral and molecular genetic data are correlational. However, even

correlational genetic designs yield data that are useful in pointing toward likely causal mechanisms, because they control for potential confounds between genetic and environmental influences – confounds that go undetected in most developmental studies of genetically related family members. Behavioral and molecular genetics research, in addition to experimental and quasi-experimental studies of the effects of familial and extra-familial experiences in development, are important contemporary approaches to understanding the contributions of both genes and environments to human development (Collins, Maccoby, Steinberg, Hetherington, & Bornstein, 2000).

Molecular genetic techniques

The Human Genome Project revealed that there are around 30,000 functional human genes – far fewer than the 100,000 that researchers expected to find. Genes are the functional parts of chromosomes that synthesize proteins. These proteins act as enzymes that are the building blocks for neurotransmitters, hormones, and other bio-chemicals. Human chromosomes come in pairs, and people have one allele (i.e., form) of a gene on one chromosome and one allele on the second. There are variations in alleles; some are longer or shorter or more complex than others, and these differences correspond to differences in protein synthesis and the production of chemicals involved in guiding human behavior. Base pairs are the unit of analysis in genome scans, and variability in base pairs at specific gene loci is related to variability in the production, destruction, and expression of enzymes. For instance, single base pair substitutions/single nucleotide polymorphisms (SNPs) and simple sequence repeats (SSRs) are structural variations that are associated with complex trait expression (Craig & McClay, 2003).

Consider as an example the dopamine receptor D4 gene (DRD4), which plays a role in determining the number of dopamine receptors in the brain. Having more dopamine receptors typically translates into greater dopamine activity in the brain, which is related to novelty seeking, attention problems – and, in more extreme cases, schizophrenia and disorganized attachment (Ebstein, Benjamin, & Belmaker, 2003). DRD4 alleles come in at least ten forms (Kluger, Siegfried, & Ebstein, 2002), but the most common are the 4- and 7-repeat alleles, often referred to as the short and long forms of DRD4, respectively. The long form is associated with higher levels of novelty seeking (Ebstein et al., 2003). DRD4 and the serotonin transporter 5-HTTLPR gene have received substantial attention in molecular genetics research, because they are thought to have widespread effects on complex human behaviors.

Molecular genetic techniques allow scientists to identify specific genes involved in the expression of complex human traits and behaviors, based on the analysis of structural differences in DNA like the differences just described in the DRD4 gene. Linkage and association approaches to studying genetic similarity (e.g., allele sharing and allelic frequency at specific locations on chromosomes) among family members have vastly increased our knowledge about individual genes implicated in some of the most widely studied human attributes, including how those genes are differentially expressed in individuals. More recent advances in molecular genetics have focused on understanding the complex processes involved in gene structure and functional expression.

A small number of genes involved in individual differences in early childhood have been identified, and little is understood about the intricacies of the expression of these genes in terms of their products (i.e., proteins, enzymes) and the effects of those gene products. Nevertheless, this research is progressing rapidly, and the work that has been done already greatly enriches our appreciation for the importance of examining gene–environment processes. The decades ahead will be filled with major discoveries regarding variation in structure and function of genes and networks of genes, their products, and the transactions between these and non-genetic factors. These will include discoveries arising from the search for relevant genes (based on genome scans) as well as from investigations of candidate genes in particular neurotransmitter systems implicated for specific attributes (based on existing knowledge from the human and animal biopsychology literatures).

Quantitative genetic techniques

Unlike molecular genetic approaches, quantitative genetic techniques are based on mathematical models that employ principles of population genetics to estimate the proportions of variance that are accounted for by genetic and environmental factors. Studies of sibling and parent–offspring pairs that vary in their genetic similarity (e.g., biological and non-biological relatives in intact, step, and adoptive families; twins; families that have used egg or sperm donation) allow for estimation of genetic, shared environmental, and non-shared environmental effects on outcomes of interest. If family members who are more genetically similar (e.g., identical versus fraternal twins) are more similar on a trait, then genetic variance or heritability is said to account for the greater similarity. If genetic similarity is controlled and family members continue to show similarity, shared environmental variance is said to be present. Non-shared environmental variance includes effects of all the non-genetic influences that lead to dissimilarity among family members, and includes measurement error (Reiss, Neiderhiser, Hetherington, & Plomin, 2000).

The overwhelming majority of research on the effects of nature and nurture in early childhood has employed quantitative techniques, but that trend is changing as molecular genetic techniques become more accessible (Plomin & Rutter, 1998). With this in mind, we turn to review the research on the contributions of genes and environments to children's early physical, cognitive, and psychosocial development.

Stature and Physical Development

A good place to start in considering research on gene–environment processes is with the literature on indices of stature – most scientists agree on what these observable attributes (i.e., *phenotypes*) are, and how they are best measured. There is also consensus on how these should be measured, and if used correctly, the measurement tools yield data that are highly reliable and valid. Quantitative genetics research has indicated substantial

genetic variance in children's height, weight, and body mass index (BMI). Several twin and adoption studies have revealed heritability estimates that increase from early to middle childhood (e.g., Cardon, 1994; Phillips & Matheny, 1990). A study of 14- to 36-month-old twins showed that, even at these young ages, an average of two-thirds of the variance was attributed to genetic factors. Shared environmental variance was highest at 20 and 24 months for all measures, but remained modest, with the exception of moderate shared environment for BMI at 20 and 24 months (Chambers, Hewitt, Schmitz, Corley, & Fulker, 2001).

Environmental effects on BMI are reflected in rapid generational changes, evidenced as increases in the rates of obesity in children in the US. From 1988 to 1994, the rate of obesity in 2- to 5-year-olds rose from 7.2% to 10.4% (Ogden, Flegal, Carroll, & Johnson, 2002). Environmental conditions are implicated because genetic influences do not change this rapidly. Correlational research revealing that breastfeeding in infancy reduces children's risk for childhood obesity also points to the importance of early environmental experiences in physical development (Dietz, 2001). Yet the changing social conditions that promote overeating and sedentary lifestyle probably interact with genetic risk for obesity in some children (see below for more discussion of gene–environment interaction). It is to be hoped that researchers will continue to concentrate on identifying genetic variation as it interacts with environmental factors that put some children at increased risk for obesity and related health problems.

Cognitive Development

We now consider some of the psychological attributes in early childhood that have been investigated in genetically informative studies. Individual differences in children's cognitive development include a number of interrelated domains of skill and performance, ranging from processing speed and capacity, to complex problem solving, to language understanding and use. We concentrate in the following section on the two areas of inquiry that have received the most attention among researchers studying early childhood development – general cognitive ability (e.g., intelligence or IQ) and verbal communication skills.

General cognitive ability

Typically, general cognitive ability is estimated to be moderately heritable, based on twin and adoption studies of preschoolers. Longitudinal studies also suggest that genetic influences on general cognitive ability increase over early and middle childhood, while shared environmental effects are modest and often disappear by middle childhood (Bishop, Price, Dale, & Plomin, 2003; Cherny, et al., 2001; McCartney, Harris, & Bernieri, 1990; Petrill et al., 1998; Plomin et al., 2001; Wilson, 1983). This may reflect developmental changes arising from shifts in the degree to which children have more control, and parents

less control, over their environments and daily experiences (Scarr & McCartney, 1983). Nevertheless, interventions for improving cognitive performance have been shown to be effective (Ramey & Haskins, 1981), and it is important to emphasize that about half of the variance in cognitive abilities is accounted for by non-shared environmental influences.

Single-gene disorders and chromosomal abnormalities are the most common causes of major deficits in general cognitive ability. Down's syndrome is a chromosomal abnormality characterized by the presence of a third twenty-first chromosome, and it is the most widespread cause of mental retardation in both males and females. The single-gene disorders of Fragile X syndrome and Rett syndrome are responsible for the second largest number of cases of mental retardation in males and females, respectively (Plomin et al., 2001). The single-gene disorder PKU is caused by a mutation of the PAH gene, and provides a clear example of how genes and environments work together. The mutation of the PAH gene prevents proper breakdown of phenylalanine, a substance commonly ingested through red meat and other foods. When phenylalanine levels build up, it damages the developing brain and leads to mental retardation and other symptoms. Maintaining a strict diet can prevent the great majority of the effects of PKU. Discovering the genes involved in disorders and how they function can open doors to developing environmental interventions that reduce or alleviate the effects of genetic problems (Plomin et al., 2001).

Language and communication

Many components of language and literacy development are moderately heritable. In this domain, the effects of the shared environment are often more evident, compared to the domain of general cognitive ability. Expressive language skills – compared to receptive skills – appear to be more genetically variable, and more of this genetic variance overlaps with genetic influences on general cognitive ability. In contrast, shared environmental influences appear to be more prominent for receptive language skills, compared with expressive skills (Young, Schmitz, Corley, & Fulker, 2001). Dale, Dionne, Eley, and Plomin (2000) reported heritability estimates of .25 and .39 for lexical and grammatical development, respectively, in 2-year-olds. Shared environmental effects were estimated at .69 for grammar and .48 for lexical development.

Common genetic and environmental processes are thought to underlie lexical and grammatical development, but it is less clear whether general verbal and non-verbal language development shares genetic and environmental influences (Dale et al., 2000). Verbal and non-verbal skills in 2-year-olds are moderately correlated, and less than half of this similarity is accounted for by common genetic influences (Price et al., 2000). Similarly, Dale et al. found low to moderate correlations between lexical and grammatical development and non-verbal skills. However, in contrast, Colledge et al. (2002) found extensive overlap in the genetic influence on verbal and non-verbal skills in 4-year-olds.

Genetic factors appear to be highly influential when it comes to more severe language and communication problems and disorders (Plomin et al., 2001). Dale et al. (1998)

found that heritability in vocabulary development was greater, and shared environmental variance smaller, among those scoring in the lowest 5% of the performance range in their large and diverse sample of 2-year-old twins. Similarly, variance in vocabulary scores for children with *persistent* language problems in early childhood was largely accounted for by genetic factors, whereas variance in vocabulary scores for children with *transient* language problems was more likely to be accounted for by environmental factors (Bishop et al., 2003). The genetic basis of dyslexia and other reading and communication disorders is currently under intense study, and the results of this research will allow for a clearer understanding of how genes and environments work together in shaping children's language development (Plomin et al., 2001).

To summarize, genetic variance is moderate to substantial in studies of cognitive and language functioning and performance in early childhood. There also is evidence for shared environmental influences; these are largest in early childhood, and dissipate with development. In contrast, non-shared environmental influences are present from early in life, and persist into middle childhood and beyond.

Temperament

Next, we consider temperament and its component parts, as the domain of social-emotional development that has received the most attention in behavioral genetic research. The estimates of heritable and genetic variance in these studies vary to some degree, due to differences across study designs (e.g., measurement, twin or adoption study).

Temperament is the framework for personality. It is rooted in biologically based individual differences, is moderately stable over time and across settings, and is modified by gene-environment processes. Individual differences in temperament are observable from infancy and are implicated in many crucial aspects of children's development and adaptation (Emde & Hewitt, 2001; Prior, 1999). Rothbart's theory of temperament posits that there are multiple dimensions of behavior that represent reactivity to stimuli and regulation of those reactions (Rothbart & Bates, 1998). Relevant domains in this literature that we highlight here include negative affectivity, effortful control, extraversion/surgency, sociability, and adaptability (see Rothbart, Posner, & Kieras, this volume).

Negative affectivity

The temperament dimension of Negative affectivity includes anger, sadness, discomfort, and low soothability. Quantitative genetic research indicates that approximately one-third to two-thirds of the variance in negative affectivity is heritable (Goldsmith, Buss, & Lemery, 1997; Oniszczenko et al., 2003; Plomin, Pedersen, McClearn, Nesselroade, & Bergeman, 1988). Angry reactions to restraint and the initiating of fights are estimated to be heritable, and this genetic variance appears to contribute mainly to the observable stability of individual differences (Emde, Robinson, Corley, Nikkari, & Zahn-Waxler,

2001). Some evidence for shared environmental influence also has been found, and environmental sources of variance (shared and non-shared) contribute to both continuity and change in these behaviors across infancy and the preschool years (Emde et al., 2001).

Molecular genetic research has implicated dopamine and serotonin genes in negative emotionality. Infants who have at least one long DRD4 allele display less negative emotionality (Ebstein, Levine, Geller, Auerbach, Gritsenko, & Belmaker, 1998) and less anger in response to restraint (Auerbach, Faroy, Ebstein, Kahana, & Levine, 2001). Mothers' reports of high levels of aggression in 4-year-olds were also found to be associated with the presence of the long form of DRD4 (Schmidt, Fox, Rubin, Hu, & Hammer, 2002). Twelve-month-olds who have two copies of the short form of the serotonin transporter 5-HTTLPR gene showed less pleasure than others during free play (Auerbach et al., 2001).

Effortful control

The dimension of Effortful control includes anticipation and enjoyment of low-intensity stimulation, perceptual sensitivity, and enhanced control of attention and impulses. High levels of effortful control are correlated with lower levels of negative emotionality (Rothbart, Ahadi, & Evans, 2000). Many studies have indicated moderate heritability in the components of effortful control, including task orientation, persistence, and related aspects of "difficult" temperament (Goldsmith et al., 1997; Lemery & Goldsmith, 2002; Manke, Saudino, & Grant, 2001). Molecular genetics research has linked the DRD4 gene to attentional control (Fan, Fossella, Sommer, Wu, & Posner, 2003), but this finding has not yet been replicated in young children. Shared environmental effects stemming from family socio-economic status and observed maternal warmth account for some of the variability in task persistence in early childhood (Petrill & Deater-Deckard, 2004).

Extraversion or surgency

The dimension of Extraversion or Surgency includes activity level, novelty seeking, positive affect, and low shyness. Activity level refers to the amount and intensity of physical movement and it is one of the most thoroughly researched dimensions of early childhood temperament. Overall, activity level has been found to be moderately heritable and to be relatively uninfluenced by shared environmental factors (Goldsmith et al., 1997). Among children at the extremes of activity level, the strength of genetic effects may increase (Manke et al., 2001), and the genetic effects on activity level appear to be moderately to highly stable across time points from infancy to 3 years of age (Saudino & Cherny, 2001). Variance in positive affect and general cheerfulness also appears to be mainly accounted for by heritability and non-shared environmental factors (Eid, Reimann, Angleitner, & Borkenau, 2003; Robinson, Emde, & Corley, 2001).

Research with newborns has identified genes in the serotonin and dopamine systems that are linked with temperament, especially components of temperament that relate to surgency. Among 2-week-olds, the presence of one or two alleles of the long form of the DRD4 gene was associated with higher scores on orientation, range of state, motor organization, and regulation of state (Ebstein et al., 1998). Additionally, an interaction between the DRD4 gene and the serotonin transporter 5-HTTLPR gene was found. Neonates without the long form of DRD4, and who also had only the short form of 5-HTTLPR, had significantly lower orientation scores than other infants (Ebstein et al., 1998). In a follow-up study of the infants in the Ebstein et al. study, Auerbach and colleagues (2001) found that the presence of the long form of DRD4 was associated with higher activity level at 12 months of age.

Sociability

The temperament dimension of Sociability refers to the enjoyment of interpersonal interaction (contrasted with shyness and enjoyment of being alone). Sociability is moderately heritable, with one-quarter to three-quarters of the variance attributed to genetic influences. Some studies also show evidence of shared environmental effects (Eid et al., 2003; Plomin et al., 1988; Schmitz, 1994). Genetic effects on sociability and shyness are moderately to substantially stable across 14 to 36 months of age (Saudino & Cherny, 2001). As with surgency, the heritability of more extreme forms of sociability is greater than that found for moderate sociability (Manke et al., 2001). Molecular genetic research on shyness implicates the 5-HTTLPR gene in increased shyness in second graders (Arbelle et al., 2003), but related serotonin genes have not been found to predict shyness in 4-year-olds (Schmidt et al., 2002).

Adaptability

The dimension of Adaptability is often identified as an important component of temperament, and it includes flexibility, distress in response to novelty, emotional regulation, and high soothability. Adaptability is moderately heritable, and evidence for modest shared environmental effects is sometimes found (Oniszczenko et al., 2003; Rusalov & Biryukov, 1993). Goldsmith, Lemery, Buss, and Campos (1999) reported substantial shared environmental influence in 3- to 16-month-olds' soothability scores. However, genetic effects accounted for all of the twin similarity in distress to novelty. The Ebstein et al. (1998) finding that the presence of the long form of DRD4 was associated with increased regulation of state in neonates suggests that genetic variation in the dopamine system also may play a role in adaptability.

To summarize, like the research on cognitive and language abilities, there is ample evidence of genetic influences in young children's temperament attributes. These findings include moderate heritability estimates as well as associations with specific dopamine and serotonin genes. Also like the research on cognitive and language abilities, there is ample

non-genetic variation – much of this is non-shared, but again there is evidence of some shared environmental variance depending on the particular attribute in question.

Psychopathology

We turn briefly away from consideration of typical variation in cognitive and socio-emotional outcomes toward early indicators of psychopathology among young children. The environmental and genetic influences on psychopathology in early childhood vary depending on the type of symptom being examined, child age, and gender. The evidence for genetic variance is greatest and most consistent for externalizing problems. Internalizing problems (i.e., depression, anxiety, somatic problems) are moderately heritable throughout early and middle childhood, but the effects of the shared environment are less consistent (Murray & Sines, 1996; Schmitz, Fulker, & Mrazek, 1995). In one study, nearly one-quarter of the variance in girls' internalizing problems from ages 4 through 12 years was attributed to the shared environment, but there were no shared environmental influences found for boys (Murray & Sines, 1996). With respect to age differences, Schmitz et al. showed that the effects of the shared environment decreased and the effects of genetics increased on both internalizing and externalizing problems (i.e., aggression, non-compliance, delinquency, attention problems) from early to middle childhood (but see Gjone, Stevenson, & Sundet, 1996, who did not find this pattern for externalizing problems). Other investigations of externalizing problems in early childhood converge to show similarly moderate to high heritability estimates (Arseneault et al., 2003; Dionne, Tremblay, Boivin, Laplante, & Perusse, 2003; van den Oord, Verhulst, & Boomsma, 1996; van der Valk, van den Oord, Verhulst, & Boomsma, 2001; van der Valk, Verhulst, Stroet, & Boomsma, 1998; Zahn-Waxler, Schmitz, Fulker, Robinson, & Emde, 1996).

Molecular genetics research has revealed potential gene–gene interactions that affect certain aspects of early childhood psychological health. Gene–gene interaction is said to occur when the effect of one gene's expression on a trait is moderated by the effect of another gene. One example is found in a study of disorganized infant–caregiver attachment. Infants from a low-risk sample were found to have four times the risk for a disorganized attachment classification if they had a specific form of the DRD4 gene. In addition, the presence of a particular SNP on the DRD4 gene had no main effect on attachment classification but increased the risk of disorganized attachment for children who also had the risk allele to ten times that of children who had neither the risk allele nor the risk SNP (Lakatos et al., 2002).

Gene–Environment Transactions

Up to this point, we have summarized findings regarding additive genetic and non-genetic effects on individual differences measures. However, these effects are not independent,

nor do they operate in isolation from each other. Contemporary genetic theories of development place an emphasis on transactions between the genotype and the environment – specifically, gene–environment correlations and interactions.

Gene–environment correlation

Gene–environment correlation (r_{g-e}) refers to the non-independence of individuals' genetic make-up and the environments in which they exist. The pairing of genetic and environmental factors that interact to influence individual development is not random. Passive and non-passive types of r_{g-e} have been identified using quantitative genetic techniques (Plomin, 1994). Gene–environment correlation can be estimated through quantitative genetic models that include actual measures of the environmental variables of interest, and not just measures of the developmental outcome of interest (Plomin, 1994).

Passive r_{g-e} occurs when biological parents provide environmental conditions for their children that are correlated with their genetic make-up. For example, children who are highly sociable are more likely to have biological parents who also are fairly sociable because sociability is moderately heritable. These parents may expose their children to more people and social interaction than do other parents, and so these children will have many opportunities to further enhance their social skills. These children may develop to experience high levels of social interaction and positive reinforcement from others, and this may appear to be a result of their early exposure to high levels of interpersonal interaction. However, because the experiences they had may have arisen in part from genetic influences, so too do their later outcomes. Often, results from studies of related family members lead to conclusions of environmental causation, but the same findings could also indicate the presence of passive genetic influence. For instance, maternal education has been found to predict aspects of preschoolers' theory of mind development (Pears & Moses, 2003). One might conclude that well-educated mothers interact with their children in ways that promote their understanding of others' minds. Alternatively, the same genetically influenced cognitive abilities that facilitated the mothers' educational attainment might promote early theory of mind understanding in the children. Determining the appropriate interpretation of such findings can be resolved only by using genetically informative study designs that measure the outcomes and relevant environmental factors of interest (Petrill & Deater-Deckard, 2004).

Non-passive r_{g-e} arises when individuals either seek out environments and experiences that are correlated with their genetically based propensities (active r_{g-e}) or elicit responses based on their genetically based attributes that further reinforce those attributes (evocative or reactive r_{g-e}). As an example of active r_{g-e} , children who are low in activity level, a partially heritable trait, may select hobbies and peers that do not promote physical activity. These environmental factors are consistent with their genetically influenced tendencies and may make it less likely that their activity levels increase. Evocative r_{g-e} may be experienced by children struggling with early reading skills, partly on the basis of their genetic make-up, who elicit negative attention from their teachers in a way that serves to further dampen their persistence with and interest in reading. Evocative r_{g-e} also has been impli-