

Chapter 2

Gene-Environment Interplay in Parenting Young Children

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Parental behaviors are generally perceived as a cornerstone of socio-emotional development in infancy and early childhood (Boivin et al. 2005). For instance, parental sensitivity or sensitive responsiveness—the caregiver’s ability to detect the infant’s needs and respond to them fittingly—has long been posited to contribute to a secure parent-child attachment relationship, thus creating a positive context for the child’s later socio-emotional adjustment (Bowlby 1982; Bretherton and Waters 1985; De Wolff and van IJzendoorn 1997). Conversely, insensitive interactive behavior marked by inconsistencies in parental responses and a tendency to adopt hostile, restrictive and punitive behaviors toward the child, has been associated with the development of insecure attachment and later externalizing problems (Boivin et al. 2005; Bradley and Corwyn 2007; Campbell et al. 2007; Garai et al. 2009). Over-soliciting parental practices, including insensitive care, over-responsiveness, overprotection, and intrusion also predict concurrent or future internalizing problems in the child, such as social phobia, depression, and agoraphobia (Becker et al. 2010; Gray et al. 2011; Kim 2011; Lieb et al. 2000; Nishikawa et al. 2010; Spokas and Heimberg 2009).

Thus, many aspects of early parental behavior appear to be involved in the infant’s socio-emotional development, and specific practices, especially those involving punishment, overprotection and lack of sensitivity, have been associated with attachment disorganization, as well as externalizing and internalizing psychopathology. It has been suggested, however, that rather than being a reflection of

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environmental contributions on socioemotional development, these associations could result from shared genetic vulnerabilities between parent and infant (Collins et al. 2000). In such a scenario, genetic contributions would be confounding factors and the parenting behaviors would mediate the relation between parental genotype and infant development. For instance, short-tempered parents that tend to display insensitive, punitive parenting behaviors may have short-tempered children that tend to show irritable and disorganized behaviors. While it is possible that the parenting behavior led to infant developmental characteristics, it is also possible that both behavior patterns are attributable to genetic similarities.

Most models of early development have argued that parenting is rooted in a complex social system where parental characteristics, contextual factors and child characteristics interact over time (Belsky 1984; Belsky and Jaffee 2006; Vondra et al. 2005). Parental personality and life experience may influence their early interaction with their infant, often in complex ways through their interface with immediate environmental conditions (Bornstein 2002). For example, teen parenthood (Morley et al. 2011) and parent mental health (Atkinson et al. 1999; Tarabulsky et al. 2008) have been linked with adverse parenting behaviors. Contextual stressors, such as economic hardship and stressful life experiences (e.g., domestic violence) may also add to the burden of parenting (Lee et al. 2011; McConnell et al. 2011; McLoyd 1998). There is also empirical evidence to suggest that a child's early behavioral characteristics predict parental behaviors and perceptions (e.g., Caspi et al. 2004; Jaffee et al. 2004a; Kiff et al. 2011; Putnam et al. 2002), although there is disagreement about the importance and meaning of these potential "child effects" (Collins et al. 2000; Dodge 1990).

It is clear that a comprehensive study of early parenting should consider a variety of factors related to the infant, the parent and the family context as they interact over time, as well as possible bidirectional associations between them. However, the understanding of such a complex developmental system is limited by an overreliance on studies based on simple correlational designs, even when the latter are longitudinal (i.e., predictive). Given that experimental manipulations other than randomized interventions (which imply important logistic and financial challenges) are often precluded for ethical and legal considerations, behavior-genetic designs are useful for testing hypotheses regarding environmental-developmental processes. Although behavior-genetics designs, also being correlational by nature, cannot provide clear-cut demonstrations of causality, they can, by statistically disentangling genetic from non-genetic sources of inter-individual variance, test hypotheses regarding the environmental nature and direction of the association between child behavior and parenting. For instance, they may help evaluate the extent to which a child's genetic predispositions account for caregiver involvement in specific parental practices. They may also test theoretical models regarding the complex gene-environment processes underlying the predictive association between parental behavior and subsequent child outcomes.

Behavior-genetics studies on early parenting have occupied a growing portion of the recent child development literature. In the past decade, studies using twin, adoption, step-family and linkage (i.e., molecular) designs have provided

important new evidence regarding early parenting and the nature of its association with child socio-emotional development. The goal of this chapter is to review this emerging evidence. In this chapter, we first introduce the reader to attachment theory, a dominant figure of the theoretical landscape regarding early parenting. This theoretical framework will serve as a starting point to posit specific empirical questions relating to the developmental role of early parenting in child development. We then provide a review of extant empirical evidence from behavioral-genetics studies on early parenting and infant socio-emotional development, and discuss its significance for our understanding of the developmental role of early parenting. We conclude with a discussion of methodological concerns and future directions regarding behavior-genetics literature on early parenting.

2.1 Attachment Theory: A Theoretical Framework

Attachment theory, as conceived within an evolutionary framework, posits that the human infant is born with a set of innate neurological and behavioral systems selected for increasing the chances of survival (Bowlby 1982). These mechanisms are deployed within a dyadic regulatory system where associated bonding behaviors are normally activated by impending or perceived external danger and by states of internal distress (i.e., illness, fatigue); and there is a predetermined selectivity of caregivers (i.e., personal relationships) that may provide protection and soothing to the infant. An important theoretical feature is that attachment relationships are formed during the course of interactions with caregivers. Infants gather information on the reliability of caregiver responses in different interactive circumstances, especially in situations where they are alarmed, and by the end of the first year of life, specific representations are formed regarding the caregivers, the self and the nature of interpersonal relationships. These representations influence children's attachment patterns concurrently, and serve as blueprints for the manner in which they will initiate subsequent social relationships (Ainsworth 1985).

2.2 Attachment and Environmental Causation

Bowlby's attachment theory aims to explain individual differences in attachment patterns by individual variations in caregiver behavior. The assumption of an environmental mediation of attachment is grounded in early empirical evidence linking observed sensitive and responsive caregiving behavior at home and characteristic secure attachment behavior patterns in the laboratory-based Strange Situation (SS; Ainsworth et al. 1978). Although many subsequent studies confirmed a significant link between early care and attachment, they varied greatly in the estimated strength of the relationship. De Wolff and van IJzendoorn (1997) reviewed 66

studies to evaluate the mean association between caregiver sensitivity and attachment. They showed that sensitivity, however measured, was far from being an exclusive determinant of the quality of attachment (effect sizes ranged from 0.24 to 0.32). In fact, several other characteristics of parental interactive behavior were identified as playing an equally important role, including mutuality, synchrony, positive attitude, emotional support, and stimulation (De Wolff and van IJzendoorn 1997). Thus, while the theoretical importance attributed to sensitivity as a precursor of attachment is warranted, empirical evidence suggests that other factors may well be involved in the elaboration of this first relationship.

In addition, demographic risk factors, especially if accumulated (e.g., Cummings and Davies 2002; Nair and Murray 2005), and the psychological health of parents, especially mothers (e.g., Martins and Gaffan 2000; Murray 1992), may be involved in the development of attachment, presumably through their proximal or distal influence on early parenting. A recent review of attachment-related findings found that family SES and maternal depression, along with maternal sensitivity, were both independent predictors of specific patterns of attachment (Campbell et al. 2004; McElwain and Booth-LaForce 2006). Again, these results underline that sensitive parenting may not be the only parental factor involved in child development.

Much of the above-cited empirical evidence points toward determinants other than Bowlby's (1982) sensitivity-focused, proximal environmental causation hypothesis. However, it is not clear whether these determinants operate through environmentally mediated processes. Behavior-genetics designs may help to more thoroughly put to test the central assumptions of attachment theory regarding the developmental role of early parental practices, including but not limited to sensitivity. A first assumption is that although attachment relationships are formed in the course of interactions with significant caregivers, children's behavior are to a significant extent influenced by sensitive responsiveness of caregivers, over and above initial "child effects". From a broader, behavior-genetic standpoint, this claim can be verified, for instance, by determining the extent to which genetic and environmental factors respectively account (1) for parental behaviors, and then, (2) for the association between early child characteristics and later parental behaviors, thereby isolating "pure" environmental variance from genetically mediated "child effects" on parenting. A second assumption is that the quality of the relationship with the caregiver influences children's behavior contemporaneously and subsequently. Again, this can be verified by determining the extent to which parental behavior is associated with various child developmental outcomes, and whether this association is mediated by genetic factors or not. This question can also be extended to examining the possible moderating effect of child or parent genetic vulnerability (i.e., gene-environment interaction) qualifying that association. These forms of G-E analyses are made possible when genetic factors are assessed directly (i.e., measured genes) or indirectly (through twin or adoption designs). They help delineate the genetic and environmental architecture of caregiving contribution to child behavior. Specifically, three empirical questions may be analyzed regarding the theoretical claims of attachment theory on the developmental role

of early parenting: (1) To what extent is parent-infant attachment environmentally mediated? (2) Is early parenting a “pure” environmental factor? (3) What type of gene-environment interplay is involved in early parenting?

2.3 To What Extent Is Parent-Infant Attachment Environmentally Mediated?

Most univariate genetically sensitive studies of parent-infant attachment have focused on observed parent-infant interactions. Six twin studies have estimated the genetic and environmental contributions to parent-infant interaction quality, as assessed through child-, parent- or child-parent-focused observational procedures. These six studies typically used a univariate ACE approach where genetic (A), shared environmental (C) and non-shared environmental (E) sources of inter-individual differences on a given measure of parent-infant interaction quality were assessed.

Adoption studies, which typically examine the correspondence in attachment patterns in biologically related and unrelated infant-parent dyads, also provide insight on the possible contribution of shared genes on infant attachment. As adoptive parents and adopted children do not, in principle, share genes by common descent, significant associations between parent and infant attachment-related experience in adoption contexts may arise as a consequence of dyadic interaction histories (Verissimo and Salvaterra 2006). Three such adoption studies examined similarities in attachment among biologically unrelated mother–infant dyads.

Finally, another method used to assess the genetic and environmental contributions to infant attachment patterns is to examine potential independent contributions of functional variants in genotype. Seven such linkage studies have estimated the association between specific genotype variations and infant attachment disorganization.

2.4 Twin Studies of Infant Attachment

The earliest twin studies to examine potential genetic contributions to early attachment patterns used a modified version of the SS, the Louisville Twin Study Procedure (LTS; Matheny et al. 1984), as an index of parent-infant attachment. Similar to the SS, the LTS procedure implies having each twin experience two separations and two reunions with the mother. In a first study, videotapes of 34 MZ pairs and 26 DZ 18–24-month pairs at ages 18 and 24 months were rated using the LTS procedure (Finkel et al. 1998). MZ concordance for attachment was 67.6 %, significantly greater than the DZ concordance of 38.5 %, thus suggesting significant heritability (Finkel et al. 1998). Such findings are in contrast to those from an early quantitative review of parent-infant attachment data on twins

(Ricciuti 1993), which found high concordance in both MZ and DZ pairs, suggesting little genetic mediation. Another report using archival data from a sample of 99 MZ pairs and 108 DZ pairs (mean age = 24 months) from the Finkel et al. (1998) study found an MZ concordance for attachment of 62.6 %, significantly greater than the 44.4 % DZ concordance, with 25 % of the variability in attachment attributable to genetic factors, and the remaining 75 % attributable to non-shared environment (or measurement error; Finkel and Matheny 2000).

Two twin studies assessed parent-infant attachment using the SS (Ainsworth et al. 1978), a well-known seven-episode procedure designed to assess how a child uses the parent as a secure base for exploration. In a first study, 110 42–45-month twin pairs were coded using conventional four-way classifications and a continuous measure of attachment security (O'Connor and Croft 2000). Intraclass correlations were equally high in MZ and DZ twin pairs, suggesting little genetic contribution. Intraclass correlations on the continuous measure of attachment security were 0.48 and 0.38 for MZ and DZ pairs respectively, also suggesting modest heritability. However, contrary to Finkel and Matheny (2000), significant contributions of shared and non-shared environment were found (O'Connor and Croft 2000).

A second study assessed 57 MZ and 81 DZ 12–14-month twin pairs using the SS (Bokhorst et al. 2003). For secure/non-secure attachment classification, 52 % of the variance was accounted for by shared environment, leaving 48 % to non-shared environment. Non-shared environment, which includes measurement error, mainly explained the variance in organized/disorganized classification. Differences in temperamental reactivity, which were heritable at 77 %, were not associated with attachment concordance (Bokhorst et al. 2003).

In a first twin study on *infant-father* attachment, mothers of 14–16-month MZ ($N = 21$) and DZ ($N = 91$) twin pairs sorted the Attachment Q-Sort (AQS; Vaughn and Waters 1990) with a focus on the infant's behaviors in the presence of the father (Bakermans-Kranenburg et al. 2004). Attachment security was explained by shared (59 %) and non-shared environment (41 %), and it was uncorrelated with infant dependency (a contrasting construct assessed with the AQS), which was heritable at 66 % (Bakermans-Kranenburg et al. 2004).

Taken together, findings from twin studies of parent-infant interactive behavior suggest that early secure base behavior towards the mother *and* the father is mainly a function of environments that differ between families (i.e., parenting, family environment). However, two studies using a modified version of the SS found genetic (Finkel et al. 1998) and non-shared environmental mediation (Finkel and Matheny 2000). Unfortunately, these different results could not be replicated, as no other study employed this specific procedure.

Several twin studies assessed constructs neighboring to parent-infant attachment. Two combined studies assessed observed parent-child dyadic mutuality (i.e., shared positive affect, responsiveness, and cooperation; Deater-Deckard and O'Connor 2000). The first study included 62 MZ twin pairs and 58 DZ twin pairs (mean age = 3 years). Heritability and non-shared environment each accounted for half of the variance in mother-child dyadic mutuality, with a non-significant

shared environmental contribution. These findings were replicated in a second observational study of 102 pairs of adoptive and biological siblings in matched comparison families (Deater-Deckard and O'Connor 2000). Using the same observational procedures and measures from videotaped interactions of 3-year children with their parents, full siblings (who share 50 % of their genes as do fraternal twins) were found to correlate at the same level as fraternal twins in the first study, indicating environmental mediation of parent-child mutuality (Deater-Deckard and O'Connor 2000). Another study (Roisman and Fraley 2006) assessed infant-mother relationship quality using the Nursing Child Assessment Teaching Scale (NCATS; Summer and Spietz 1995). In this procedure, the infant's primary caregiver is asked to teach the target child a task just beyond his capacity. A total infant-caregiver score is derived by summing all items within a single indicator characterizing the degree to which parent and child fruitfully employ a "teaching loop", whereby the primary caregiver (a) is observed to properly alert the child, thereby setting up expectations, (b) effectively instructs the child by making suggestions, asking questions, etc., (c) provides time for the child to respond to the instruction and (d) offers adequate and sensitive feedback to the child, and the target infant (e) sends clear cues to the caregiver and (f) is appropriately responsive to caregiver cues. Data was collected on 127 MZ and 333 DZ 9-month twin pairs. Genetic variation was non-significant and the shared and non-shared environmental contributions were substantial in accounting for the infant-caregiver relationship quality (Roisman and Fraley 2006).

Findings from twin studies of parent-infant interactions have been remarkably consistent. In most cases, the estimated genetic contribution to parent-infant attachment security was modest or close to zero. Also, there was evidence in most reports of substantial shared and non-shared environmental contributions to parent-infant attachment security and disorganization, respectively. Inversely, studies using modified versions of well-validated paradigms (Finkel et al. 1998; Finkel and Matheny 2000) found genetic and non-shared environmental (or measurement error) contributions to the parent-infant attachment security.

Despite this notable consistency, several features of the samples of the above-mentioned studies preclude any firm and definitive conclusion regarding genetic-environmental mediation of parent-infant attachment. First, one of the major limitations of this work is its modest-sized samples of unknown representativeness, which may bias the ACE estimates in an unknown way (ACE estimates always depend on the accessible variance of each component in the sample), as well as limit the external validity of results. Second, a huge part of the published reports are based on the same sample or subsamples thereof, albeit with different focus (i.e., mother-child security, father-child security; Roisman and Fraley 2008), which may partly account for the relative uniformity of results across studies. Moreover, the nature of the observational procedures used may have led to a programming effect of context, which might partly explain the shared environmental findings. As context may drive human behavior, especially in stressful settings, a procedure whereby the mother is subjected within a short period of time to the same stressful interactive task is likely to yield a stream of similar behaviors

regardless of who this person interacts with. This could lead to an overestimation of shared environment linked to the context of the observational settings, such as in the SS (the AQS, being a home-visit procedure, is considered more naturalistic). Yet, such settings may also provide the opportunity for understanding the diversity of maternal behavior by giving context for interpretation. For instance, observation of micro-processes within mother–infant interactions often leads coders to recognize the considerable variability of mother responses to infant outstretched arms, as well as the variability of infant signals that may trigger the same response in two mothers.

Finally, most recent work in this area moved beyond univariate ACE models to examine the G-E etiology of the covariance between parent–infant attachment and measured parenting. This particular type of bivariate analysis is especially important in that it may—and does, as will be discussed—provide insight on possible child effects involved in the process, while at the same time providing evidence that some mechanisms underlying the predictive significance of parenting for several developmental outcomes are non-genetic in origin.

2.5 Adoption Studies of Infant Attachment

A first adoption study investigated the concordance between foster mothers' attachment state of mind (i.e., quality of mother's processing of thoughts and feelings regarding her own attachment experience with her child, as assessed through a process of discourse analysis; Main and Goldwyn 1998) and foster infants' attachment quality in a sample of 50 mother–infant (12–24 months) dyads (Dozier et al. 2001). The correspondence between maternal state of mind and infant attachment quality was 72 %, similar to the level seen among biologically intact mother–infant dyads, thus pointing toward a non-genetic mechanism in the inter-generational transmission of attachment (Dozier et al. 2001). A second study examined whether the adoptive mother's internal attachment representation predicted infant attachment security in 106 mother–infant dyads at 3 years (Verissimo and Salvaterra 2006). Scores reflecting the presence and quality of maternal secure base scripts concurrently predicted infant security according to the AQS (Verissimo and Salvaterra 2006). A third study tested associations between maternal state of mind regarding attachment upon their adopted infant and emotional themes appearing in doll play narratives obtained from their recently adopted and previously maltreated 4–8-year children (Steele et al. 2003). Significant associations were found between maternal state of mind and infants' story-completions. Specifically, mothers judged insecure (dismissing or preoccupied) tended to have adopted children who, 3 months after placement, provided story-completions with higher levels of aggressiveness as compared to the stories provided by children adopted by secure-autonomous mothers. Moreover, children whose adoptive mothers displayed unresolved mourning regarding past loss or trauma provided story completions with higher scores for emotional themes such as 'parent appearing

child-like' and 'throwing out or throwing away' (Steele et al. 2003). Thus, three adoption studies of attachment-related experience in adopted (i.e., biologically unrelated) mother–infant dyads bring additional support to the notion that intergenerational transmission of attachment-related experience involves non-genetic (i.e., most likely environmental) processes.

2.6 Linkage Studies of Infant Attachment

Another exciting avenue for research on genetics of infant attachment is provided by molecular genetics. Once identified, measured genes can be incorporated into regular studies and analyzed together with other measured variables, such as attachment. Evidence so far has linked infant disorganization with the DRD4, 5-HTT and OXTR gene polymorphisms.

The DRD4 gene polymorphism has been linked to novelty seeking, pathological impulsive and compulsive behavior in adults (Benjamin et al. 1996; Comings et al. 1999), as well as ADHD in children (Faraone et al. 2005); most of these phenotypes potentially having in common impairments of regulation of emotional arousal and a possible dysregulation of the reward system. Functional variants of the DRD4 gene polymorphism may thus be involved in infant engagement and activity level during interactions with caregivers, thus eliciting adaptive or maladaptive responses from the caregiving environment (Mills-Koonce et al. 2007). Therefore, significant associations between DRD4 variations and infant attachment disorganization (i.e., the lack of coherent behavioral strategy to cope with social stresses) may be plausible on theoretical grounds.

The earliest linkage study of infant attachment assessed attachment disorganization of a low-social-risk sample of 90 20-month infants with the SS (Lakatos et al. 2000). A significant association was found between the DRD4 7-repeat (i.e., long allele) and attachment disorganization: the DRD4 7-repeat was significantly more frequent in disorganized infants than in non-disorganized infants (Lakatos et al. 2000). The estimated relative risk for disorganized attachment among carriers of the DRD4 7-repeat was fourfold (Lakatos et al. 2000). The authors extended these findings by genotyping the same infants for the functional -521 C/T single nucleotide polymorphism in the upstream regulatory region of the DRD4 gene, in order to test the association with disorganization both alone and in interaction with the DRD4 7-repeat (Lakatos et al. 2002). There was a significant interaction between the DRD4 7-repeat and the 521 C/T promoter polymorphism, the odds ratio for disorganized attachment increasing tenfold in the presence of both risk alleles (Lakatos et al. 2002). Findings from these two reports indicate that the DRD4 7-repeat promotes independent genetic risk for infant disorganization. This risk may be amplified if an individual also possesses the 521 C/T promoter polymorphism risk factor.

In another report using the same sample, observed response to a novel, anxiety-provoking stimulus (i.e., SS) was investigated for 90 12-month infants genotyped

for the DRD4 7-repeat and for 5-HTTLPR risk alleles (Lakatos et al. 2003). Combined genotype contributions were found: infants with at least one copy of both the DRD4 7-repeat and the long variant of 5-HTTLPR responded with less anxiety than other infants. Inversely, infants with the DRD4 7-repeat and who were homozygous for the short form of 5-HTTLPR showed more anxiety and resistance to the stranger's initiation of interaction (Lakatos et al. 2003).

A replication of the first two Lakatos studies (2000, 2002) in a larger sample did not confirm the contribution of the DRD4 7-repeat and the -521 C/T promoter gene on disorganized attachment (Bakermans-Kranenburg and van IJzendoorn 2004). Although the sample used was larger ($N = 132$; mean age = 50 months), which may have enhanced the power to find significant DRD4-C/T interactions, the association was not found. Even when the authors combined their sample with the Lakatos sample, the interaction of the DRD4 and -521 C/T polymorphisms on disorganization was not confirmed (Bakermans-Kranenburg and van IJzendoorn 2004). These results contradict those obtained by Lakatos et al., which indicated independent contribution of the DRD4 7-repeat and interaction with the -521 C/T polymorphism.

Following up the results of their previous population linkage studies, the Lakatos group performed extended transmission disequilibrium tests (ETDT) on the same sample to determine whether biased transmission of the DRD4 7-repeat occurred to infants displaying disorganized and secure attachment behavior with their mothers (Gervai et al. 2005). A trend for preferential transmission of the DRD4 7-repeat to disorganized infants and a significant non-transmission of this allele to secure infants were observed, suggesting that results from the Lakatos et al. studies were not due to population stratification (Gervai et al. 2005).

Finally, a recent investigation examined the oxytocin receptor OXTR gene, purportedly involved in social stress regulation, as a possible source of variation in infant attachment, as assessed by the SS (Chen et al. 2011). In a sample of 176 12–16-month infants, the A allele of OXTR rs2254298 was significantly associated with attachment security, but only in non-Caucasian infants (Chen et al. 2011).

Findings from linkage studies of infant attachment have been rather inconclusive. In the earliest studies (Lakatos et al. 2000, 2002), significant links were found between infant disorganization and the DRD4 7-repeat. These findings were later extended using ETDT (Gervai et al. 2005). However, one study using a larger sample, even when combining its own sample with the sample from the Lakatos studies (Bakermans-Kranenburg and van IJzendoorn 2004), could not replicate such findings. There may be multiple reasons for non-replication of such association studies (e.g., low statistical power, population stratification). The primary cause of non-replications may lie in the nature of polygenic inheritance (Comings 1998). Complex human behaviors, such as parent-attachment, are likely accounted for by multiple genes (and functional variants) each individually having a small effect. There may be an inverse relation between the odds of finding significant independent genetic contributions to a human trait, and the sophistication of the trait of interest (Turkheimer and Waldron 2000). The possible genetic heterogeneity across populations (i.e., specific combinations in genotype being more

prevalent in some populations than in others) may also explain the lack of replication. Negative findings may also reflect the low power of individual studies that, when combined in a meta-analysis, may yield a significant albeit small effect.

In brief, findings from linkage studies remain equivocal regarding the importance of the DRD4 7-repeat and its association with infant disorganization. Yet, DRD4 variations have previously been linked to high reward-dependence, low behavioral inhibition and low self-regulation traits or conditions. These associations may be plausible on theoretical grounds, as infant activity level and capacities of regulation of emotional arousal could have—through possible “child effects”—a proximal or distal effect on early parenting. Subsequently, early adaptive or maladaptive interactions between the parent and the infant may contribute to the development of infant secure base behavior (Bowlby 1982). Conclusive demonstration of such assumptions within behavioral-genetics designs is however still lacking.

With only a few exceptions, findings from univariate twin and adoption studies generally suggest that parent-infant attachment is mainly environmentally mediated, with shared environment prevailing in explaining variance on attachment security (e.g., Roisman and Fraley 2006), and non-shared environment (or measurement error) explaining most of the variance on attachment disorganization (Bokhorst et al. 2003), which raises issue of the validity of these measures to assess disorganization. Moreover, most findings from linkage studies indicate a significant contribution of specific genes on infant disorganization (e.g., Lakatos et al. 2000, 2002), although a more powerful study yielded negative findings (Bakermans-Kranenburg and van IJzendoorn 2004). Therefore, no clear-cut conclusion can be drawn regarding the possible links between infant genotype and early attachment phenotype.

In brief, results from twin and linkage studies generally suggest little genetic influence on infant attachment behavior. The possibility of genes interacting with putative environmental risk factors still remains. As will be discussed in further sections, G-E processes involved in early parenting and infant attachment were investigated in numerous bivariate studies.

2.7 Is Early Parenting a “Pure” Environmental Factor?

Analyzing parenting in a behavior-genetic design allows for the assessment of the G-E etiology of parental behaviors. In univariate ACE twin designs, finding significant heritability for a parental behavior may be indirect evidence of “child effects” on parenting. It is a first step in assessing possible G-E correlation involving parenting behaviors, as will be further discussed. Inversely, finding significant associations between maternal genotype and parental behaviors tend to mitigate the importance of these child effects, as will also be discussed. Four univariate ACE twin studies estimated the G-E etiology of parental perceptions and self-reported styles, one adoption study assessed home environment in biologically

related and unrelated siblings, and three linkage studies estimated the association between maternal sensitivity and maternal genotype.

2.8 Twin Studies of Early Parenting

Four reports have documented the genetic and environmental contributions to parents' self-reported perceptions and behaviors toward their infants. In a first report, a sample of *twin parents* of children under 8 completed the Parental Attitudes Toward Childrearing Questionnaire (PATC; Easterbrooks and Goldberg 1984) assessing positive support and negative control (Losoya et al. 1997). Moderate heritability was found for all parenting variables, and shared environment mediation was significant for non-affective control. These results suggest child effects on all parenting variables but non-affective control, which may be a function of parental characteristics. Yet, small sample size made statistical distinctions among models difficult in most cases (Losoya et al. 1997). In a second study, *twin parents* completed a questionnaire assessing four parenting styles: over-protective, rejecting, supportive, authoritarian (Spinath and O'Connor 2003). Genetic and non-shared environmental mediation were found for all parenting styles but the rejecting one, mainly mediated by non-shared environment (Spinath and O'Connor 2003). Thus, most parenting styles may be accounted for by genetic factors in the child and non-shared environmental factors; except for the rejecting style, which may be a function of within-family differences and/or measurement error.

In a third report, parents of 5-month twins completed a questionnaire assessing four parenting dimensions: self-efficacy, perceived parental impact, hostile-reactivity and overprotection (Boivin et al. 2005). Shared environment mainly accounted for each parenting dimension. Maternal hostile-reactive behaviors were also moderately heritable, and this association was mainly mediated by infant difficulty (Boivin et al. 2005). In a follow-up of this study, the same group performed genetic analyses on 292 mothers' self-reported hostile-reactive behaviors toward each of their twins at 5, 18 and 30 months (Forget-Dubois et al. 2007). The heritability of maternal hostile-reactive behavior was modest and longitudinal analyses indicated that genetic factors at five and 30 months, although present, were uncorrelated. Shared environment was the main source of variance at the three ages and were highly correlated through time (Forget-Dubois et al. 2007). It was concluded that children's heritable characteristics may evoke maternal hostility at specific times, but were not responsible for its stability from infancy to toddlerhood (Forget-Dubois et al. 2007).

Findings from univariate studies of self-reported parenting are consistent. First, studies of twin infants and *twin parents* have shown that most parenting perceptions may be a function of characteristics that differ between families, although time-specific dimensions of parenting behavior involving harshness toward the child are partly a function of child effects or shared genes. Thus, while most forms of parental perceptions seem to be driven by characteristics of the early caregiving

environment, self-reported harsh parenting may be child-evoked at specific times (e.g., during challenging phases of development).

This pattern of results is similar for parenting styles assessed in *twin parents* of infants. One study found that most parenting styles are heritable among twin parents, whereas a highly maladaptive style (i.e., rejecting) is mainly a function of non-shared environment (i.e., characteristics that differ between twin parents from a same family). As with parental perceptions, a wide range of parenting styles may be a function of—potentially heritable—parental features, while self-reported negative parenting within a normative range (i.e., harshness, hostility towards the child) may be partly a function of child characteristics (see also the work by Jaffee described in the rGE section of the present chapter).

2.9 Adoption Studies of Early Parenting

One adoption study examined the resemblance of 105 non-adoptive and 85 adoptive sibling pairs on the Home Observation for Measurement of the Environment (HOME), at 12 and 24 months of age (Braungart-Rieker et al. 1992). Non-adoptive sibling correlations were found to be greater than those for adoptive sibling pairs at both ages, suggesting genetic contributions on the HOME. Moreover, phenotypic and cross-sibling correlations between family environment and subsequent behavior problems were greater for non-adoptive siblings than for adoptive pairs at 24 months, suggesting genetic mediation of this association (Braungart-Rieker et al. 1992). These results suggest that early home environment quality may be a function of child effects or shared genetic vulnerabilities between parent and infant.

2.10 Linkage Studies of Early Parenting

Three linkage studies examined possible contributions of genotype to sensitive parenting. A first study tested the association between mothers' serotonin transporter (5-HTT) and oxytocin receptor (OXTR) genes, both posited to modulate affiliation responses to offspring during interactions, and observed sensitive parenting in 159 mothers toward their 2-year-old infants at risk for externalizing problems (Bakermans-Kranenburg and van IJzendoorn 2008). Significant contributions of 5-HTTLPR SCL6A4 and OXTR rs53576 to maternal sensitivity were found. Controlling for maternal education, depression and marital discord, parents with less efficient variants of these genes showed lower sensitivity (Bakermans-Kranenburg and van IJzendoorn 2008). Another study examined the links between oxytocin receptor (OXTR), peripheral oxytocin (OT) and sensitive parenting of 272 mothers and fathers toward their 4–6-month infants (Feldman et al. 2012). CD38 risk allele of OXTR, which mediates the release of brain OT, was also assessed. Reduced plasma OT and both OXTR and CD38 risk alleles were related

to less observed parental touch. The interaction of high plasma OT and low-risk CD38 alleles predicted longer durations of observed parent-infant gaze synchrony. Parents reporting greater parental care showed higher plasma OT, low-risk CD38 alleles, and more touch toward their infants (Feldman et al. 2012). Finally, a recent study examined the associations between the arginine vasopressin receptor 1A (*AVPR1A*) and observed parenting in a normative sample of mothers of infants ($M_{\text{age}} = 3.5$ years; Avinun et al. 2012). The *ABPR1A* RS3 allele has been linked with stress hyperreactivity (see Avinun et al. 2012). Multilevel regression analyses revealed that mothers who are carriers of the *AVPR1A* RS3 allele tend to show less structuring and support throughout the interaction independent of the child's sex and RS3 genotype (Avinun et al. 2012). Taken together, findings from three linkage studies suggest independent contributions of genotype to sensitive parenting in high- and low-risk samples. Functional variants of genes involved in affiliation across various interpersonal contexts may also predict the quality of the infant-caregiver relationship, as parental involvement in sensitive care toward the infant was significantly linked with 5-HTT and OXTR variations. Moreover, genes involved in stress hyperreactivity may also predict the quality of parenting, as *AVPR1A* was linked to less structuring and support throughout a parent-child interaction.

Overall, findings from univariate twin, adoption and linkage studies of early parenting have been relatively consistent. Four twin studies indicated that most early self-reported parenting dimensions are a function of parents' own characteristics (i.e., shared environmental variance in twin infants designs, and genetic variance in twin parents designs), although highly maladaptive forms of parenting may partly be mediated by child genetic vulnerability at certain periods of development (i.e., heritability variance in twin infants designs, non-shared environmental variance in twin parents designs). One adoption study also indicated that child genetic risk extends to early environment (Braungart-Rieker et al. 1992). This pattern of results is consistent with several non-genetic studies suggesting that child temperamental features such as frustration or fearfulness may elicit maladaptive parenting (e.g., Martini et al. 2004; Rubin et al. 2003). A recent meta-analytic review of twin studies of parenting (n.b., including, but not limited to, parenting in infancy and early childhood; Klahr and Burt 2013) also supports this idea. This study indicated that 40 % of the individual differences in parental negativity are accounted for by genetic factors, while other dimensions of normative parenting (i.e., control, warmth) included significant, but very small (23–26 %) genetic contributions.

At the same time, recent univariate linkage studies have revealed independent contributions of maternal genes known to be involved in social bond formation (e.g., Feldman et al. 2012), suggesting some maternal genetic underpinning of sensitive parenting, over and above child genetic risk. Thus, it appears that parents' own genetic characteristics predict their parenting perceptions and child-rearing practices. This idea is also supported by the above-mentioned meta-analysis (Klahr and Burt 2013), which concluded to significant contributions of parental genetic makeup to parental negativity and warmth. Such key genetic contribution from the parent to early parenting behaviors could, across generations, take the form of shared environmental variance in the context of child-based twin studies of early parenting.

In other words, there seems to be a dual intergenerational process where genetic factors could play a role in parenting: (1) an elicitation process (i.e., “child effects”) driven by child genetic risk associated to early environment and parenting, and (2) a direct parental genetic contribution to sensitive parenting that is independent of child-driven evocations. However, maternal genetic vulnerability may not operate independently of child genetic risk, as shared genes with the child could bolster parental involvement in maladaptive parenting. As will be further discussed, parental genetic risk may also *interact* with child genetic risk in predicting specific parental practices and child outcomes. Notwithstanding these more intricate G-E joint contributions, this review suggests that while child effects or shared genes may contribute to time-specific negative parenting, parental characteristics may independently account for a wider range of self-reported and observed parenting behaviors. This however excludes the prospect that early parenting is a “pure”, parent-driven environmental factor, and thus provides indirect support for Belsky’s (1984) multi-causal model of early parenting.

As stated earlier, finding evidence of child genetic contribution to purportedly “environmental” features such as early parenting is a first step toward assessing more directly potential G-E correlations (Scarr and McCartney 1983) involving parenting behaviors. Such G-E correlations may take the form of two processes. A first process, referred to as an evocative (or reactive) G-E correlation (Plomin et al. 1977), may be seen as child effects on the relationship with caregivers. A second process, the passive G-E correlation (Plomin et al. 1977), refers to shared genetic vulnerabilities between parent and child. Although genes do not have to be directly measured to test for a potential G-E correlation within a behavioral-genetic framework, child behavior and the putative environmental variable (e.g., harsh parenting) have to be measured directly. When the measured child behavior is significantly associated with the measured environmental feature, a twin design makes it possible to evaluate the extent to which this association is accounted for by the child’s genes, thus pointing to a potential G-E correlation; although the exact type of correlation—evocative or passive—is not specified. Evidence for G-E correlation in early parenting comes mainly from studies using twin, adoption and step-family genetic designs.

2.11 Gene-Environment Correlations in the Context of Early Parenting

2.11.1 Twin Studies

Three pioneering studies examined the possibility of G-E correlation using direct behavioral observation of parent-child interactions. In a first study, the question of differential treatment during observed interactions involving parents of twins and of male singletons was investigated (n.b., undetermined age; Lytton 1977). Four conclusions were drawn by the authors from correlational analyses: (1) parents treat

MZ twins more alike than DZ twins in some respects; (2) they do not introduce systematically greater similarity of treatment for MZ twins in actions which they initiate themselves; (3) the greater homogeneity of treatment of MZ twins, where it occurs, is in line with their actual, rather than their perceived, zygosity; (4) parents respond to, rather than create, differences between the twins (Lytton 1977). In a second study, the same group used biometrical genetic analysis to study interactive behavior of 24-month male twins with their parents in home and laboratory (Lytton et al. 1977). A model, which included non-shared and shared environmental mediation best fitted most variables, except for instrumental independence and speech rate which showed significant heritability (Lytton et al. 1977). A third study examined differential observed maternal treatment as a function of 7–9-month twins' zygosity (DiLalla and Bishop 1996). Mothers tended to treat both children similarly, regardless of zygosity, suggesting that maternal traits drove the mother–infant interactions. Therefore, even though identical twins were more similar than fraternal twins, mothers tended to treat both types of twins comparably (DiLalla and Bishop 1996).

Thus, two pioneering studies using correlational (Lytton 1977) and univariate twin designs (Lytton et al. 1977) provided indirect evidence of G-E correlation in the context of infant-parent interactions, though in one study this result was specific to precise components of the interaction (Lytton et al. 1977). However, another study of differential maternal treatment (DiLalla and Bishop 1996) found that mothers tend to treat both twins similarly, regardless of zygosity, which reduces the likelihood of child effects on parental behavior.

In a direct test of attachment theory's main assumption—caregiver's sensitivity directly affects infant attachment security through environmental causation—two studies examined the G-E etiology of the association between sensitive parenting and infant attachment. One study assessed maternal sensitivity in the home at 9–10 months, and infant attachment security was observed in the laboratory at 12 months (Fearon et al. 2006). Shared environment in maternal sensitivity was able to account for some of the similarity in attachment security, and weak non-shared associations appeared to suppress the magnitude of the correlation between attachment and sensitivity; thus suggesting little genetically mediated child effects (though child effects may also come out through a non-shared environmental pathway; Fearon et al. 2006). In a second study, 485 same-sex twin pairs were used to test for G-E associations between observed parenting quality and infant attachment security (Roisman and Fraley 2008). In line with the results of Fearon et al. (2006), both constructs observed at 24 months, as well as their covariation, were accounted for by shared (85 %) and non-shared (15 %) environmental variance (Roisman and Fraley 2008).

Thus, findings from two twin studies suggest that infant security during the first two years of life is mainly a function of characteristics of the early caregiving environment, but also, that considerable within-family differences may be involved. Though mainly pointing to shared environmental contribution, these findings raise a doubt on attachment as due to an absolute, all-embracing shared environmental effects. They underline the continuing challenge posed to attachment theory by within-family differences (which may involve child effects) in socio-emotional processes.

A great deal of bivariate reports examined the possibility of child effects in explaining maladaptive forms of early parenting such as harshness or negativity. Indeed, significant genetic correlations were found between parental negativity and various parent-rated developmental problems in the infant: conduct problems (although shared environment mediation was found for observations; mean age = 43 months; Deater-Deckard 2000; mean age = 4 years; Alemany et al. 2013), low prosocial behavior (ages 3, 4 and 7; Knafo and Plomin 2006), externalizing problems (but only in boys; 7, 9, 14, 24 and 36 months; Boeldt et al. 2012), antisocial behavior (ages 4 and 7; Larsson et al. 2008). However, there is evidence of environmental mediation of the risk for antisocial behavior via parental negative feelings toward the infant (Larsson et al. 2008). Shared environment also contributes to several protective cycles of parenting, namely the association of high parental positivity with prosocial behavior (Knafo and Plomin 2006) and low externalizing behavior (but only in girls; Boeldt et al. 2012).

Moreover, four studies using a MZ twin differences design investigated non-shared environmental associations of parental positivity/negativity with various developmental outcomes. Because MZ twins do not differ genetically, associations between early parenting and their behavior can be directly ascribed to non-shared environment. Thus, although this method does not directly test for G-E correlations, it is still somewhat informative of the etiology of associations of parenting with child behavior. In a first study, parental warmth, control and responsiveness covaried in expected ways with twin differences in temperament, prosocial behavior and behavior problems; mean age = 3, 5 years; Deater-Deckard 2001). The twin who received more supportive/positive and less punitive/negative parenting was also higher in positive mood/prosocial behavior and lower in negative mood/behavior problems when compared to his twin; suggesting non-shared environmental mediation (Deater-Deckard 2001). Another study found non-shared environmental associations between two early parenting measures (harsh discipline, negative feelings) and four infant outcomes (anxiety, prosocial behavior, hyperactivity, conduct problems), especially for the extreme portion of the parenting- and behavior-discordant distributions (mean age = 4 years; Asbury et al. 2003). More non-shared associations were found between overprotection and boys' social reticence, and between hostile parenting and girls' social reticence, only with high levels of depressive symptoms in fathers (mean age = 30 months; Guimond et al. 2012). Non-shared associations were also found between high maternal negativity/low warmth and child antisocial behavior (mean age = 5 years; Caspi et al. 2004).

Thus, findings from twin studies mainly point to "child effects" in the form of disruptive behavior on early parental negativity. Within-family differences are also likely to be involved in explaining links between parental negativity and various behavioral and interpersonal outcomes. This pattern of results excludes shared environmental contributions to maladaptive parenting. Rather, shared environment might account for protective cycles linking parental positivity with child prosociality and low externalizing behavior.

Potential G-E correlations were also investigated in explaining the well-documented association between physical maltreatment and later antisocial behavior.

A study using a sample of 1116 5–7-year twin pairs found that child antisocial behavior at 5 (i.e., inferred “child effects”) did not account for the prospective association between mother-reported physical maltreatment at 5 and teacher-reported child antisocial behavior at 7 (Jaffee et al. 2004b). These findings suggest that early maltreatment plays a causal role in the later development of antisocial behavior, over and above “child effects” (Jaffee et al. 2004a). In a second study using data from the same sample, the limits of such “child effects” were tested on parental behavior that ranged from the normative (i.e., corporal punishment) to the non-normative (i.e., physical maltreatment; Jaffee et al. 2004a). Shared environment accounted for most of the variation in corporal punishment, as well as in and physical maltreatment. However, corporal punishment was partly genetically mediated (which was not the case for physical maltreatment), and the genetic factors that accounted for corporal punishment were largely the same as those that accounted for child antisocial behavior, suggesting “child effects” (Jaffee et al. 2004a). Thus, risk factors for maltreatment are unlikely to reside within the child and more likely to reside in features varying across families; though normative discipline in the form of corporal punishment may partly be a function of child effects or shared genes. This is consistent with results from epidemiological studies identifying more extreme adverse early environments, such as inadequate housing (Palusci and Loeb 2011) or public aid as a source of income (Parrish et al. 2011), as risk factors for child maltreatment. As stated earlier, bidirectional links between early parenting and these broader psychosocial risk factors are likely.

In this regard, one MZ twin differences study investigated non-shared associations of two broad psychosocial risk factors, namely birthweight-discordance and early family environment, with behavior problems and academic achievement at 7 (Asbury et al. 2006). MZ differences in anxiety, hyperactivity, conduct and peer problems and academic achievement correlated significantly with MZ differences in birthweight-discordance and family environment (Asbury et al. 2006). Associations increased at the extremes of discordance, even in a longitudinal, cross-rater design, with effect sizes reaching 12 %. Some of these associations operated partly as a function of SES, family chaos and maternal depression. Higher-risk families generally showed stronger negative associations (Asbury et al. 2006). This suggests that broad risk indicators of early adversity may be linked with child behavioral and social development through a non-shared environmental pathway. Another study attempted to identify the factors comprising the shared environmental variance on cognitive performance in early childhood (mean age = 3, 5 years; Petrill and Deater-Deckard 2004). SES and parental warmth, taken together, accounted for most shared environmental covariance between task engagement and cognitive skills, indicating that early indicators of environmental adversity such as SES may also contribute to child cognitive development.

Overall, most bivariate findings point to the shared and non-shared environmental etiology of the associations of characteristics of the early caregiving environment with child behavioral and socio-emotional outcomes. This is especially true in the context of protective cycles. This pattern of results points to the importance of features of the early caregiving environment and of specific parental treatment

within mother–infant dyads, over and above initial child effects. Inversely, there is evidence of child effects on more adverse forms of parenting in predicting various developmental problems in the child. Thus, in the normative range, there seems to be some kind of a continuum between adaptive parenting, mainly a function of parental characteristics and family environment, and maladaptive parenting, possibly driven by child characteristics having a proximal or distal effect on the caregiving environment (or shared genes). Nevertheless, there seems to be limits to such child effects on adverse parenting, as infant disruptive behavior cannot account for extreme forms of maladaptive parenting ranging in the non-normative spectrum—such as physical maltreatment—, which may partly be a function of broader social-risk factors and their possible bidirectional relations with early parenting.

2.11.2 Adoption Studies

Using a prospective adoption design, one study investigated possible genetic contributions to associations between early family environment and behavior problems at 7 (Braungart-Rieker et al. 1995). Patterns of correlations for non-adopted and adopted boys indicated that links between quality of family environment (i.e., conflict, cohesion, expressiveness) and externalizing behavior in home and school were genetically mediated; indirectly suggesting a G-E correlation. For girls, these links were associated with shared environmental mediation (Braungart-Rieker et al. 1995).

Another recent study investigated the developmental underpinnings of children's socially disruptive behavior using a genetically sensitive design that allowed examination of parent-on-child and child-on-parent (evocative G-E correlation) effects (Elam et al. 2013). Using an adoption-at-birth design, this controlled for passive G-E correlation and directly examined evocative G-E correlation while examining the associations between family processes and children's peer behavior. In 316 linked dyads of birth mothers, adoptive parents and adopted children, this study examined the evocative effect of genetic influences underlying toddler low social motivation on mother–child and father–child hostility and the subsequent influence of parent hostility on disruptive peer behavior during the preschool period (Elam et al. 2013). Results showed that birth mother low behavioral motivation predicted toddler low social motivation, which predicted both adoptive mother–child and father–child hostility. This suggests the presence of an evocative G-E correlation (Elam et al. 2013).

2.11.3 Step-Family Studies

One study used a step-family quantitative genetic design to estimate G-E etiology of 4-year children's behavior problems and prosocial behavior, as well as negativity in their relationships with their mothers and mothers' partners (mean

age = 4 years; Deater-Deckard et al. 2001). Behavior problems and partner-child negativity were mainly heritable, and shared environmental variance accounted for mother- and partner-child negativity. 1/5 to 2/3 of the variance was accounted for by non-shared environment. The link between parental negativity and behavior problems was mediated by genetic covariance, and the link between parental negativity and prosocial behavior was mediated by environmental covariance (Deater-Deckard et al. 2001). This pattern of results suggests that the adverse association of parental negativity with behavior problems may be a function of “child effects”. The protective link between low parental negativity and prosocial behavior may be a function of the early caregiving environment (Deater-Deckard et al. 2001).

Five general conclusions can be drawn according to results from twin, adoption and step-family studies that examined G-E correlations in the context of early parenting. First, a wide range of early parenting practices providing a positive context for child development are mainly a function of characteristics of the caregiving environment, and are hardly accounted for by child heritable characteristics. Thus, protective factors for a several child outcomes seem to lie in environmental features that differ between families. However, as will be further discussed, several features of adaptive parenting—although independent of “child effects”—may not be free of genetic factors, as they are partly driven by parental genotype. Part of the prominence of statistically estimated environmental mediation within bivariate twin studies may, in fact, reflect parent-driven genetic processes. Second, adverse parental practices within a normative range may be regarded as developmental incidents partly involving “child effects”, whereby parental negativity or corporal punishment, for instance, are mainly explained by child heritable characteristics. Shared genes may also partly account for such associations. This is consistent with results from univariate studies indicating that negative parenting, especially time-specific, is mainly a function of child characteristics or shared genes (Forget-Dubois et al. 2007), but also that a wider range of practices, especially adaptive ones, are driven by parental characteristics (i.e., shared environment or parental genotype). Third, more extreme forms of adverse parenting (e.g., physical maltreatment) may not result from customary developmental incidents, as child evocation does not account for physical maltreatment. Hence, there are limits to child effects on maladaptive parenting, which may be parent-driven in its more extreme forms.

2.12 Which Gene-Environment Interactions Are Involved in Early Parenting?

Several univariate studies suggest that a wide range of early parenting behaviors are environmentally-driven. However, evidence from bivariate twin studies suggests that early parenting does not necessarily operate independently of genes. As suggested earlier, most developmental outcomes may result from joint, rather than additive, contributions of genetic and environmental factors (Rutter and Silberg 2002). Therefore, another G-E process of interest regarding early parenting and

its association with child behavior refers to a possible G-E interaction (i.e., interacting processes between a putative environmental risk factor and an infant or parental genetic vulnerability), which would be indicated if, for example, the association of harsh parenting with child externalizing problems were differentially manifested as a function of genetic risk for such problems. This mechanism is consistent with the diathesis-stress model of psychopathology, according to which an environmental stressor is more likely to lead to maladjustment if pre-existing genetic vulnerabilities are present (Zuckerman 1999). Beyond the diathesis-stress model, the broad concept of G-E interaction encompasses differential susceptibility, which refers to variations in the degree to which an individual is affected by specific environments—not only to adverse but also to protective ones—according to his or her genotype (Belsky and Pluess 2009).

To evaluate a potential G-E interaction, genes do not have to be directly measured, but a putative environmental variable has to be. Moreover, finding statistical evidence for a G-E interaction is most likely if the measured behavior is under strong genetic influence and if, in contrast, the measured environmental variable has little relation to genetic factors (Rutter and Silberg 2002). In the absence of a G-E interaction, genes and exposure to a specific environment may independently, thus additively, contribute to child outcomes; indicating general (main) rather than conditional (joint) contributions of environment and genotype.

2.12.1 Twin Studies

To our knowledge, four twin reports have examined potential G-E interactions involving early parenting and infant behavior. A first study tested whether the association of parent-reported physical maltreatment with risk for parent- and teacher-rated conduct problems was strongest among infants who were at high genetic risk for externalizing behavior (i.e., co-twin conduct disorder status and the pair's zygosity), using data from 1116 5-year twin pairs and their families (Jaffee et al. 2005). The experience of maltreatment was associated with an increase of 2 % in the probability of conduct disorder among children at low genetic risk, but an increase of 24 % among children at high genetic risk (Jaffee et al. 2005). Thus, context of adversity in the form of physical maltreatment seemed to interact with child genetic vulnerability to predict conduct problems in early childhood (Jaffee et al. 2005). A second study investigated G-E interactions linking three early environmental indices (i.e., family chaos, instructive vs. informal parent-child communication) with verbal ability in a sample of 4-year twins (Asbury et al. 2005). Heritability for verbal ability was greater in high-risk environments, all in the direction of diathesis-stress models (i.e., high family chaos, high instructive communication, low informal communication), rather than in low-risk environments (i.e., low family chaos, less instructive communication and more informal communication) environments, suggesting increased heritability in high-adversity environments in the case of verbal ability (Asbury et al. 2005).

Evidence for a G-E interaction linking early familial adversity (as assessed via a composite score of seven prenatal and postnatal risk factors) and cortisol reactivity was also found in a sample of 346 19-month twins (Ouellet-Morin et al. 2008). In that initial paper, in low-adversity settings, genetic and non-shared environmental factors accounted for cortisol reactivity, with heritability explaining the similarity observed within twin pairs. Under conditions of high adversity, shared and unique environmental factors accounted for variance in cortisol reactivity. In other words, genetic risk for cortisol reactivity was found in low-adversity settings, but not in high-adversity settings. In a second paper on the same sample, this time focusing on cortisol data at 6 months (i.e., one year earlier), an inverse pattern was found: significant heritability for morning cortisol was found under high family adversity, but not in low adversity (Ouellet-Morin et al. 2009). Taken together, these studies indicate a shift in the G-E etiology of the physiological stress response in the context of adversity; consistent with the diathesis-stress model, gene expression (i.e., heritability) was significant at 6 months, but was shut down and replaced by shared environment at 19 months, where genetic factors now played a role in the context of low adversity. This pattern of results indicates that G-E contributions to the cortisol response vary as a function of family adversity (i.e., thus statistically a G-E), but also as a function of age. This evolving pattern of G-E is consistent with the progressive establishment of an environmental programming process in the course of the second year of life (Ouellet-Morin et al. 2009). As the main proximal environment of the child, early parenting is likely to be involved. However, further research is necessary as these studies used broad indicators of early environment, including but not limited to early parenting practices.

Findings from twin studies suggest that early high-risk environments interact with child genetic risk to predict various behavioral, physiological and cognitive outcomes. Some environments seem to increase heritability for various outcomes; in line with diathesis-stress models. In other cases, adverse environments, such as those in which economic and social hardship promote enduring contextual stress (Ouellet-Morin et al. 2008), may supersede genetic contribution to other outcomes (e.g., physiological stress response), perhaps through epigenetic processes. Yet, it is not clear whether similar G-E processes operate for other phenotypes during infancy. Further longitudinal research is needed at this time, as single assessments may be of little help in determining sequential variations in such G-E processes.

2.12.2 Adoption Studies

Adoption studies documenting G-E interactions typically test moderation of child genetic vulnerability (as assessed through birth parent characteristics and/or child temperament) on the association between early environment (assessed through adoptive parents' behavior, quality of the home environment, etc.) and a child outcome. Significant moderation of child genetic risk suggests a G-E interaction.

One prospective adoption study using adoptive parent(s)-birth parent(s)-adopted infant triads found interactions between infant genetic risk for behavior problems (i.e., birth mother's history of depression) and adoptive mother's structured parenting on infants' behavior problems (mean age = 18 months; Leve et al. 2009). This pattern of results suggests that the contribution of structured parenting to infant behavior problems varies as a function of child genetic risk (Leve et al. 2009). Using a similar method, another study found that infants at genetic risk for externalizing problems (i.e., birth parents' externalizing behavior) showed heightened attention to frustrating events only when the adoptive mother had higher levels of anxious/depressive symptoms (mean age = 9 months; Leve et al. 2010). A similar pattern of results emerged in yet another study testing the interaction of adoptive parents' depression and responsiveness with genetic risk for depression (i.e., birth mother's history of depression) in predicting fussiness in 9–18-month adopted children (Natsuaki et al. 2010). Independent contribution of adoptive mothers' depression to infant fussiness was found, as well as a significant interaction between birth mothers' depression and adoptive mothers' responsiveness. Indeed, children of birth mothers with depression showed higher levels of fussiness at 18 months when adoptive mothers had been less responsive (Natsuaki et al. 2010). Another study found moderation of birth mother anger/frustration on the link between adoptive parental harsh discipline (9 months) and infant anger/frustration (18 months; Rhoades et al. 2009). Yet another study found moderation of externalizing problems at 3 years on the link between adoptive mother's over-reactive parenting (9 months) and infant negative emotionality (18 and 27 months), as well as moderation of birth mother negative emotionality on the association of adoptive mother's over-reactive parenting with the same outcome (Lipscomb et al. 2012).

Taken together, findings from prospective cross-sectional and longitudinal adoption studies suggest that adopted children at genetic risk for several developmental problems might inherit specific vulnerabilities that makes them more sensitive to environmental risk factors (e.g., adoptive mother's harshness, depression), suggesting significant G-E interaction, although independent contributions of infant genetic risk (e.g., birth parents' history of affective or externalizing problems) are occasional.

2.12.3 Linkage Studies

More evidence of G-E interplay in early parenting comes from molecular studies testing for interactions between gene variations and measured risk factors in predicting a developmental outcome. Two approaches allow testing of such G-E interactions in molecular designs: (1) to assess the moderating effect of *child genotype* on the association of a putative environmental risk factor (e.g., parenting) with a child outcome; (2) to assess the moderating effect of *parental genotype* on the association of a putative risk factor—in the child or in the parent—with a parental outcome (e.g., parenting). Research using infant and parent molecular markers has examined

G-E interactions involving genes modulators of self-regulatory capacities during stressful social situations, social bond formation (i.e., DRD4, DRD2, 5-HTT, MR, FKBP5, COMT, DAT1), as well as externalizing problems (i.e., MAOA).

2.12.4 DRD4 and COMT

Molecular genetic studies have found significant interactions between the presence of the DRD4 7-repeat (i.e., long allele) and early maternal care (i.e., sensitive parenting, affective communication) or very close correlates (e.g., parental stress) in predicting various outcomes in infancy: externalizing behavior (mean age = 10 months; Bakermans-Kranenburg and van IJzendoorn 2006; ages = 18–30 months; Propper et al. 2007; mean age = 3–4 years; DiLalla et al. 2009; mean age = 3 months; Zohsel et al. 2014), attachment disorganization (mean age = 12 months; Gervai et al. 2007; mean age = 14 months; Luijk et al. 2011a, b), peer problems (DiLalla et al. 2009) and temperamental sensation-seeking (mean age = 18–21 months; Sheese et al. 2007). Some of these studies were conducted in the context of differential vulnerability hypothesis. For instance, it was shown that infants carriers of the DRD4 7-repeat were differentially susceptible to early care in predicting externalizing behavior (e.g., Bakermans-Kranenburg and van IJzendoorn 2006): carriers of the DRD4 7-repeat had more externalizing problems than the non-carriers when exposed to early adverse conditions, but less externalizing problems in early adaptive conditions.

Two randomized controlled trials brought further support to the differential vulnerability hypothesis. There was a moderating role of the DRD4 7-repeat in the effect of a video-feedback intervention to promote positive parenting and sensitive discipline on 1-to-3-years infants' daily cortisol (Bakermans-Kranenburg et al. 2008a) and externalizing behavior (Bakermans-Kranenburg et al. 2008b). The intervention led to a lesser amount of daily cortisol and less externalizing problems in carriers of the long allele of the DRD4 7-repeat, but no intervention led to more daily cortisol and more externalizing problems in carriers of the same genotype, suggesting differential susceptibility as a function of DRD4 7-repeat polymorphism.

Gene-environment interactions were also investigated in a more extreme form of adverse parenting: child maltreatment. A recent study examined the extent to which variation in DRD4 and 5-HTTLPR genotype were differentially associated with the development of attachment security and disorganization in maltreated and non-maltreated 13-month infants, and the extent to which the effect of preventive interventions aimed at promoting attachment security was moderated by genes. Among maltreated infants, DRD4 and 5-HTTLPR variations had minimal associations with improvement in attachment disorganization (Cicchetti et al. 2011). However, among non-maltreated infants, both polymorphisms accounted for attachment security and disorganization at age 2 and the stability of attachment disorganization over time (Cicchetti et al. 2011). In line with earlier findings on

the cortisol response (Ouellet-Morin et al. 2008), this pattern of results suggests that early conditions of high adversity, including maltreatment, could have a programming effect on indicators of emotional regulation in the infant, overriding gene expression. However, it is unclear whether HPA axis activity, the main neurobiological underpinning of physiological regulation of stress response, is involved such associations between early maltreatment and various outcomes understood as behavioral indicators of emotional dysregulation. Still, several studies have found associations between the presence of early physical abuse and heightened cortisol stress response (e.g., Carpenter et al. 2011), which points towards the likelihood of HPA axis involvement in the above-cited G-E processes.

G-E interactions were also investigated in regard to maternal personal history. In one study, maternal unresolved loss or trauma (i.e., the quality of the processing and integration of childhood experiences of loss and/or trauma in mothers, assessed via questionnaire) was associated with infant disorganization, but only in the presence of the long allele of the DRD4 7-repeat in the 10–11-month child, suggesting infant genotype of the inter-generational transmission of maladaptive attachment-related experience (van IJzendoorn and Bakermans-Kranenburg 2006).

Two studies examined interactions between parent dopamine-system genotype and several risk factors in predicting sensitive parenting toward the infant. Specifically, COMT variations are associated with variations in emotional resilience against negative mood states (Smolka et al. 2005; van IJzendoorn et al. 2008) and in cognitive distractibility (Drabant et al. 2006). As parental sensitivity requires constant attention of the infant's signals even in stressful circumstances, low distractibility and efficient self-regulation may help parents to remain focused on their child. One study tested whether COMT and DRD4 polymorphisms moderated the negative association of levels of daily hassles with sensitive parenting (mean age = 23 months; van IJzendoorn et al. 2008). In parents with the combination leading to the least efficient dopaminergic functioning (COMT val/val or val/met, DRD4 7-repeat long allele), more daily hassles were associated with less sensitive parenting, and lower levels of daily hassles were associated with more sensitive parenting. This suggests differential susceptibility to hassles depending on parental genotype (van IJzendoorn et al. 2008). Another study tested the interaction of the DRD4 7-repeat with 6 months infants' fussy-difficult temperament in predicting sensitive parenting (Kaitz et al. 2010). Mothers with the long allele of the DRD4 7-repeat were more sensitive to fussier infants and less sensitive to less fussy infants, suggesting differential susceptibility to infant fussiness according to the DRD4 7-repeat (Kaitz et al. 2010).

Overall, molecular findings suggest that in infants, the long version of the DRD4 7-repeat polymorphism, allegedly involved in the early development of self-regulation, may increase infants' sensitivity (i.e., differential susceptibility) to adaptive or maladaptive early environments in the development of various behavioral, interpersonal and physiological outcomes. Moreover, as early conditions of extreme adversity tend to program attachment behavior over and above genetic contributions during the second year of life, early conditions of low adversity are associated with heightened DRD4 gene expression.

Furthermore, G-E interactions involving parental dopamine-system polymorphisms may explain why some parents are more and others less impacted by contextual stressors and infant irritability in responding sensitively to their offspring's signals. Variations in genes involved in regulation of emotional arousal (e.g., DRD4, COMT) may contribute to hostility of parental reactions to infant signals. However, reports in this literature appear equivocal, as one study indicates less sensitivity is linked with daily contexts of fussiness in parents carrying the DRD4 7-repeat, reflecting hostile reactions to contextual infant signals, and another study indicates more sensitivity is linked with high infant fussiness in carriers of this genotype. This apparent contradiction may be explained by the fact that, as stated earlier, enduring parenting behaviors may be a function of parents' own genetically-driven characteristics, over and above initial child effects; while time-specific negative parenting is mainly a function of contextual stressors (e.g., child evocation, temporary economic hardship, etc.; Boivin et al. 2005), regardless of parental characteristics.

2.12.5 DRD2

The dopamine receptor D2 gene (DRD2) A β 1 polymorphism has been linked with sensitivity to reward (Suhara et al. 2001), novelty seeking (Noble et al. 1998) and substance abuse disorders (Oscar-Berman and Bowirrat 2005) in adults. However, data remains inconclusive in children (see Mills-Koonce et al. 2007). In a recent study, it was hypothesized that, along with DRD4, the DRD2 gene polymorphism could be involved in infant difficultness and activity level, as assessed in the course of interactions with caregivers at 6, 12 and 36 months; both potentially evoking specific classes of parental behavior (Mills-Koonce et al. 2007). This study examined a possible interaction between the DRD2 A β 1 risk allele in mothers and children, and maternal sensitivity in predicting subsequent child affective problems (Mills-Koonce et al. 2007). Evidence was found for a moderating role of child genotype in the association between maternal sensitivity and later child affective problems (Mills-Koonce et al. 2007). A second study from the same group revealed that the DRD2 A β 1 allele interacted with insensitive parenting to predict low vagal withdrawal in response to maternal separation during the first year of life (Propper et al. 2008). These findings suggest that DRD2 genotype moderates associations of parental sensitivity with early behavioral and physiological indicators of emotional regulation, thus making the infant vulnerable to specific environments in the development of affective regulation impairments (see above).

2.12.6 DAT1

Activation of the mesolimbic dopamine tract is necessary for maternal behavior in rats (see Numan 2007), and DAT1 binding in the nucleus accumbens is correlated

with maternal behavior in rats (Champagne et al. 2004). Inversely, DAT1 knock-out in mice is associated with disrupted maternal behavior (Spielewoy et al. 2000). Robust data is however still lacking in adult humans. One study tested the association of the 40-bp variable number tandem repeat polymorphism of the dopamine transporter (DAT1) gene with three dimensions of observed parenting: positive parenting, negative parenting, total maternal commands (Lee et al. 2010). A significant interaction was found between maternal DAT1 and 5-year children disruptive behavior, as the association between DAT1 and negative parenting was stronger among mothers whose children were highly disruptive during a mother-child interaction task (Lee et al. 2010). Significant non-additive associations were also found between maternal DAT1 and both negative parenting and total commands during the same task, even after controlling for demographic factors, maternal psychopathology and disruptive child behavior (Lee et al. 2010). This pattern of results suggests that maternal genetic vulnerability to maladaptive parenting is partly moderated by infant difficultness. Thus, more difficult infants (i.e., fussy, difficult) may trigger parental genetic vulnerabilities to predict the use of adverse (i.e., negative, harsh) parenting practices during early parent-child interactions.

2.12.7 5-HTT

Individuals who are either homozygous for the short allele (ss) or heterozygous (sl) of the 5-HTTLPR have been found to be at risk for a range of emotional and behavioral maladaptive conditions such as under-regulated, impulsive, aggressive and risk-taking behavior, executive function deficits, alcohol use, as well as depressive/anxious symptoms (e.g., Brown and Hariri 2006; Lesch and Bengel 1996; Lucki 1998; Posner et al. 2007). Again, in accordance to a self-regulation framework, 5-HTT variations could modulate infant self-regulatory capacities. Such early capacities are susceptible to elicit reactions from the caregiving environment, thus shaping G-E correlations between early parenting and infant behavior. Moreover, human and macaque evidence suggests complex associations of 5-HTT genotypes with activation of brain regions involved in imitation, social cognition and communication (e.g., Canli and Lesch 2007; Watson et al. 2009). In light of this evidence, 5-HTT variations may have adaptive or maladaptive consequences in several interpersonal contexts, such as during mother-infant interactions (Mileva-Seitz et al. 2011).

So far, molecular studies have found significant interactions between variations in infant 5-HTTLPR polymorphism and early maternal care (i.e., responsiveness, quality of parenting behavior) in predicting attachment security (mean age = 7 months; Barry et al. 2008), attachment disorganization (mean age = 12 months; Spangler et al. 2009) and later negative emotionality and fear (mean age = 18 months; Pauli-Pott et al. 2009). Significant interactions were also found with child attachment-related experiences (i.e., child-mother attachment relationship, attachment representation, etc.) in predicting alpha amylase response to separation (ages = 12

to 18 months; Frigerio et al. 2009), electrodermal reactivity (mean age = 7 years; Gilissen et al. 2008) and self-regulation (mean age = 15 months; Kochanska et al. 2009). These findings indicate that the quality of early care and of infant-caregiver attachment relationship both serve to amplify or offset the risk conferred by the 5-HTT genotype in the development of interpersonal (i.e., attachment disorganization), behavioral (i.e., self-regulation, negative emotionality) and physiological (i.e., electrodermal reactivity, alpha amylase reactivity) child outcomes which possibly have common impairments in regulation of emotional arousal.

One study tested if mothers' early life experiences and 5-HTT genotype interact in predicting self-reported sensitive parenting (mean age = 6 months; Mileva-Seitz et al. 2011). Main contributions and significant G-E interactions were found: mothers with no S or L (G) alleles oriented away more frequently from their babies if they also reported more negative early care, and mothers with the S allele and with positive early care scored higher on ratings of perceived attachment to their infant. Regression results also showed that with increasing care quality, mothers with the L(A)L(A) genotype (no S or L(G) allele) oriented away less frequently, while S or L(G) allele carriers showed no significant change. In contrast, with increasing early care quality, L(A)L(A) (no S or L(G) allele) mothers scored lower on perceived attachment to their infants, whereas S or L(G) allele carrying mothers scored higher (Mileva-Seitz et al. 2011).

2.12.8 MAOA

The MAOA gene encodes the MAOA enzyme, which helps metabolizing neurotransmitters such as norepinephrine, serotonin and dopamine (Shih et al. 1999). In adult humans, genetic deficiencies in MAOA activity have been linked with male antisocial behavior (e.g., Brunner and Nelen 1993). As with the DRD2 gene polymorphism, data remains inconclusive in children. Two studies have tested G-E interactions involving the MAOA gene during infancy. In their seminal study, Caspi and colleagues found that MAOA gene polymorphism moderated the association of maltreatment at 3 years with adult antisocial behavior, pointing that maltreatment predicted antisocial behavior only when there was an infant genetic vulnerability in metabolizing neurotransmitters involved in emotion regulation (Caspi et al. 2002). Another study found that, in girls, MAOA-LPR interacted with stressful life events and family adversity (early parenting being likely involved in both risk factors) from 6 months to 3½ years to predict hyperactivity at ages 4 and 7 (Enoch et al. 2012). In boys, the interaction between MAOA-LPR and stressful life events between 1½ and 2½ years predicted hyperactivity at age 7 (Enoch et al. 2012). This is in line with the well-documented role of MAOA in overactive and impulsive behavior (Brunner and Nelen 1993).

2.12.9 MR

The glucocorticoid receptor (GR) and the mineralocorticoid receptor (MR) have been implicated in the variability of HPA axis responses to social stressors

(DeRijk and De Kloet 2008). Their role in infants' behavioral regulation during their very first interpersonal relationships is thus theoretically plausible. One study found a significant interaction between the minor MR allele and sensitive responsiveness during infancy in predicting attachment security (mean age = 14 months; Luijk et al. 2011a, b). Carriers of the minor MR allele (vs. carriers of the major allele) had a more secure attachment if their mothers showed more sensitive responsiveness and a less secure attachment if their mothers showed more insensitivity, suggesting differential susceptibility to sensitive parenting according to MR genotype (Luijk et al. 2011a, b).

2.12.10 FKBP5

As differences in physiological stress response during the SS have been predominantly attributed to the quality of attachment (Oosterman and Schuengel 2007) and that genetic factors may play a role in explaining variance in HPA axis activity (Steptoe et al. 2009), one study tested the interaction between parent-infant attachment and the minor allele of the haplotype of FKBP5 (rs1360780) in predicting cortisol reactivity during the SS (mean age = 14 months; Luijk et al. 2010). A main contribution of FKBP5 rs1360780 on cortisol reactivity was found. Moreover, a significant interaction was found between insecure-resistant attachment and FKBP5 rs1360780 in predicting the same outcome. This indicates a double-risk for heightened cortisol reactivity levels during the SS in infants carrying the minor allele of the FKBP5 and an insecure-resistant attachment relationship with their mother (Luijk et al. 2010). Thus, the early development of physiological arousal in stressful social situations is mediated by the additive *and* joint contributions of genotype and early interactions with caregivers.

2.12.11 COMT

Carrying the minor allele of numerous dopaminergic system genes has been linked to infant difficult temperament (see Ebstein 2006) and ADHD (e.g., Faraone and Khan 2006). Although temperament was not found related to attachment security, it might be involved in infants' activity levels during interactions with caregivers. Moreover, a protective effect has been reported for COMT heterozygotes for Val/Met alleles (vs. homozygotes Val/Met) in the form of dopamine levels associated with optimal neonatal neurobehavioral features (Wahlstrom et al. 2010). Neonatal neurobehavioral organization, as assessed via examiner-rated scales, was linked to more secure attachment (Grossmann et al. 1985) and less attachment disorganization (Spangler et al. 1996). Therefore, distal associations between COMT genotype and infant attachment make this gene a potential candidate for the study of complex G-E interactions involving early parenting.

One study found a significant interaction between COMT and parental sensitivity in explaining variance on infant disorganization (mean age = 14 months; Luijk et al. 2011a, b). Yet, this finding could not be replicated across samples (Luijk et al. 2011a, b). Another study found that COMT significantly interacted with infant-parent attachment in the SS to predict alpha amylase basal levels (ages = 12–18 months; Frigerio et al. 2009). These findings suggest that COMT polymorphism may increase infant susceptibility to specific environments in predicting dysregulation of arousal during a stressful situation (i.e., SS) and basal stress level (i.e., alpha amylase).

Taken together, findings from linkage studies indicate that specific variations in infant dopaminergic and serotonergic genotype, possibly through their contribution to infant temperament and self-regulation, moderate the association of early parenting with several behavioral, physiological and cognitive child outcomes; thereby creating a positive—or otherwise adverse—context for the child's later psychological adjustment. Several of these G-E interactions were found in the context of the differential susceptibility hypothesis.

On the other hand, variations in parental genotype, also involving genes from the dopaminergic and serotonergic systems linked to self-regulatory capacities and emotional resilience in stressful situations, moderate the association of parental personal history, contextual stressors and infant characteristics with specific parental practices. Such G-E interactions may explain why some parents are more and others less impacted by child characteristics and stressful contexts in responding adaptively to their offspring's signals. Thus, robust evidence points to the importance of child *and* parental genetic risk (as well as their interaction) as moderators of putative risk factors in the child and in the parent.

While evidence of significant contributions of child genotype to features of the early caregiving environment suggests evocation processes in the form of child effects, evidence of unique parental genetic contribution to early parenting suggests partial independence of such child-driven evocations and point to the importance of parents' own characteristics in predicting their child-rearing practices. However, the reader should still bear in mind that infant and parent genetic risk are not necessarily independent, as shared genetic vulnerabilities with the child could account for parental involvement in specific practices. Thus, while evidence of child effects and independent contributions of maternal genotype represent distinct etiological processes, both may often coincide and jointly predict parental behavior, thereby shaping G-E interactions.

2.13 Conclusion

As this review shows, behavioral-genetics studies can provide comprehensive understanding of the nature of early parenting and its contribution to infant socio-emotional development. By disentangling genetic from environmental variance, important theoretical questions about the developmental role of early parenting

and its interaction with child and parental genetic risk may be investigated. Also, genetically informed data helps specifying the direction and magnitude of developmental associations between early parenting and child development. In the context of the present chapter, we showed that *both* child and parental genotypes have a unique contribution *and* interact to predict multiple developmental problems in the child. However, a wide range of adaptive parenting practices are accounted for by parental characteristics and life experiences, whereas child heritable characteristics may account for specific, negative parenting practices in the normative range (but not in the more severe range). Such genetically informed studies may inform the prevention of child developmental problems, as well as early interventions promoting adaptive parent-infant interactions. The actual evidence suggests that interventions promoting sensitive and warm parenting should mainly focus on parental characteristics, perceptions and behaviors, while interventions promoting positive parent-child interactions may center both on child temperamental characteristics (and their purported effects on the caregiving environment) and parent training. Still, there are several methodological limitations inherent to the reviewed studies that should be considered for future research. We close this chapter by outlining these methodological caveats. Finally, we examine future directions in genetically sensitive studies of early parenting.

Many behavioral-genetics studies involving early parenting and child outcomes rely on parent or teacher reports to facilitate data collection and to reach the power necessary to detect complex genetic and environmental contributions. This approach, however, may produce biased results with respect not only to the nature of parent-child interactions (i.e., there is considerable inter-individual variability in perceptions of interpersonal relationships), but also in terms of their underlying etiology (e.g., parents may overestimate similarity between identical twins, thus artificially bolstering heritability estimates; Gervai 2009). The use of parent reports may also be problematic when the same person assesses both twins of the same family, as such assessments may be more informative of the parents' cognitive biases and beliefs toward their children—or else, of features of the family environment—than of children's actual characteristics. Thus, the fact that the characteristics of the twins and the features of the environment are not assessed independently for each twin may yield biased estimates. Despite the potentially high expenses and logistic challenges, twin studies might benefit from the use of multiple and independent assessments of child behavior (Gervai 2009). For that matter, observational data, which allows the investigation of micro-processes within parent-child interactions, may be used more systematically in future research. However, as stated earlier, investigators should take into account the fact that within experimental parent-infant interactions, parental behavior may be somewhat programmed by experimental context; thus falsely enhancing shared environmental variance (De Wolff and van IJzendoorn 1997). One adequate, although burdensome, way to estimate within-family differences in such contexts would be to conduct more than one assessment of observed parenting within a single design in order to control for contextual factors (e.g., unfamiliarity, experimental stress).

Another important problem with existing research, especially twin studies of infant attachment, concerns the limitations in statistical power (Roisman and Fraley 2006). Reliance on small samples is typical of the methodology based on detecting significant differences between twin correlations, although it considerably precludes generalization of results. Low statistical power is also an inherent methodological problem in molecular linkage studies, as the polygenic nature of inheritance of complex interpersonal phenotypes and small samples tend to limit replication of positive findings linking single gene variations with parenting practices or attachment behavior. As stated earlier, negative findings may reflect the low power of individual studies that, when combined, could yield a significant albeit small effect. Thus, although meta-analytic reviews of linkage studies of early parenting and infant attachment would be an appealing option for future research, researchers would also benefit from the long-term use of pooled genotyped samples, gathered from molecular genetics studies conducted across multiple countries. Combining samples from a variety of populations would partly control for population stratification (i.e., selection bias) and increase statistical power of future studies.

Additionally, although G-E correlations are hypothesized to cumulate and thereby become more important as the child grows up (Scarr and McCartney 1983), there is little evidence to support this assertion because of the lack of genetically informative longitudinal data on early parenting. Thus, future twin, adoption and linkage studies should use longitudinal designs with multiple measurements through infancy and early childhood in a more systematic fashion, in order to assess this purportedly increasing magnitude of G-E correlations. In addition, bivariate twin designs can show evidence of G-E correlations, but cannot discriminate between passive and evocative G-E correlations; which considerably restricts interpretation of results. Only adoption studies—through the assessment of birth parent(s)-adoptive parent(s)-adopted infant triads—can answer this question, but such designs cannot statistically disentangle shared and non-shared environmental sources of inter-individual variance, which also limits interpretation of results. To overcome this obstacle, future genetically sensitive studies of early parenting may, for instance, focus on validating G-E correlation evidence by assessing more systematically concordance of parent and infant genotype of interest or inferred genetic risk (e.g., indicators of emotional dysregulation).

2.13.1 Future Directions

Exciting avenues for future research on G-E processes in the context of early parenting may be offered by the better identification of the specific “causal factors” that lead low risk (i.e., sensitive, secure, positive) parents to have secure and well-adjusted children, given that observations of parenting quality are only moderately correlated with ratings of infant attachment security (e.g., Bokhorst et al. 2003; De Wolff and van IJzendoorn 1997; Fearon et al. 2006), as well as evidence

that non-shared environmental processes account for a substantial proportion of the variation in infant attachment security (e.g., O'Connor and Croft 2000) and its covariation with parenting quality (Roisman and Fraley 2008). Certain authors (e.g., Fearon et al. 2006; Roisman and Fraley 2008) concur that these findings present a challenge to attachment and early parenting researchers who, until recently, have almost solely focused on identifying the antecedents of attachment security in variations in parenting quality assumed to be largely shared within families. These specific “causal factors” could be identified (and then incorporated in genetically sensitive designs), for a start, through the study other theoretical models of parent-infant interactions than attachment theory; for instance, parental reflective functioning (e.g., Grienemberger et al. 2005; Slade 2006), parenting styles (e.g., Cheah et al. 2009), etc.

On the other hand, as economic and psychosocial hardship and adversity may create considerable contextual stress and thus provide a negative background for early parent-infant interactions (Lee et al. 2011; McConnell et al. 2011; McLoyd 1998) which may be otherwise adaptive, “causal factors” may be investigated more systematically within behavior-genetics designs in the form of psychosocial risk indicators (but for a few exceptions, see Asbury et al. 2006; Ouellet-Morin et al. 2008) such as family income, social support, familial history of substance abuse disorders or parents' education. Identification of specific mechanisms through which psychosocial adversity may affect early interactions with the infant (e.g., creation of a sense of hopelessness in the parent, parental anger/frustration or depressive symptoms, lesser amount of time spent with the infant, fatigue) would also be necessary. That is to say, a more competent identification of such factors of environmental nature may considerably impact results from studies investigating G-E processes in the context of early parent-infant interactions, as the genetic-environmental etiology of the covariance between two modestly related phenotypes may differ from the genetic-environmental etiology of the covariance between two strongly related phenotypes. Precisely, there may be a positive association between the adequacy of a “causal factor” embedded in the infant's early environment and the odds of finding significant shared environmental mediation. For instance, the well-documented prominence of shared environmental variance on infant attachment behavior in univariate twin studies (e.g., Bakermans-Kranenburg and van IJzendoorn 2004; Roisman and Fraley 2006) may potentially be consolidated if more proficient environmental factors were identified and incorporated into bivariate twin designs.

As this review shows, both parental and infant genotype—interfaced with environmental putative risk factors—may play a role in the development of early parental practices and various child outcomes. Although such findings are stimulating, specific pathways through which genotype contributes to interpersonal behavior in parents and children during infancy often remain elusive. For that matter, the detection of different functional variants of specific genes is a first step to examine causal differences among alleles. However, these functional differences may be embedded in a complex, multifaceted system involving at least three levels of analysis: (1) the functional activity of the gene product itself; (2) the levels of

its expression in different brain circuits or at different times during early childhood, and (3) its differential expression to environmental risk factors (e.g., Chen et al. 2011). A deeper understanding of these neurochemical processes may help clarify the G-E processes involved in early parent-infant interactions that contribute to individual differences in both adaptive and maladaptive socio-emotional development (Chen et al. 2011).

Moreover, recent human and animal research (though it cannot be translated directly to humans) suggests involvement of early parenting in epigenetic regulation in the human brain (Gervai 2009). For instance, the methylation pattern of the promoter of glucocorticoid receptor gene in the hippocampus of suicide victims with a history of childhood abuse differs from that of suicide victims with no childhood abuse (McGowan et al. 2009). Thus, there is a good reason for hypothesising that epigenetic modification of gene expression plays a role in the development of early parent-infant relationship, even if the study of such processes in human infants is currently not feasible. This avenue may offer an opportunity to gain deeper knowledge on intergenerational transmission of attachment and parenting (Gervai 2009).

In conclusion, behavior-genetics research on early parenting is still under development, as new compelling evidence surfaces monthly. The initial reports clearly show the potential of behavior genetics to deepen our understanding of the determinants and consequences of early parenting. Through the continuing study of G-E processes, this line of research will likely help to clarify the unique contribution of early parenting and child-parent attachment relationship to child functioning. The main challenge now lies in our capacity to use these powerful tools to understand the complex G-E interplay underlying the multifaceted aspects of early parenting and infant attachment in a fully developmental perspective.

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