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Parenting in the Context of the Child: Genetic and Social Processes

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Abstract

The focus on the role of parenting in child development has a long-standing history. When measures of parenting precede changes in child development, researchers typically infer a causal role of parenting practices and attitudes on child development. However, this research is usually conducted with parents raising their own biological offspring. Such research designs cannot account for the effects of genes that are common to parents and children, nor for genetically influenced traits in children that influence how they are parented and how parenting affects them. The aim of this monograph is to provide a clearer view of parenting by synthesizing findings from Early Growth and Development Study (EGDS).

EGDS is a longitudinal study of adopted children, their birth parents, and their rearing parents studied across infancy and childhood. Families (N = 561) were recruited in the United States through adoption agencies between 2000–2010. Data collection began when adoptees were 9 months old (males = 57.2%; White 54.5%, Black 13.2%, Hispanic/Latinx 13.4%, Multiracial 17.8%, other 1.1%). The median child age at adoption placement was 2 days (M = 5.58, SD = 11.32). Adoptive parents were in their 30s and predominantly White, coming from upper-middle- or upper-class backgrounds with high educational attainment (a mode at 4-year college or graduate degree). Adoptive parents were mostly heterosexual couples, married at the beginning of the project. The birth parent sample was more racially and ethnically diverse, but the majority (70%) were White. At the beginning of the study, most birth mothers and fathers were in their 20s, with a mode of educational attainment at high school degree, and few of them were married. We

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have been following these family members over time, assessing their genetic influences, prenatal environment, rearing environment, and child development.

Controlling for effects of genes common to parents and children, we confirmed some previously reported associations between parenting, parent psychopathology, and marital adjustment in relation to child problematic and prosocial behavior. We also observed effects of children's heritable characteristics, characteristics thought to be transmitted from parent to child by genetic means, on their parents and how those effects contributed to subsequent child development. For example, we found that genetically influenced child impulsivity and social withdrawal both elicited harsh parenting, whereas a genetically influenced sunny disposition elicited parental warmth. We found numerous instances of children's genetically influenced characteristics that enhanced positive parental influences on child development or that protected them from harsh parenting. Integrating our findings, we propose a new, genetically informed process model of parenting. We posit that parents implicitly or explicitly detect genetically influenced liabilities and assets in their children. We also suggest future research into factors such as marital adjustment, that favor parents responding with appropriate protection or enhancement. Our findings illustrate a productive use of genetic information in prevention research: helping parents respond effectively to a profile of child strengths and challenges rather than using genetic information simply to identify some children unresponsive to current preventive interventions.

Chapter I: The Prospective Parent-Offspring Adoption Design: An Introduction to the Monograph

"Foolishness is bound in the heart of the child, but the rod of discipline will drive it far from him." (Proverbs 22:15). For millennia, clergy and laity alike believed in the strong influence of parenting and parents' mental health on the development of their children. Careful observation, supplementing ancient beliefs, noted a variety of other influences on children's development, from neighborhood to peer group to day care and schools. However, the most serious challenges to ancient beliefs have come from the field of genetics. First were challenges to how mental health of parents may affect children's development. For example, children of parents with alcoholism are likely to have alcohol problems only if they are biological offspring, but not if they are adopted, suggesting that the transmission of alcoholism from parent to child is genetic (Marmorstein, Iacono, & McGue, 2012). Second, and more surprising, parents' genetic makeup appears to influence their child rearing practices, such as the harshness of their discipline (Klahr & Burt, 2014). Following from this were data suggesting that observed relationships between parenting and child development might be due to genetic factors that influenced parenting and, when passed down to offspring, influenced their behavioral development. Currently, it is urgent that we clarify in what ways parenting matters for children by using research designs that allow a fuller understanding of the role of both genetic and environmental mechanisms in the relationships between parents and their children. One major approach to this challenge is the study of relationships among adopted children and their birth and rearing parents. This monograph presents results and applications from a prospective parent-offspring adoption study called the Early Growth and Development Study (EGDS; Leve et al., 2019). We have studied birth parents, the children they placed for adoption, and the adopted children's

rearing parents. We began our study during the adopted children's infancy and have continued our observations through adolescence. Our plans are to continue studying these children well into their adult years. Within this illuminating design, we have been able to observe the interplay of genetic and environmental mechanisms that account for the transmission of behavioral patterns from parent to child and from child to parent. The overall aims of this monograph are to summarize the unique features of this type of adoption design, to summarize what it can reveal about parent and child effects on children's behavioral development, and to integrate our findings into a genetically informed model of family process. We do so by describing and synthesizing exemplar findings from the study's published papers. We include a final set of chapters that present a new, genetically informed framework and that link our findings to implications for prevention and intervention, with the ultimate objective of sufficiently applying knowledge to inform the development of new initiatives that can modify the developmental trajectories of maladaptive behaviors.

This monograph is divided into nine chapters. In this first chapter, we briefly describe the history of adoption research, current perspectives on family transmission mechanisms, and the opportunities afforded by the design of the EGDS. Chapter II describes the EGDS design, measures, and analytic approaches. Chapter III discusses associations between parenting and parental characteristics and child development, illustrating how our study removes the confounding effects of shared genes between parent and child. This feature of the adoption study is a design element not possible when children are raised by their biological parents. Chapter IV describes a second feature of the adoption design: the ability to examine child effects on their rearing parents and on their rearing environment. We delineate how these child effects can sometimes be traced back to specific genetically influenced characteristics that children have inherited from their birth parents, and how child effects can initiate a dynamic and transactional process that serves to reinforce the child's trajectory towards problematic or adaptive behavior. Chapter V presents the third methodological advantage provided by the adoption design: the ability to examine whether inherited qualities in children can modify the impact on them of positive and negative qualities of the rearing environment. We also have evidence that specific characteristics of the rearing environment can modify child behavior (Hyde et al., 2016; Waller et al., 2016). The longitudinal effects of rearing environments and reciprocal parent-child interactions are the focus of Chapter VI. Chapter VII leverages the findings from Chapters III – VI to outline a new, genetically informed model of parent-child relationships. The monograph concludes (Chapter VIII) by linking findings from EGDS to preventive interventions, with implications for prevention science. Chapter VIII rests on our findings from Chapters III – VI, suggesting that: (1) children bring characteristics to their families that are influenced by genetic factors and by the prenatal environment; (2) rearing parents show evidence of responding to these characteristics in their direct behavior towards their children, in their relationships with their spouse or partner, and in their self-descriptions as parents; and (3) some parents provide their children with an environment that reinforces these characteristics to either optimize or hinder development; our data provide clues as to why some parents do optimize and some do not. In addition to discussing implications for prevention, Chapter VIII also presents future directions that we are pursuing by adding a sample of biological and non-biological children – siblings to the adopted children – in the adoptive and birth family homes in EGDS, and

how the inclusion of these siblings enhances our ability to link study findings to the field of prevention science. Chapter IX summarizes our findings and our inductively derived model. Throughout each chapter, we note the sample limitations of EGDS and where comparable research with more socioeconomically and racially/ethnically diverse samples exists, we discuss the extent to which our results are like or different from other studies.

We are fortunate to be conducting this work at a time when there have been great strides in the field of prevention science, specifically studies that have deployed developmental models and used experimental designs to study the effects of early preventive interventions. As we allude to in each chapter and expand upon in Chapter VIII, we focus on a set of preventive and clinical trials where the intervention methods and their timing were derived from developmental studies and where sustained intervention effects on a broad arc of child and adolescent development have been obtained (e.g., F. Campbell et al., 2014; C. P. Cowan, Cowan, & Barry, 2011; Enoch et al., 2016; Heckman, Holland, Makino, Pinto, & Rosales-Rueda, 2017; Heckman & Karapakula, 2019a, 2019b; Kellam et al., 2012; Kerr, DeGarmo, Leve, & Chamberlain, 2014; Olds et al., 2014; Rhoades, Leve, Harold, Kim, & Chamberlain, 2014; Sanders, 2012; Shaw et al., 2019; Wolchik, Tein, Sandler, & Kim, 2016). As noted, these prevention studies are *planned experiments* designed to test theories of child and adolescent development. In evaluating our own findings derived from the *natural experiment* of adoption (Rutter, 2005, 2007; Thapar & Rutter, 2019), we ask how our work and the outcome of these studies converge, focusing on how each form of experiment, planned and natural, informs the other. We think that a reciprocal exchange and comparison of findings between planned and natural experiments in child and adolescent development is an important route to transform the present state of developmental studies from one that is heavily freighted by uninterpretable associations to genuine causal science.

A Brief History of Adoption Study Research

A rich history of adoption research paved the way for our study to be successful. Nearly a century has passed since the first scientific adoption study was conducted (Burks, 1927). This study, a doctoral dissertation by Barbara Stoddard Burks under the supervision of Lewis Terman (1927), used data from adopted children to understand genetic and social influences on children's IQ. Burks collected a sample of White, middle-class California families (Goldberger, 1976); 100 of them were rearing their own children and 200 had adopted a child, most within a few months of birth. Burks measured parental and child IQ, and a "culture index" (parental education, speech habits and interests, and quality of the home library and furnishings). Her results highlight three core findings relevant to the complex transmission of intellectual advantages across generations (Burks, 1927) that remain core components of adoption research today. First, she found that correlations between the Stanford-Binet "mental age" (an approximation of total IQ) of parents and the adopted child's Stanford-Binet IQ were much lower for the adopting parent-child dyads than the biological parent-child dyads, suggesting that genetic factors account for most of parent-child similarities. Second, she identified modest but significant correlations between the "culture index" of the adopting parents and the adopted child's IQ, reflecting environmental processes, as there was no genetic relationship between the adoptive parent and child. Third, these same correlations were almost twice as high for biological families as

for adoptive families. Differences of this kind suggest that observed association between the home environment and the child's IQ are partially explained by genes common to parents and their biological offspring. That is, genetic factors in the parents that influence how they shape the home environment, when transmitted to their biological offspring, are expressed in the children's IQ scores. The Burks study was foundational to the field of adoption research, and many of the design advantages and findings of this first study resonate within the field today.

It is important to note that major controversies arose in the study of genetics and intelligence. Under the cloud of the eugenics movement and its horrifying use by the Nazis, researchers and lay people abhorred research that might be used to classify people as inferior because of innate, inborn deficits (Schulze, Fangerau, & Propping, 2004). Matters were made worse by linking genetics to racial differences in intelligence scores. However, research on the genetics of intelligence — and more broadly on the genetics of psychiatry disorders — has become increasingly acceptable for three major reasons. First, the role of genetic differences among individuals in their intellectual and psychological development can only be ascertained among individuals who share specific risk factors and cultural assets. Later in this monograph, we will summarize, for example, data that the role of genetics in individual differences in intelligence can vary dramatically between groups under severe economic distress compared to those who are economically secure. Second, and because of the first, it is now widely recognized that genetics can only account for individual differences within groups, not differences between groups. Finally, methods of genetic research have improved greatly so it is possible to recognize many environmental factors, specific to individuals, that either enhance or diminish genetic influences. This, as we will show in abundance later in this monograph, includes how children are parented. In some cases, the quality of parenting may dramatically alter the effects of genetic factors: a genetic factor ordinarily thought of as risk may be "converted" into an asset. Yet, environmental effects such as these have not been demonstrated for the inheritance of intelligence.

In the decades following Burks' seminal contribution, scores of studies have used adoption samples, to explore the dynamics of the adoption process or unique features of the development of adopted children (e.g., Brodzinsky, 2006; Grotevant, 1997). Far fewer have compared adoptive families with families where birth parents rear their own children (see the Colorado Adoption Project for an important exception; Plomin & DeFries, 1983; Rhea, Bricker, Wadsworth, & Corley, 2013). Many of these studies have compared the adoptive family environment or the developmental outcomes of the children in adoptive compared to birth families (for a recent review, see O'Brien & Zamostny, 2016). Even fewer adoption studies have been designed to address the interplay of genetic and environmental processes in the transmission of adaptive and maladaptive social characteristics from parents to their children. These few have played a decisive role in understanding parent-child relationships, especially in the domain of developmental psychopathology. For example, they provided the first widely accepted evidence of the role of genetic factors in the transmission of schizophrenia (Heston, 1966; Kety, Rosenthal, Wender, & Schulsinger, 1968; Rosenthal et al., 1968), the first delineation of the central role of genetic processes in the transmission of alcoholism from parents to children (King et al., 2009; Malone, Iacono, & McGue, 2002; Marmorstein, Iacono, & McGue, 2009), and environmental processes

in the transmission of depression (Tully, Iacono, & McGue, 2008). These studies have provided further corroboration and refinement of Burks' findings on the role of genetic processes in the transmission of cognitive abilities (Plomin, Fulker, Corley, & DeFries, 1997), and the first decisive evidence of gene by environment interaction in the development of psychopathology (Cadoret, Cain, & Crowe, 1983). For a more complete review of the central role of adoption studies in child development studies, see Reiss et al. (2016).

A major extension of Burks' design by subsequent investigators was adding data about birth parents to adoption study samples, a difficult recruitment feat that often takes years to accomplish in collaboration with community partners. In many of the studies that followed Burks and incorporated birth parent data, these data came from existing records of birth parent hospitalizations, incarceration, or adoption agency records, and reflected parental characteristics manifesting well after the child's birth (Cadoret & Cain, 1981b; Cadoret et al., 1983; Cloninger, Bohman, & Sigvardsson, 1981; Horn, Loehlin, & Willerman, 1979; Wahlberg et al., 2004). One study assessed birth mothers during their third trimester of pregnancy, with a small number enrolled and assessed postpartum (Plomin et al., 1997). Although these assessments of birth parents were limited, they supported three crucial advances. First, they provided striking evidence of how environmental factors moderated genetic influences on child development, a theme we examine in Chapter V. For example, Cadoret and colleagues (1983) showed that genetic influences on antisocial behavior in children and adults were manifest mainly when the rearing environments were adverse (e.g., the parents had severe psychopathology, were incarcerated, or were separated or divorced). Second, they provided surprising data on specific characteristics that a child at risk for severe psychopathology brings to the family. For example, Wahlberg and colleagues (1997) showed that children of mothers hospitalized for schizophrenia bring a *sensitivity to the environment* to the family, rather than a nascent thought disturbance. Indeed, adopted children with schizophrenic birth mothers who were raised in well-functioning families showed *less* evidence of thought disorder than a control group of adopted children whose birth parents had no severe psychiatric disorder, whereas those raised in adverse adoptive environment showed more evidence of thought disturbance (Wahlberg et al., 1997). Finally, access to birth parent data provided the most robust evidence for children's impact on their parents, a theme we explore in Chapter IV. For example, using the Cadoret sample, Ge and colleagues showed that genetically influenced hostile behavior in adolescents had as much or more influence on parental behavior than did parenting influences on the adolescent (Ge et al., 1996; Plomin, Corley, Caspi, Fulker, & DeFries, 1998).

The launch of the Colorado Adoption Project in 1975 made a major advance in the adoption study design by assessing birth parents directly, rather than relying solely on records or administrative data. The EGDS builds on the design elements of the Colorado Adoption Project and extends the methodological approach one step further, by assessing birth parents longitudinally over time – a design innovation that is presented in Chapter II and incorporated into the analyses and results in Chapters III – VI. To our knowledge, EGDS is the only prospective adoption study with long-term, longitudinal observation of both birth and adoptive parents (both mothers and fathers) and children.

As reviewed above, the science and methods underlying adoption study research have advanced in the century since the very first adoption study. Each generation of new adoption research has built upon the foundation laid by the adoption research that preceded it, while strengthening prior designs. This monograph reports on longitudinal observations of birth parents, rearing parents, and adopted children, with data collected up to age 15 years. We have analyzed data across childhood and report some of those results in this monograph (data collection from age 13 years onwards is still underway as of the writing of this monograph). Our adoption design magnifies the ordinary advantages of a typical longitudinal design in developmental science. We can distinguish between genetic and postnatal environmental effects, and then define whether these genetic or postnatal environmental effects are sustained across development or occur only in restricted time periods. We can also ask whether children's genetically influenced impacts on their rearing parents are transient, time-specific, or sustained. Finally, we can be more assured that genetically influenced characteristics of our birth parents, who are also followed over time and who are themselves developing young adults, are fully expressed and measured. The next section provides an overview of five specific design advances leveraged in EGDS to strengthen our conclusions about child and family processes.

Distinct Advances Made Possible by the Adoption Design

The adoption design, along with research designs using twins, is a major scientific tool in the field of *quantitative genetics.* In this field, the influence of genetic factors and environmental factors is estimated by comparing associations between individuals of known genetic relationships. As noted, the very first adoption study by Barbara Burks compared the associations between the intellectual abilities of parents and their biological offspring with the same correlations between adoptive parents and their adopted children. Since the former were higher than the latter, Burks inferred notable genetic influences on intellectual abilities. This is because parents share exactly half their genes with each biological offspring, but adoptive parents share no genes with the children they are raising. Most important among these genes are those that account for individual differences among humans. These are called "segregating genes" because the random allocation of many of them to sperm and eggs account for the contribution of genes to differences among children. The other major tool of quantitative genetics is the twin design. Here, inferences are drawn from comparing the associations of identical twins, who share all segregating genes, with fraternal twins who share approximately half of their segregating genes.

In contrast to quantitative genetics, *molecular genetics* requires a direct identification of specific genes. Genetic influences on a specific characteristic are assumed if there is notable correlation between a measured gene, or of a set of genes, and that characteristic. As we will discuss, the two approaches often do not align. Quantitative genetics, because its computations reflect the influence of the whole genome, often suggests more genetic influence in a human trait than do molecular techniques. There is a consensus that these discrepancies are due to undiscovered genes or non-genetic portions of the chromosome that influence a characteristic or to the unmeasured effects of interactions among genes. That is, at this stage of the science, molecular genetics often underestimates the effect of genes on human behavioral development. With each new generation of adoption research,

new insights into the parent-child relationship were made possible based on advances in the study design, research methods, and analytic approaches. At least five distinctive advances are now possible, which we further highlight in Chapters III – VI. Table 1 illustrates these advances and the associated traditional terminology used in the field of behavioral genetics to describe each feature (where relevant). Throughout this monograph, we refer to these specific processes and effects using terms that will allow us to think about the interplay of biological and social processes in the family, rather than using more traditional behavior genetics terminology. As developmental and prevention scientists, we anticipate that this language will lead more easily to translating our findings for preventive interventions. Nonetheless, some readers will find Table 1 a useful reference tool to assist translation across diverse disciplines.

The first design advance is the *removal of the influences of shared genes on associations between adoptive parents and adopted children.* When children are placed from birth with adoptive families with whom they are not genetically related, associations between rearing parent characteristics and adopted child characteristics must be attributable to postnatal environmental mechanisms, as parents and children share no segregating or individual difference genes. This feature of the adoption design is different than when children are reared by their biological parents (or are placed with biological family members after birth, including kinship foster care placements), as is traditional in the vast majority of child development research. We explore this novel design feature further and discuss the inferences this feature allows us to make in Chapter III.

The second design feature allows researchers to examine *how genetic characteristics in the child can elicit or evoke behaviors from others in the rearing environment* in predictable ways. When data from birth parents are collected, researchers can examine the correlation between birth parent and adoptive parent characteristics. Notable correlations between birth parent characteristics and the parenting behavior or family environment of rearing parents provide the strongest evidence currently available for assessing the impact on the rearing parents of their children's genetically influenced or prenatally acquired characteristics. Richard Q. Bell termed these associations "child effects" (1968). In other words, our adoption design allows us to detect effects that must originate from the child via genetic or prenatal transmission, rather than from the rearing parent. We describe this design feature and associated evidence from EGDS in Chapter IV. More traditional parent-child research has also delineated the effects of children on their parents. However, these designs cannot distinguish between those child characteristics that are intrinsic to the child from those that—earlier in development—reflected parental influence.

Third, the adoption design allows researchers to *separate prenatal from postnatal environmental influences.* As noted earlier, associations between birth parent characteristics and the development of the child they placed for adoption are ordinarily considered a good indication of the role of genetic factors in these associations. However, known effects of prenatal environments must be considered (or ruled out), and unknown effects are always a possible confound to genetic interpretations. In EGDS, we have been able to collect detailed prenatal medical records and birth parent self-report data on the prenatal period to better understand associations that are genetic in origin and are mediated through prenatal

environment, as compared to genetic influences that do not appear to be passed on through prenatal mechanisms. Without careful measurement of the prenatal environment, earlier adoption studies were unable to understand or account for these pathways, which could lead to the misspecification of genetic effects. Studies in Chapters III-VI rigorously control for prenatal environmental influences.

Fourth, although earlier adoption studies used public, clinic, or adoption records for birth parent data (e.g. Cadoret & Cain, 1981a; Cloninger et al., 1981) or birth parent data prior to placement (Plomin et al., 1997), when birth parents are followed longitudinally postpartum we can use statistical methods to *generate constructs for birth parent characteristics that incorporate multiple time points and measures,* strengthening the reliability of measurement and inferences about the behaviors and characteristics that children acquire from their birth parents. In the entire span of time since the first adoption study—nearly 100 years—the EGDS is the first adoption study to extend the observation of children placed for adoption from infancy onwards while obtaining extensive postpartum assessments across time directly from birth parents. Chapter II describes our longitudinal measurement approach, and Chapters III-VI present examples of measurement that incorporate birth parent characteristics over time.

Fifth, adoption studies that start in infancy *can provide glimpses of the earliest expression of genetic influences on child behavior.* By examining the associations between favorable or unfavorable characteristics in birth parents, and identifying associations with adopted child characteristics, we can also examine whether these early manifestations of genetic influence (correlations between birth parents and adoptees) are expressed uniformly across families or, depending on the parents' parenting and other family factors (e.g., the quality of the adoptive couple's relationship, parent's social support, or parental well-being), are expressed only in some families. Prospective longitudinal adoption studies that begin early in development can trace these interactive processes beginning in infancy. We discuss these processes and findings in Chapters V and VI. Each of these five design features allows an opportunity to provide novel insights into family processes, and the ways in which children and parents influence each other across development. In the next section, we situate our study within the context of current perspectives of family influences in the field of child development.

The Adoptive Family: Is it a Model for Family Process in Biological Families?

There are two reasons to inquire whether findings from adoptive families are generalizable to the much more numerous studies of biological families of comparable ethnicity, socioeconomic circumstance, and parental age. First, there is a growing body of literature on hormones secreted in the peripartum period, from both maternal and fetal sources, that aid in restructuring the maternal brain — especially during the gestation and birth of the first child. The brain changes are presumed crucial to mothers' early adaptation to her maternal role (see Champagne & Curley, 2016 for a review of animal studies and recent prospective studies of pregnancy brain changes in first time pregnant women; Hoekzema et al., 2017; Hoekzema et al., 2020). Adoptive mothers get no such boost from hormones associated with childbirth and lactation. Second, the path to adoption can be difficult: the stress of trying to

conceive a child, the long, difficult waits for a suitable adoption, and the stigma that may be associated with infertility and adoption may impair adoptive parents' child rearing.

However, although data remain sparse, the prevailing evidence is that neither of these two circumstances lead to qualitative differences between adoptive and biological families. First, even before they secure an adopted child, couples seeking an adoption show notable strengths in comparison to biological parents expecting their first child. They have more secure attachment to their remembered caregivers and to each other, and they report greater marital satisfaction (Pace, Santona, Zavattini, & Di Folco, 2015). Thus, despite the challenges of the adoption process, it not surprising that infants adopted soon after birth show the same level of secure attachment as children biologically related to their parents in both an earlier single study (Singer, Brodzinsky, Ramsay, Steir, & Waters, 1985) and in a more recent meta-analysis of 17 studies (van den Dries, Juffer, van Ijzendoorn, & Bakermans-Kranenburg, 2009). Exceptions in both studies were transracial and some transnational adoptions, where more adopted children showed insecure attachment in childhood than children raised by their biological parents. Although EGDS does include transracial families, it does not include families who participated in transnational adoptions.

In addition, detailed observational comparisons of adoptive and non-adoptive mothers show both to be responsive, attentive, and respond effectively and contingently to their child's social and emotional clues (Suwalsky et al., 2012; Suwalsky, Hendricks, & Bornstein, 2008). These behavioral observations have been confirmed by EEG studies of biological and adoptive mothers responding to recordings of crying babies as well as pictures of babies; both groups of mothers were clearly different than non-mothers, suggesting brain functioning had been adapted to motherhood in adoptive mothers without the hormonal changes occasioned by birth and lactation (Perez-Hernandez, Hernandez-Gonzalez, Hidalgo-Aguirre, Amezcua-Gutierrez, & Guevara, 2017). However, one study did find very subtle differences in EEG frequencies that might reflect the absence of these hormonal changes, though the functional significance of these findings seems slight (Hernandez-Gonzalez, Hidalgo-Aguirre, Guevara, Perez-Hernandez, & Amezcua-Gutierrez, 2016).

There are a few reports in the literature that compare adoptive and nonadoptive families when the adopted children are adolescents. These reports are drawn from a study where most of the adoptions were transnational. As noted, young children in transnational adoptions show less favorable attachment security to their rearing parents. Therefore, it is not surprising that parent-child relationships in adoptive families in these reports from a transnational study, when the children become adolescents, show greater conflict and less warmth in parent-child relationships (Rueter, Keyes, Iacono, & McGue, 2009; Samek & Rueter, 2011; Walkner & Rueter, 2014). Thus, these studies have uncertain relevance for our sample, where adoptions were entirely domestic.

Current Developmental Perspectives on Estimating Family Influences

Across many disciplines, the concept of transmission refers to the passing on of some vital characteristic from one person to another. The field of child development has incorporated different theoretical and methodological approaches to the study of familial transmission;

four approaches that significantly influenced the EGDS design are presented in this section: family systems, child effects, gene by environment interaction, and prenatal influences.

Family systems.—The development of a family systems perspective has transdisciplinary historical roots. As Kaslow notes (1981) its origins are in the child guidance movement in the early 20th century, but also informed by psychoanalytic theories (Bowlby, 1969; Freud, 1955; Sullivan, 1953), early family therapy (Ackerman, 1958; Bateson, Jackson, Haley, & Weakland, 1956; Minuchin & Fishman, 1979), and eventually, social learning (Patterson, 1982) perspectives. Rather than focusing on individual family members or even individual dyadic relationships, a family systems perspective views the development of both prosocial and problem behavior as a function of an embedded web of relationships among all family members (e.g., siblings, parents, parent-child, and parent-parent interactions). While early research in this area often operationalized a family systems perspective by focusing on only two family members, more recent research has been able to account for interactional processes of more than two family members.

Cross-sectional and longitudinal observations of families and their children, without attempts at intervention, have provided the bulk of quantitative evidence relevant to the family system approach and constitute a major foundation of current developmental psychology. Among the fundamental early efforts to apply a family systems approach were Rutter's early studies distinguishing the effects on child development of marital conflict from more traditional findings on the effects of parenting and parental absence (Rutter, 1971). In the half century since Rutter's prescient study, a range of studies have documented these associations of marital quality and many facets of infant, child, and adolescent development (e.g., see a recent review (Harold & Sellers, 2018). Equally central was the New York Longitudinal Study, designed and executed by the child psychiatrists Thomas and Chess. Their study focused on the interplay of child temperament and parental influences (Chess & Thomas, 1990; Chess, Thomas, Rutter, & Birch, 1963). Based on their observations, Chess and Thomas developed brief interventions for parents to help them adjust the unique features of their child's temperament; this perspective enhanced the "goodness of fit" between the child's temperament and parental expectations and behavior (Thomas & Chess, 1984). A third important turn in family systems work was a delineation of different roles and effects of mothers and fathers in the development of their children. Much research on fathers was stimulated by family experiences during World War II when the absence of fathers at war raised interest in what the child might be missing when only mothers were in the home (e.g., Sears, Pintler, & Sears, 1946). More nuanced explorations involved direct observations of the interactions of mothers and fathers with their children and an effort to characterize the unique contributions of each (e.g., Power & Parke, 1983). While most socialization studies had previously focused on the effects of maternal parenting on multiple facets of child development (e.g., Maccoby, 1994; 2007), especially in the past decade research also has attempted to identify the unique contributions of paternal caregiving on child development (Cabrera, Fagan, Wight, & Schadler, 2011; Feldman & Shaw, 2021; Paquette & Bigras, 2010; Volling, Stevenson, Safyer, Gonzalez, & Lee, 2019).

Child effects, including those that have genetic origins.—A particular and under-appreciated component of family systems research came from Richard Q. Bell in a foundational paper underscoring the impact of children on their parents, an obvious phenomenon obscured by the very long history of assuming the centrality of parental influence on children's development (1968). Bell presciently used genetic data in his analysis. He reasoned—as had Chess and Thomas—that heritable child characteristics such as different dimensions of infant temperament influenced parenting behavior.

Following Bell's initial paper and follow-up volume (1977), the child effects perspective gained additional momentum with a paper written in 1981 by Rowe that cautiously reported evidence from a tiny sample of adolescent twins, that adolescents' perceptions of the quality of parenting they had received was genetically influenced (Rowe, 1981). Two years later, Rowe published stronger evidence that adolescents' perception of their parenting was genetically influenced (1983). Although this work was quite controversial at the time, history would prove Rowe's early findings to have significant merit. In the decades that followed, research underscored genetic influences on a very broad range of measures of the social environment, from social class to marital quality to parenting (Kendler & Baker, 2007; Plomin, DeFries, Knopik, & Neiderhiser, 2016). This field-changing revelation raised the possibility that any observed association between a measure of environmental influence and a measure of child development could be confounded by genetic influences common to both. There are two ways this can happen. First, an association could be due to the same set of genetic factors that influence a parental variable, for example, harsh parenting, being passed on to a child where those same genetic factors also influence the child's development of impulsivity and aggression. In the behavioral genetics literature, this phenomenon is known as *passive gene-environment correlation*, as noted in Table 1. Second, a heritable feature in a child might evoke certain types of parenting and influence the child's own development as well, and therefore, could account for an apparent environmental effect that was, in fact, mostly or entirely attributable to genetic influences. For example, a child who was genetically at risk to be more aggressive might evoke harsh parenting from their parents. In some cases, this evoked harsh parenting, in turn, might enhance the child's liability for later impulsive and aggressive behavior. In this instance, the evocative effect of the child's heritable features must be considered part of the mechanism of genetic expression, though it is "outside the skin" (Kendler, 2001). In the genetically informed literature, this type of child effect is commonly termed *evocative gene-environment correlation* in the behavioral genetics field, as noted in Table 1. These findings, on both common gene effects and genetically influenced child effects, challenge an extensive line of research that inferred parental inferences simply from their predictive association with child outcomes (see a recent review of these genetically informed studies, (Plomin et al., 2016). Despite frequent reports on this confounding effect across two decades of research (see Jami, Hammerschlag, Bartels, & Middeldorp, 2021; Reiss, 2016 for recent reviews), most parenting studies in the developmental literature do not attend to this potential confound.

The interaction of genetic and environmental influences.—A third perspective on familial transmission evolved from research on the interaction between genetic and environmental influences. Among the earliest examples came during the 1950s, via the

discovery of phenylketonuria (PKU) by Folling (1934). This metabolic defect, arising from a single gene regulating the metabolism of a common dietary component (phenylalanine), was expressed as an intellectual disability only when children were fed food containing phenylalanine. Strong proof of this interaction of diet and genetic influence was first demonstrated in a single case clinical trial by Bickel (1953). The concepts underlying the PKU research were first translated to the world of behavioral development using adoption study designs by Cadoret's groundbreaking studies of gene by environment interaction in the development of aggression and antisocial behavior. Cadoret et al. found that adopted children at genetic risk for antisocial behavior—because their birth parents had severe antisocial and/or alcohol problems—became antisocial themselves only if reared in an adverse family environment of marital strife and/or rearing parent psychopathology (1983). This is an instance of just one of several types of genotype by environment interaction: in the presence of a genetic diathesis, an adverse environment leads to an unfavorable outcome. Closer to Chess and Thomas' concept of "goodness of fit," is the work of Wynne and colleagues in a study of children of mothers hospitalized for schizophrenia who were placed for adoption early in development. Those children reared by adopting parents whose verbal communications were confusing and contradictory developed severe thought disorder, whereas those who were raised by adopting parents with clear verbal communication had lower scores on thought disorder than the control group of adopted children without a family history of severe psychiatric disorder (Wahlberg et al., 1997). Recently, many researchers have been interested in a third type of interaction. Some data suggest that children may inherit a sensitivity not only to stress but also to favorable environments (J. Belsky, Bakermans-Kranenburg, & Van Ijzendoorn, 2007). Children with that genetic makeup might develop psychopathology if they grow up in stressful or abusive families but may have a favorable outcome if their families are warm and supportive. This type of gene by environment interaction has been termed "differential susceptibility." In subsequent chapters, we examine our own data for corroboration of this idea.

Prenatal environment influences.—A fourth conceptual approach that has informed the development of the EGDS research is a focus on the prenatal environment, and how the experiences that parents have during pregnancy might influence the developing fetus, and ultimately, affect their child's behavioral and cognitive functioning. Prenatal influences include maternal drug use during pregnancy as well as her emotional state (Hannigan et al., 2018; Yip et al., 2014). As we will show in Chapter IV, the adoption design can be used to estimate some prenatal effects on children's subsequent development. However, the rapidly growing literature on prenatal influences motivated our extensive measurement of these influences to assure that they were not confounding our estimates of genetic and postnatal environmental influences. Taken together, these major lines of inquiry—family systems, child effects, genetic analysis in the interpretation of apparent environmental influences, and prenatal environmental exposures—are now widely supported by compelling evidence from child developmental studies. This monograph focuses specifically on the prospective parent-offspring adoption design to integrate and advance these prior lines of inquiry. It is worth noting that the adoption design is one of several complementary designs that each advance the understanding of genetic and family transmission in novel ways. Before detailing the EGDS adoption design, we provide a brief review of other study designs

that can complement the parent-offspring adoption design in advancing understanding of mechanisms of family transmission.

Current Approaches to Integrating Genetic Information into the Study of Family Relationships

The last three decades of child development research have seen a surge in the number of novel study designs that integrate genetic information into the study of family relationships and child developmental outcomes. Comprehensive systematic reviews that summarize the design types and findings exist (see Jami et al., 2021), so here we provide only a brief overview of some of the recent designs and showcase their novel strengths. In doing so, our intent is to convey that the adoption design, while unique, is part of a family of designs that, when findings are considered together, can greatly advance the understanding of the contributions of children and parents in influencing children's maladaptive and adaptive outcomes. Here, we briefly discuss the in vitro fertilization design, the children of twins design, and molecular genetics approaches.

In vitro fertilization (IVF).—IVF studies are a close relative of the parent-offspring adoption design and provide a related strategy for comparing parent-child pairs who are genetically related versus those who are not. Specifically, genetically unrelated parent-child pairs result from IVF where both sperm and egg are donated by individuals unrelated to the rearing couple (embryo donation; Harold, Elam, Lewis, Rice, & Thapar, 2012; Rice, Lewis, Harold, & Thapar, 2013). In this sense, this group of IVF families share some of the design features highlighted in Table 1. In addition, IVF designs can contain a pregnancy surrogacy group, where an embryo is implanted into the uterus of a genetically unrelated woman. Selection issues for potential surrogates notwithstanding, the surrogacy group allows for a separation of prenatal and genetic influences in unique ways not afforded by parent-offspring adoption designs like EGDS.

The children of twins (CoT) design.—CoT is a second approach that shares some attributes with the adoption design while offering other novel features related to the detection of common genes and rearing environment effects. In the CoT design, when the rearing parents are monozygotic twins, the child of one twin also shares 50% of their segregating genes with the co-twin (the child's aunt/uncle). This strategy permits estimation of the effects shared by genes independent of the shared rearing environments (passive gene-environment correlation), and, in the extended CoT approach, allows an estimate of child effects on parents that are genetic in origin (evocative gene-environment correlations). These associations are achieved by simultaneously estimating the impact of children's genes on their behavior and parents' genes on their own behaviors (Lynch et al., 2006; Marceau et al., 2013; McAdams et al., 2017; McAdams et al., 2015; Narusyte, Andershed, Neiderhiser, & Lichtenstein, 2007; Narusyte et al., 2011; Narusyte et al., 2008; O'Reilly et al., 2020).

Molecular genetic approaches.—As already noted, a third approach is to use *molecular genetic methods* to measure genetic influences in the child. This approach has yielded a rich harvest beyond twin and adoption designs—where family processes have been carefully measured—where genetic information comes from the direct assay of particular

genes whose variation in structure contributes to individual differences among individuals. These genes are set to be *polymorphic* (see Reiss, 2016). To date, the most successful approach to these measurements has been genome wide association studies (GWAS). By using exceptionally large samples, these GWAS studies have found that scores and sometimes hundreds of polymorphic genes are reliably associated with behavioral traits—each accounting for a tiny amount of variation in the associated trait. Data from GWAS allow computation of indices of genetic influence that are often referred to as either polygenic scores (PGS) or polygenic risk scores (PRS). These scores can be computed for all individuals in a sample by noting their number of favorable or risk polymorphisms weighted by the degree of association of each of those polymorphisms with the trait or characteristic of interest. These methods are now being integrated into studies of social processes in the family and in broader social processes such as stratification (e.g., D. W. Belsky et al., 2018; Wertz et al., 2019). Although quite distinct in the approach conceptually, the adoption design— through its comprehensive measurement of birth parents—is like the genome-wide approach in a GWAS. Both procedures examine significant portions of genomic effects, with the former being unable to disaggregate the effects of any single gene but typically including more comprehensive measurement of the rearing environment. The next section describes how we have leveraged the design strengths of the adoption design to create a schema for examining the role of parenting in child adjustment and maladjustment across development.

The Early Growth and Development Study (EGDS)

The EGDS is a contribution to the genre of integrated research designs that incorporate genetic information into the study of family relationships. In the rearing family, building on family systems models, we can study the separate impact of mothers and fathers, their combined influences, and the separable impact of qualities of the couple or marital relationship. Using our extensive information about birth parents, we can identify genetically influenced characteristics that the child brings to the family. Through careful measurement of birth mothers' prenatal environment and incorporation of birth father data, we can consider birth parent influences on adopted children's development that may be mediated by prenatal exposures and those that do not appear to be mediated by the prenatal exposures measured in EGDS. As noted in Table 1, throughout the monograph we will use the term *child effects.* This term emphasizes that these are effects of the child on the family system arising from characteristics that children bring to the family rather than reflecting, in whole or in part, earlier influences of the rearing family. As ordinarily used, the term "child effects" includes effects that are direct and originate from genetically influenced or prenatally acquired characteristics, and those that are an influence of the rearing family and then have a subsequent impact of their own on the family. The later form of child effects is best thought of as part of the reciprocal process between parents and children (discussed further in Chapter VII). As we discuss in Chapter III, conventional longitudinal studies of child development cannot distinguish between these two forms of child effects. For emphasis, we use the term "child effects" to refer only to characteristics that children bring to their rearing family.

Adoption design assumptions.—Before presenting a full description and visual schema of the analytic possibilities in EGDS, it is important to provide a comment on potential design assumptions that can interfere with the ability to draw causal inferences about the various aspects of family transmission if these design assumptions are not met. First, the adoption process might selectively place children into rearing families that are like their biological parents or that reflect an effort to counter the possible environment provided by biological parents with adoptive families unlike them ("selective placement"). Second, almost all adoptions in the 21st century are somewhat fluid in terms of contact and knowledge shared between the biological parents, the rearing parents, and the offspring ("adoption openness"). Third, rearing parents—even in closed adoptions—may learn key facts about the biological parents that might alter their expectation for their adopted child and influence their rearing patterns. These expectations can be engendered even in fully closed adoptions if the birth parents learn anything about the characteristics of the birth parents. Fourth, research staff assessing birth parents might influence the assessment of the rearing family. Table 2 presents each of these design assumptions, followed by information about the measurement and analytic approach used in EGDS to address all four of these challenges. Some additional detail is also provided in Chapter II when we present the EGDS measurement approach.

Analytic opportunities in EGDS: A visual schema.—The unique opportunities for analysis in EGDS are illustrated in the schemas presented in Figure 1A through 1D. Figure 1A illustrates the basic logic of the EGDS design. It is built around the separation of influences on child development that occur before birth (prenatal environments and genes passed to the child from birth parents) and those that occur after birth (via the adoptive family environment). As described in Chapter II and re-introduced in subsequent chapters, prenatal and genetic influences are measured by coding obstetrical records (prenatal influences) and by rigorous postnatal interviews of birth mothers and birth fathers using a life-history calendar approach (about the prenatal period and about current characteristics). The separation of prenatal and genetic influences from postnatal influences is reinforced by the design and assessment procedures we describe throughout the monograph. Most studies of parent-child relationships cannot account for the confound between prenatal and postnatal influences; this confound is represented by path *t* (for "traditional studies") in all four figures. Of note, associations between facets of the prenatal environment and later child development can be confounded by common gene effects, i.e. genes that influence how parents influence the prenatal environment (such as drug use) that when passed on to their offspring also influence child development. This confounding is emphasized by a recent review (Jami et al., 2021) and by a methodologic critique (Rice, Langley, Woodford, Davey Smith, & Thapar, 2018). One of our solutions to this confounding is to include in our analyses a comprehensive index of the prenatal environment that includes many factors known to affect fetal development. It is then examined as a potential mediator or moderator of genetic influences on child development, or included as a control variable. As Figure 1A shows, our estimates of genetic influences are derived from partialing out known prenatal influences. This partialing is represented by the curved line without arrows, labeled *a.* Assuming the effectiveness of this partialing, it is possible to independently estimate the influence of genetic factors and postnatal environmental influences provided by parents

on child development. A second approach, that we describe in Chapter IV, is to compare the associations with child development of birth mother and birth father scores; where the former exceeds the latter we can infer intrauterine effects. It is also important to note that we do not estimate *all* genetic influences; only those indexed by specific measures of birth parents that have been included in EGDS. In some studies, we use data just from birth mothers because we recruited birth mothers for all but five children in EGDS, in which case only the birth father was recruited. In other studies, we also included data from birth fathers, often imputing missing data for the 63% that were not recruited into the study. In Figure 1A we have shown "child outcome" at two separate time periods to emphasize schematically the longitudinal nature of our design. We could have extended this schematic to include many additional time periods (see Chapter II for measurement time points), but to ease readability, we only illustrate two. In addition, we could have represented the longitudinal measurements of birth parent and rearing parent in the same manner, but, for simplicity's sake, have not done so.

Figure 1B emphasizes the focus of Chapter III in this monograph: the effects of parenting and rearing parent characteristics on child development. In these analyses, we still measure prenatal and genetic influences, but we do so to separately estimate adoptive parenting effects free of the confound of genes shared between biological parent and child and from prenatal environmental influences. Because postnatal environmental influences are the focus of Chapter III, we have greyed out the genetic influences and prenatal pathways in the figure. The path $e_1 \cdot e_3$ reflects our analyses aimed at eliminating the effects of reverse causality (e.g., from child to parent, in this instance).

Figure 1C summarizes the aims of Chapter IV: to report findings on genetically influenced child effects on parenting and the family environment. Some of our analyses reported here infer these child effects from associations between birth parent measures and parenting or parent characteristics in the rearing families. As noted earlier in this chapter, our design permits rigorous causal reasoning from these associations because once the adoption design assumptions are met, the only explanation for the association between birth and adoptive parents is via the adopted child. In other words, the only way such associations can be observed is because a genetically influenced characteristic of the child *caused* a behavior of the rearing parent. Other analyses identify a putative child characteristic that mediates this effect as represented by the path $g_1 \cdot g_2$. Of special interest is *path* $g_1 \cdot g_2 \cdot g_3$. This pathway represents those circumstances where a genetically influenced child characteristic evokes parenting behavior that, in turn, augments the child's characteristic and related behaviors. In this case, the evoked parenting becomes part of the mechanism by which the original genetic influence is expressed (Reiss & Leve, 2007).

Figure 1D summarizes the findings presented in Chapters V and VI: the moderation of the effects of parenting by a genetically influenced child effect. The moderation is represented by paths g_4 and g_5. The data used to estimate these paths is the interaction between a birth parent measure and a measure of parenting on a measure of child development. These interactions can be interpreted as either a child effect moderating the impact of parenting on the child (Chapter V) or as a parenting effect moderating the expression of a genetic influence on the child (Chapter VI). This second possibility is represented by the path e_4. As

the monograph focuses heavily on child effects that influence the dynamics of the family, we give primary emphasis to interpreting these interactions as genetic moderations of parenting. However, Chapter VI includes examples from reports that are best understood as moderating parenting effects on genetic influences on the child.

In Chapters III-VI, we delve more deeply into the schemas presented in Figures 1A – 1D and use them to propose an integrated conceptual schema in Chapter VII. Before doing so, we provide a detailed description of our research design in Chapter II to provide readers an understanding of the measures, assessments, and analytic approaches in EGDS, including a synopsis of the limitations of EGDS adoptive families in terms of their socioeconomic and racial/ethnic diversity.

Chapter II: Design and General Methodology of the Early Growth and Development Study (EGDS)

The EGDS is a prospective parent-offspring adoption study of families linked through adoption. In 2002, the EGDS was originally launched to investigate the interplay among genetic, prenatal, and postnatal environmental influences on child development and family functioning in early childhood (see early reports on EGDS; Leve, Neiderhiser, Scaramella, & Reiss, 2010; Leve et al., 2013). An adoption-linked family (or adoption triad) consists of birth parents (birth mothers in all but 5 families, and birth fathers in 37% of adoption-linked families), adoptive parents, and an adopted child (see Figure 2, highlighted in yellow). EGDS has since expanded to include follow-up assessments in middle childhood and adolescence. In addition, two separate but interrelated samples of siblings were added (see Figure 2; Leve et al., 2019): a sample of biological siblings of EGDS adoptees raised in their birth homes (known as the Early Parenting of Children [EPoCh] sample) and any other children living in either the birth or adoptive home (Environmental influences on Child Health Outcomes; Gillman & Blaisdell, 2018). In this monograph, the primary focus is on the EGDS adopted children and their adoptive and birth parents (highlighted in yellow in Figure 2). This chapter provides an overview of recruitment, sample, and assessments of the original EGDS adoptive and birth families. These families are the focus of the empirical studies reported in Chapters III through VI. More detailed information about methods for the EGDS extension studies is available in Leve et al. (2019).

Recruitment

Recruitment of the first EGDS cohort (Cohort I) of adoptees occurred between 2003 and 2006, followed by the recruitment of the second cohort (Cohort II) of adoptees in 2007–2010 (for details see Leve et al., 2019). Similar recruitment and assessment procedures were implemented for both cohorts, with the main differences being that Cohort II recruitment began just after Cohort I recruitment ended (approximately four years after Cohort I recruitment began). To recruit the sample, the EGDS team established multiple research teams in the United States to recruit families in the Mid-Atlantic (George Washington University and The Pennsylvania State University), the West/Southwest (University of California, Davis, and University of California, Riverside), the Midwest (University of Minnesota), and the Pacific Northwest (Oregon Social Learning Center, University of

Oregon) regions. The EGDS recruitment began with the recruitment of adoption agencies into the study (N = 45 agencies in 15 states). Once adoption agencies were identified, the adoption agency staff began with outreach to potential participants. Each agency appointed a staff member who served as a liaison between the research team and participants. Liaisons identified potential families who had completed an adoption plan through their agency and met the study eligibility criteria. These criteria included (1) the adoption placement was domestic within the U.S.; (2) the infant adoption occurred within 3 months postpartum; (3) adoptive families were not biologically related relatives of the child; (4) there were no known major medical conditions (e.g., severe prematurity, need for medical surgeries); and (5) both birth and adoptive parents had English proficiency at the eight-grade level. At this first stage of recruitment, a total of 3,293 triads (adopted child, adoptive parents, and birth mother) met the study criteria.

Approximately four weeks after the child was placed in their adoptive home, the agency liaison mailed each eligible adoptive family a letter asking them permission for the EGDS to contact them (N = 2,635). Adoptive families recruited in the EGDS were often composed of two-parent households, although some single-parent families were also recruited (n = 10). Two weeks later, agency liaisons attempted to locate birth mothers whose adoptive families consented to be contacted by the EGDS (N = 1,237 birth mothers located) and called them to obtain their permission for the EGDS staff to contact them. When the birth mother agreed to be contacted by the study (N = 1,098), the liaison provided the contact information of the birth mothers to the EGDS birth parent recruiter.

The birth parent recruiters successfully recruited 864 birth mothers. After a birth mother was recruited, a separate team member was identified to recruit adoptive families linked with birth mothers who agreed to participate. Separate staff members were used to ensure a firewall, such that no information would inadvertently be shared by research staff with one's counterpart in the adoptive family (also see Chapter I, Table 2, research team bias assumption). The adoptive family recruiter used the contact information provided by the adoption agency to reach the adoptive family. Both adoptive mothers and fathers were recruited. A total of 561 adoptive families (Cohorts I and II combined) agreed to participate, which led to a final sample size of 561 EGDS adoption triads (i.e., each triad consists of an adopted child, adoptive parents, and birth mother and/or birth father). Once the birth mother and adoptive parents were recruited, the EGDS attempted to locate and recruit the birth father. There were five cases across two cohorts where birth fathers were recruited without birth mothers. The recruitment procedures for birth fathers were similar to those used for birth mother recruitment. Although the EGDS staff were only able to recruit and assess birth fathers in 37% of our adoption triad sample (n = 208), this subsample represents the most sizable sample of birth fathers in existing full adoption studies of which we are aware. The most common reasons that birth fathers were not contacted were the inability to locate them and no contact information was available from the birth mother or other sources (Leve et al., 2007). To evaluate possible systematic sampling biases, we gathered demographic information from adoption agencies on eligible participants who did not participate, and then we compared EGDS participants (N = 561 families) and eligible non-participants (N = 2,391 families whose data were available for analysis). Results, reported in Leve et al. (2013), showed no significant differences between EGDS participants

and the eligible non-participants, with a few minor exceptions with small effect sizes (*d* =.13 - .22). Compared to the eligible non-participating counterparts, participating adoptive mothers had higher educational attainment and were slightly younger. Participating adoptive fathers also had higher educational attainment, and participating birth mothers and birth fathers were younger. The primary reason for non-participation of all adult participants was the inability of the project or agency to locate them. These findings suggest that sampling bias was minimal.

Sample Description

The EGDS sample described in this monograph includes 561 adoption-linked families (with *n* = 361 in Cohort I and *n* = 200 in Cohort II): 561 adopted children, their birth mothers (*n* = 554), their birth fathers (*n* = 210), their adoptive fathers (*n* = 563), and their adoptive mothers (*n* = 570). The samples of adoptive mothers and fathers do not add up to 561 each because there are 41 same-sex parent families and additional adoptive fathers and mothers who entered the family after the initial recruitment (*n* = 14 and 4, respectively), primarily due to marital transitions within the adoptive family. Currently, the EGDS participants reside in 45 states and the District of Columbia in the U.S., as well as 8 other countries.

The demographic information of the family members by role is presented in Table 3.

As shown in Table 3, the samples of adoptive mothers and fathers were predominantly White with high educational attainment (mode: a 4-year college degree or graduate degree). Both adoptive mother and father samples were older than the birth parent sample (described later), typically in their late 30s. Most adoptive mothers and fathers were married at the outset of the project, and over 85% of them continued to be married 11 years later. As a group, adoptive families were affluent, with the median annual household income at over $100,000 at the start of the project (2002–2010) and $125–150,000 at the latest report provided by the participants (2010–2021).

The birth parent sample is more diverse than the adoptive parent sample: approximately 70% were White (see Table 3 for complete birth parent demographic information). At the start of the project, most birth mothers and fathers were in their 20s with a mode of educational attainment at a high school degree. Few of them were married at the time of placement and their median annual household income was below $25,000. The income differences between adoptive and birth homes we observed in EGDS are typical for adoption studies. As noted in our prior publications (Leve et al., 2013; Natsuaki et al., 2019) and others' work (McGue et al., 2007; Stoolmiller, 1999), adoptive families have, on average, more financial resources, and higher educational attainment than birth parents. However, the most recent assessment (currently ongoing) shows some changes in birth parents' life circumstances. Approximately half of the birth parents reported being married, and their median annual household income has grown to $25,000–40,000. The rate of college or university degree holders (2- or 4-year college or graduate degree) has increased from 7.4% at the inception of the study to 25.3% at the latest report for birth mothers and from 6.1% to 15.6% for birth fathers. Through this monograph we discuss limits to the generalizability of our findings that arise from a relatively privileged sample, especially the subsample of rearing parents.

The EGDS adoptee sample of children consists of 57.2% males and approximately 20% of children were identified as multiracial by adoptive parents (see Table 3). The median child age at adoption placement was 2 days ($M = 5.58$, $SD = 11.32$).

Timeline of EGDS Assessments and Retention Rates Through Age 11

Figure 3 illustrates the timeline of EGDS assessments and associated sample size by cohorts. The timeline is guided by child age. EGDS conducts adoptive family assessments frequently so that we can follow the child's development closely. Earlier data collection (at child ages 9, 18, and 27 months) was designed to have shorter intervals between assessments to capture the rapid development in infancy and toddlerhood. After the adopted children entered elementary school, annual assessments were conducted at age 8 or 9, with an additional focus on emerging skills and capacity to fulfill age-relevant developmental tasks, such as academic achievement and school readiness (more information in a later section). Assessments then became biannual (e.g., ages 11, 13, 15). At the time of writing, we have completed the age 11 assessments and fare nearing completion of the age 13 assessment.

Birth parents participated in an intensive data collection at 3 – 6 months postpartum (M age = 5 months postpartum), which was the earliest time point we could contact them after the placement of the child was secure and not subject to recission by the birth parents. In this initial assessment, birth parents provided detailed information about their experiences of adoption. We conducted in-person interviews with them again at 18 months to collect more information about themselves, including their behaviors, emotions, and characteristics. It is also noteworthy that the EGDS assesses birth parents in later waves because phenotypic expression of genes in birth parents may change over time.

One daunting task of prospective, longitudinal studies – especially long-term ones— is to minimize sample attrition. We have been successful at retaining families over time, maintaining a low attrition rate. We estimate the overall retention rate for both cohorts at age 11 to be at 75%. So far, recent work using later waves of data (ages 7 to 11) has shown no evidence of a systematic pattern in missingness (Ganiban et al., 2021; Natsuaki et al., 2021) with a few minor exceptions (e.g., openness was higher for families with missing data Cioffi, Griffin, et al., 2021); the Missing Completely at Random [MCAR] assumption did not hold, but data were consistent with the Missing at Random [MAR] assumption (Austerberry et al., 2021).

Our success at minimizing attrition can be attributed to several strategies we apply to the study protocol. In addition to participation financial incentives, we administer brief (15 minutes) phone interviews or mailed/emailed surveys between extensive in-person interviews. This short interview serves two purposes: to collect updated contact information and maintain rapport with the participants. Families are also asked to provide an additional contact person's information (e.g., grandparents'), which we use in case direct contact with the participant is lost. In addition, newsletters and birthday cards are sent to families annually. We also use several strategies to locate families who are lost. For instance, we use a "drive-by" option in which our interviewer travels close by to the participant's last known address by showing up at their home. We send private messages on social media, which has also been an effective way to reconnect with lost families. We offer remote participation

options (e.g., online, mail, phone) for families who have moved out of the country or with a busy schedule. We have also adjusted the in-person data collection protocol to a remote-only assessment during the COVID-19 pandemic (March 2020 - current), making study participation more accessible for families. These changes have included data collection by phone, web-based questionnaires, biospecimen collection via mail, and Zoom-based video assessments. Finally, we strive to offer flexible scheduling for interviews whenever we can (e.g., interview location, weekends, evenings). These strategies have been implemented in other long-term longitudinal studies that also found them useful in keeping the retention rate high over the decades of the study course (Ou et al., 2020).

How and What We Measure: The Core Constructs in EGDS

How we measure the core constructs.—The EGDS assessment strategy uses different sources of primary data (i.e., children, mothers, fathers, teachers, and interviewers) and methods (e.g., in-person interviews, diagnostic interviews, questionnaires, diary, observation of family interactions, collection of official records [e.g., GPA, arrest records, neighborhood-level data], medical records, and biological specimens [i.e., DNA from saliva and buccal cells, cortisol via saliva and hair, microbiome via stool samples, and hormone from hair samples]). Consistent with the tradition of multitrait-multimethod measurement (Achenbach, McConaughy, & Howell, 1987; Bauer et al., 2013; D. T. Campbell & Fiske, 1959; Kraemer et al., 2003), we have found that each reporter can potentially bring unique insights that are not readily available in other informants' reports. Additionally, having diverse sources of information allows us to mix and match the reporters in any given analysis to reduce concerns about shared source variance. For instance, we have used adoptive *fathers'* reports of parenting as a predictor of child behavior as reported by adoptive *mothers.* This difference-of-reporter design has been used in many of our publications (e.g., Leve et al., 2009; Natsuaki et al., 2010).

What we measure as our core constructs.—Table 4 provides an overview of the core constructs that EGDS assesses. Influenced by the overarching theories of development by Gottlieb (1991), Sameroff (2010), and Cicchetti & Dawson (2002), we continue to emphasize assessment at multiple levels of analysis for each family member, from genes, neuroendocrine systems, phenotypic characteristics to the environment (within and outside the family). Broadly speaking, the constructs assessed in EGDS fall into four categories: genetic influences, prenatal environment, rearing environment, and potential confounds.

Genetic influences (highlighted in gray in Table 4).: Genetic influences are estimated from birth parents' characteristics. EGDS has adopted three approaches to estimate genetic influences from birth parents' characteristics. In genetic studies, characteristics that can be directly observed are called *phenotypes.* In contrast, the *genotype* refers to an individual's genetic makeup which may or may not be associated with any given phenotype. First, some of our studies used measures of birth parent psychopathology to estimate genetic influences because biological children of parents with psychopathology often show distinctive features as early as infancy. For example, infants of depressed mothers show a distinctive pattern of negative affect, quick and intense emotional and physiological arousal, and difficulty being soothed (Tronick & Reck, 2009; Weinberg & Tronick, 1998). The profile of reactivity

described for infants of depressed mothers has now been shown to be a general risk factor not only for internalizing problems (Karevold, Roysamb, Ystrom, & Mathiesen, 2009) but also for a broad range of problem behaviors, including externalizing problems (Abulizi et al., 2017; Kostyrka-Allchorne, Wass, & Sonuga-Barke, 2020). We reasoned that these genetic factors account for transmission from parental psychopathology to infant reactivity, and the latter may serve to influence the child's development.

Second, we also drew on studies of a general liability common to major psychiatric problems. This perspective was developed from increasing awareness of the extensive concurrent and sequential comorbidity of traditional clinical syndromes (Caspi & Moffitt, 2018). The same complexity holds for parent-child similarities. For example, the offspring of parents with depressive symptoms are about as likely to show anxiety symptoms as they do depressive symptoms, the reverse being true for parents with anxiety syndromes (Weissman, 1990). These patterns of comorbidity and heterotypic sequencing led to a search for underlying latent constructs that might identify commonalities across disorders. This search has consistently led to the identification of a general psychopathology factor (Caspi & Moffitt, 2018) or p factor; for a recent discussion of this modeling effort in children, see (Rhee, Lahey, & Waldman, 2014), and recent work has shown that genes play a role in influencing this general factor (Allegrini et al., 2020). Very recently, new meta-analyses suggest that patterns of comorbidity can be decomposed into a general psychopathology factor as well as more specific transdiagnostic dimensions such as antagonism, detachment and disinhibition (Ringwald, Forbes, & Wright, 2021).

A third strategy for selecting birth parent variables is to identify underlying and stable patterns of self-regulation and environmental sensitivity that cut across traditional diagnoses. It is possible that some of these early patterns of self-regulation, particularly executive function and emotional self-regulation, are precursors to the development of general liability for psychopathology (see preliminary evidence for links between executive function, neuroticism, and general psychopathology factor in childhood and adolescence (Brandes, Herzhoff, Smack, & Tackett, 2019; Harden, 2021). The origins of this approach extend as far back as Chess and Thomas (1963), but the formative work on delineating temperaments based on behavioral observations and longitudinal study is rightly attributed to Rothbart and her colleagues (1981). Data suggest that specific temperament dimensions —such as negative emotionality or emotional dysregulation—may be an early manifestation of the genetic liability for subsequent psychopathology. An extended version of this perspective informs several analyses to be reported here: the genetic inheritance of dimensions of temperament may be a mechanism by which psychopathology or adaptative behavior is transmitted from parents to children (G. T. Smith, Atkinson, Davis, Riley, & Oltmanns, 2020).

As noted earlier, a unique aspect of the study is the inclusion of birth fathers. Birth father data offers a special advantage when estimating genetic effects; unlike birth mothers, birth fathers do not provide the prenatal environment; thus, the genetic influences estimated from their phenotype are not confounded with prenatal influences. Although the birth father sample size is smaller than that of birth mothers (37% participation rate), the birth father data can be estimated with proper missing data treatment (Blozis et al., 2013). Other

strategies we have used for including birth father data are to combine with birth mother data when both are present or to run separate models that use birth mothers' characteristics as an index of genetic influences vs. birth fathers' characteristics for replication purposes. These strategies maximize our ability to estimate influences inherited from both parents.

Prenatal environment (highlighted in blue in Table 4).: Prenatal environment is assessed via birth mothers' reports of their pregnancy and from medical records. At the beginning of the project, the EGDS asked birth mothers to recall substance use (alcohol, cigarettes, illicit drugs) and depressive and anxiety symptoms during pregnancy. To assist women in recalling these behaviors, EGDS used a Pregnancy History Calendar that is modeled after the Life History Calendar (Caspi et al., 1996; Freedman, Thornton, Camburn, Alwin, & Young-DeMarco, 1988). In this task, birth mothers generated a list of life events (e.g., birthdays, holidays) around pregnancy and used the event calendar to retrieve the memory of what they did and how they felt. They also reported medical events that occurred during their pregnancy (e.g., infections, bleeding, pre-eclampsia). Medical records obtained from health care providers covered prenatal care and birth/delivery records. To combine the prenatal and perinatal information, we created a Perinatal Risk Index, a comprehensive coding system that captures the frequency of risk exposure for the fetus (Marceau et al., 2016).

Rearing environment (highlighted in orange in Table 4).: The EGDS measures the family and school contexts for child development. The most proximal to the child is the family environment in adoptive homes. The EGDS adoptive home assessment covers (1) at-home interpersonal relationships that directly involve the child, such as parenting and parent-child relationship quality; (2) characteristics of adoptive parents that are known to impact the child's development, most notably, interparental relationships, parental psychopathology, personality, health, and cognitive functioning; and (3) resources available in the adoptive home (e.g., home chaos, income, housing, health insurance). The EGDS has been assessing these three domains of within-home processes longitudinally since infancy. When children are old enough, school becomes an important component of their social milieu. The EGDS actively recruits teachers who offer valuable insights into the child's social relationships, academic performance, and emotional and behavioral well-being at school.

The EGDS rearing environment assessment incorporates multiple family members as sources of information to fully capture the complex, dynamic web of family interactions. One salient and valuable family member in the EGDS is rearing fathers. Although developmental scientists have been alerted by numerous calls to recruit fathers (Barker, Iles, & Ramchandani, 2017; Cabrera, Tamis-LeMonda, Bradley, Hofferth, & Lamb, 2000; Parke, 2000), most of our knowledge about family functioning and child development still relies on mothers' behaviors and reports. The EGDS helps fill this void by actively assessing fathers' roles in the family and how they uniquely contribute to child development. Therefore, the EGDS collects data from both adoptive mothers and fathers about their parenting, characteristics, and relationships with each other, as well as perceptions of their child's problematic and prosocial behavior.

On a related note, the EGDS conceives parenting as a multifaceted psychological construct that is characterized by both what parents do to socialize their child and as an index of parents' experience of their relationship with their child. Thus, EGDS has used both direct observation of parenting and parental reports, each of which measures related but unique facets of parenting. Like many other developmental studies, the EGDS' observation data on parenting are based on video-recorded brief and structured parent-child interactions that are subsequently coded by reliable raters using molecular and/or global ratings. In contrast, parents' self-report parenting scales ask parents to consider their parenting strategies and/or quality of their relationship with the child typically over an extended period, reflecting parents' perceptions of their own parenting, and more globally, their self-characterizations or self-cognitions in specific reference to being a parent. Thus, a parent who acknowledges the socially undesirable parenting behaviors is partially revealing facets of their negative self-view in relationship to his or her child. From this perspective, self-report parenting scales take their place alongside a range of other approaches to family measurement that focus on parental perceptions of their children and of their relationship to them (Fonagy et al., 2016; Sawrikar & Dadds, 2018; Snarr, Slep, & Grande, 2009; Teti & Gelfand, 1991). With some notable exceptions , the correlations between directly observed parenting behavior and parent-reported questionnaires have been low (for example, an in-depth evaluation in Arney, 2004, and a recent meta-analysis by Hendriks, Van der Giessen, Stams, & Overbeek, 2018), but both provide unique and valuable information on what goes inside of the relationships between parents and children. We will come back to this issue in Chapter VII where we synthesize our data.

Child characteristics (highlighted in green in Table 4).: The assessment of child characteristics covers a wide range of variables, from biological properties (e.g., HPA regulation, anthropometrics), cognitive functions (executive functioning, academic performance, language, and literacy), psychological characteristics (e.g., temperament), emotional and physical health, to social relationships (e.g., peer relationship quality, teacher-student relationship). Developmentally salient domains of child development are evaluated by repeated assessments, and EGDS adjusts protocols and measures to be developmentally sensitive to the growth of the children, retaining the flexibility to add new constructs that emerge with age and drop constructs that children outgrow.

Confounds.: As noted in Chapter I, the adoption design rests upon several assumptions which, if neglected, can threaten the validity of the separation of genetic and environmental estimates (see Table 2). One such confound is *openness in adoption*. Because the exchange between birth and adoptive families could muddy the separation of genetic and environmental influences, the EGDS assesses the degree of information shared and the amount of virtual and in-person contact engaged between birth parents and adoptive parents. We track this information longitudinally because the amount of the exchange between birth parents and adoptive homes could change over time. Our data show a wide range of openness from completely closed (no information exchanged between the adoptive parents or birth parents) to very open (visits at least once a month and communicates several times a month; Ge et al., 2008), and levels of openness appear to decline over time (Leve et al.,

2019). The degree of adoption openness is actively incorporated in most of the published papers to control for its effect statistically.

In addition, the EGDS also assessed adoptive and birth parents' experiences of the adoption process at the initial data collection. A survey on the adoption processes asked reasons for choosing adoption, social support received during the adoption process, and post-placement adjustment. Our work shows that adoptive parents have a wide range of reasons for pursuing adoption, but fertility problems to conceive a biological child emerged as a dominant reason, with pre-adoption fertility challenges often shaping how adoptive parents navigate parenthood (J. Wang et al., 2021).

Methodological Limitations

As with any study, the EGDS has methodological limitations that need to be considered upon interpreting its findings. The first, and perhaps most important, is the sample's representativeness. As noted earlier, the majority of the EGDS adoptive parents are financially resourced and highly educated. This pattern is partially a product of regulatory practices of adoption in the U.S. The financial screening of adoptive families, which is often used by adoption agencies, ensures that adoptive families have enough financial resources to adopt and raise a child. We, along with others (e.g., Horn, Loehlin, & Willerman, 1982; McGue et al., 2007; Stoolmiller, 1999), have documented the higher family SES of adoptive families (Leve et al., 2019; Natsuaki et al., 2019). Similarly, the eligibility criteria used in the adoption process to screen potential adoptive parents create a skewed distribution of risks in rearing environments. These screening processes often lead to the selection of prospective parents with lower levels of psychosocial risks (e.g., parental criminality, history of child abuse), restricting the range of quality in rearing environment. Because adoptive parents went through extra steps to become parents, they were generally motivated, responsive, and eager to parent, especially soon after the adoption was completed. In addition, our adoptive parents have started the rearing of the adoptees at relatively older ages (many in their late 30s), in many cases, after struggling with infertility (J. Wang et al., 2021). Moreover, nearly all the EGDS adoptive families were two-parent households at the inception of the project, with most being mother-father families, and most identified as non-Hispanic White. The limited socioeconomic and racial/ethnic diversity in the adoptive parent sample characteristics may affect the scope of generalizability of our findings. More recently, we have recruited children living with the EGDS birth mothers, which increases the socioeconomic and racial/ethnic diversity of the overall sample of children, although this subsample is not a focus of the current monograph (see Chapter VIII for future directions). Finally, readers are reminded that the EGDS adoptees did not have known major perinatal medical conditions, and both birth and adoptive parents were proficient in English.

Second, despite relentless effort from the recruitment team, the EGDS birth father sample is smaller than that of the birth mothers, with an enrollment rate of 37%. The substantial missing data on birth fathers means that we are missing critical information about potential genetic influences, although we did ask birth mothers to report on some characteristics of birth fathers.

Overall Summary of This Chapter

A prospective, long-term longitudinal adoption study is particularly challenging. However, when the study design and measurement are carefully fashioned, it provides a unique opportunity to disentangle genetic, prenatal, and post-adoption rearing environmental influences and examine how these factors synergistically influence child development. The EGDS represents one such effort. In Chapters III-VI, we present key findings to leverage the unique design attributes of the prospective, longitudinal parent-child adoption design.

Chapter III: The Effect of Parenting, Marital Process, and Parental Depressive Symptoms and Anxiety Symptoms on the Developing Child

Introduction

Major developmental theories of the 20th century accord parents a critical role in shaping children's emotional and behavioral development, including Attachment Theory, Psychodynamic Theories, and Social Learning Theory (P. H. Miller, 2016). These perspectives emphasize that parents stimulate growth and affect their children's thoughts, ideas, emotions, and behaviors. Processes such as conditioning, observational learning, behavioral and emotional scaffolding, internalization, and the provision of stimulation and learning opportunities are proposed as explanatory mechanisms that bridge the gap between parents' behaviors and child outcomes. Additional models propose that parents' behaviors and approaches to child rearing are influenced by parents' own personal characteristics such as mental health and personality and are embedded within a broader network of family relationships and culture (J. Belsky, 1984; Garcia Coll et al., 1996). These "distal" factors may also be socializing forces through their impact on parenting, or through their impact on families' emotional climates and provision of additional models of behavior.

In the last decades of the 20th century, however, the power of socialization effects on child development started to be questioned. As will be discussed extensively in Chapter IV, a new generation of research emphasized the role of children's temperament and heritable characteristics in shaping parenting and family processes (e.g., Kendler & Baker, 2007; Reiss, Hetherington, Neiderhiser, & Plomin, 2000). Responding to these findings, Collins et al. strongly argued that parents' behaviors have a direct causal influence on child development that is independent of child-based genetic effects (Collins, Maccoby, Steinberg, Hetherington, & Bornstein, 2000). Their conclusions were supported by longitudinal, predictive studies, randomized controlled trials demonstrating the effectiveness of parenting interventions in improving the trajectories of child development, and findings from non-human primate studies where the presence or absence of ordinary nurturing from parents can be experimentally manipulated for even more rigorous conclusions about parental influence. However, it is notable that their defense of parenting effects was based on models that did not account for children's genetically influenced characteristics, and thus, could not directly address the degree to which apparent parent-driven socialization effects might be attributed, in whole or in part, to genes shared by parents and children..

Subsequent research has continued to find evidence of parent effects, child effects on parenting, and reciprocal effects (e.g., Kiff, Lengua, & Zalewski, 2011; Lansford,

Rothenberg, et al., 2018; Pinquart, 2017; J. D. Smith et al., 2014). Yet, most of this research literature is still not designed to consider children's genetic contribution nor genes that are shared by children and their rearing parents. Although twin studies continue to find child-based genetic influences on parenting (Spotts & Ganiban, 2015), even within this literature, there is a relative dearth of longitudinal twin studies that examine if parenting accounts for changes in children's behaviors above and beyond child-based genetic influences (for exceptions see Larsson, Viding, Rijsdijk, & Plomin, 2008; Micalizzi, Wang, & Saudino, 2015; Neiderhiser, Reiss, Hetherington, & Plomin, 1999; Vertsberger, Saudino, Avinun, Abramson, & Knafo-Noam, 2019). In addition, even within the longitudinal twin studies that do exist, evocative child effects on parenting cannot be completely separated from the effects of genes shared by parents and their twin children. The EGDS adoption design provides an additional test of direct influences of parenting on child development. In this chapter, we focus on EGDS analyses that isolate the socializing influences of parenting on children's emotional and behavioral outcomes from children's genetically influenced characteristics. The analyses described in this chapter examine adoptive parent effects on children, alongside heritable transmission. Chapters IV, V, and VI will discuss more complex models of parent effects that incorporate bi-directional paths and interactions between parent and children's genetically influenced characteristics over time. Chapters VII and VIII will focus on the implications of our findings and the development of a prevention-oriented model of parenting.

Estimating Unconfounded Parenting Effects Via the Adoption Design

As described in Chapter I, one of the prime advantages of the adoption design is the capacity to disentangle genetic influences from socialization processes on child development (see Figure 4). In the adoption design, the biological parent does not provide their biological child's rearing environment, including the parenting they receive, the rearing parents' mental health, and interpersonal relationships. Therefore, within the adoption design associations between the rearing parents' behaviors or the family environment and child outcomes primarily reflect parental influence rather than effects of genes common to parent and child (path t) or direct heritable transmission (path g). This inference is further strengthened when associations between parenting and child outcomes persist when potentially confounding influences—especially genetic and prenatal—are ruled out in analytic models. Therefore, the adoption design can provide clarity regarding the impact of parenting and family-wide contextual factors on child outcomes.

Throughout this chapter we consider three lines of investigation in the EGDS that have explored rearing parent and family effects and their independence from genetic influences on child outcomes. These include studies that focus on associations between: (1) parenting and child psychosocial outcomes; (2) parental depressive and anxiety symptoms and child outcomes; and (3) interparental conflict and child outcomes.

1) Parenting and Children's Psychosocial Outcomes: We begin by summarizing two studies that found evidence of rearing parent effects on child outcomes, after controlling for genetic influences on children and effects on parenting. The first study focused on the development of child aggression during the toddler period (Stover et al., 2016). The

second study focused on the development of self-regulation from the toddler period to early childhood (Bridgett et al., 2018).

Stover et al. (2016): parenting effects and child aggression.: Parents' self-reported and observed parenting behaviors, especially harshness and negativity, are reliably associated with child aggression (Card, Stucky, Sawalani, & Little, 2008). However, relationships are usually modest in magnitude, especially when examined in non-clinical samples and using longitudinal, prospective designs (Pinquart, 2017). Explanations for these associations have relied on social learning processes such as conditioning and modeling aggressive behavior during parent-child interactions, as well as the development of hostile attributions and poor emotion regulation skills because of negative parent-child relationships (S. Lee, Chang, Ip, & Olson, 2019; J. D. Smith et al., 2014; Snyder, 2016). However, twin studies with children (Deater-Deckard, 2000) and adolescents (Narusyte et al., 2007) suggest that associations between parenting and child aggression are also influenced by genetic influences. These findings therefore challenge whether observed associations between parenting and child aggression solely represent parenting effects. Genetic processes may also underlie associations between parenting and child aggression.

To help clarify the role of parent effects in the development of aggression during childhood, we examined links between adoptive mother and adoptive father hostility and parent-reported changes in child aggression during the preschool period with Cohort I. Within these analyses, genetic risk for aggression, as assessed by children's birth mother's history of antisocial behavior, delinquency, and substance use, were not related to the adoptees' aggression at any age. However, we found a moderate prospective association between adoptive father's hostile parenting, measured by parent report at child age 27 months, and change in child aggression from 27 months to 4.5 years (beta = .47); there was also a trend in the same direction for mothers (beta = .25; See Figure 5). Surprisingly, in a second analysis that focused on change in child aggression from 4.5 years to 6 years, the prospective association from parenting at 4.5 years to child aggression at 6 years was non-significant.

The full models tested in this study also included family variables that often serve as an influential context of parenting: marital history and financial strain; these were assessed when children were 18-months old. Both constructs were positively, albeit modestly, associated with child aggression at 4.5 at the bivariate level. However, adoptive fathers' parenting hostility mediated these associations within the full structural equation model. Consequently, adoptive fathers' behaviors, but not mothers' behaviors, were influenced by the broader family context, rather than by their children's genetically influenced characteristics.

Collectively, the findings reported by Stover et al. (2016) provide evidence of direct adoptive father effects on the development of aggression during early childhood that are independent of the effects of the children's heritable risks. A similar effect was found for adoptive mothers, albeit weaker. These effects were not replicated when Stover et al. (2016) examined changes in aggression from 4.5 to 6 years. This pattern of findings suggests that there is early susceptibility to paternal influences that becomes attenuated as children grow older. It

is also possible that changes across this time period are driven by children's exposure to new behavior models as they transition to preschool and kindergarten.

Bridgett et al. (2018): parenting effects and effortful control.: The second example of parenting effects is provided by Bridgett et al. (2018). This study examined the impacts of parenting and heritable risks on the early development of effortful control. Effortful control encompasses top-down regulatory processes that enable one to voluntarily control behavior through purposefully inhibiting behavior and regulating attention in service of a goal. Poor effortful control is associated with a host of negative child outcomes, including internalizing and externalizing symptoms, peer relationships, and academic outcomes (e.g., Morris et al., 2013; Santens, Claes, Dierckx, & Dom, 2020; Y. J. Wang & Zhou, 2019). Theoretical models have long postulated direct parent effects on the development of self-regulation (e.g., Kopp, Hoffman, 2000; Vygotsky, 1978). Positive control strategies (e.g., the provision of appropriate structure and support) are thought to foster effortful control development in children, while negative control strategies (e.g., harshness, negativity) are thought to undermine its development. Meta-analyses support these hypotheses, albeit these associations tend to be modest in magnitude during infancy and toddlerhood (Karreman, van Tuijl, van Aken, & Deković, 2006) and the preschool period (Valcan, Davis, & Pino-Pasternak, 2018) At the same time, several studies indicate that genetic factors also contribute to children's effortful control skills (Bridgett, Burt, Edwards, & Deater-Deckard, 2015), again, opening the possibility that observed links between parenting and child effortful control may reflect underlying genetic processes rather than primary parent effects.

To explore these possibilities within the EGDS, Bridgett et al. (2018) used Cohort I and II of the EGDS sample (n = 561 bio-adoptive family units) to explore associations between negative parental control (i.e., harshness) and children's effortful control skills during early childhood. Bridgett et al. used observational measures of mothers' and fathers' harshness towards their children during structured interactions when the children were 27 months old. Children's genetic predispositions towards effortful control were assessed via their birth mothers' performance on an inhibitory control task (i.e., "Go-No Go task"). Children's effortful control skills were assessed through a composite measure that incorporated the adoptive parents' ratings of child effortful control at 54 months and the children's performance on a Go-No Go task at age 6 years.

As summarized in Figure 6, analyses indicated that adoptive mothers' and fathers' harsh parenting predicted children's effortful control: for both parents, greater harshness at 27 months was related to poorer subsequent child effortful control. Furthermore, these effects were independent of children's genetically influenced propensities towards effortful control: although birth mothers' effortful control was associated with the adoptees' effortful control, it was unrelated to the adoptive parents' behaviors. Altogether, these findings again support rearing parent effects on the development of effortful control that are separate from children's behavior and genetically influenced tendencies.

Summary and implications.

As noted at the beginning of this chapter, developmental science has a long history of emphasizing the role of parenting in child development. However, several studies over the past two decades have reconsidered the meaning of associations between parenting and child outcomes. Specifically, do these associations reflect socialization processes by which parents shape, guide, and/or support their children' emotional and behavioral development or do they reflect the impact of children on parents? Collins et al. (2000) provided a compelling argument that parents have a formative influence on their children that is independent of genetic transmission and other heritable processes. Consistent with this contention, this section summarized two studies that support the presence of rearing parent effects on children's early development of aggression and effortful control. Using the EGDS adoption design, we have been able to isolate these parental effects on children's behaviors without the confound of genes they share with their children. Furthermore, these EGDS analyses also emphasize that in two-parent families, both parents play important roles in child development, especially during periods of children's sensitivity to parental influences.. In addition, these parenting effects are present in models that include potential child effects related to their genetically influenced predispositions the effects of prenatal environment, and early manifestations of temperament. Finally, we demonstrated that these parental effects can mediate aspects of the wider social context, particularly marital and financial distress. For many readers, it may seem unremarkable that we found effects of parenting on child development. Nevertheless, it is important to confirm that socialization processes persist once genetically influenced processes are accounted for. Independent estimates of environmental effects of parenting need to be uncovered to select some targets for preventive interventions, specifically qualities of parenting. Later chapters will illustrate how the adoption design may also select genetically influenced attributes of the child as targets of psychosocial preventive efforts.

2) Adoptive Parent Depressive and Anxiety Symptoms on Child Outcomes: More Evidence of Parent Effects?—Another line of research addressed by the EGDS focuses on the impact of adoptive parents' mental health on their children's development. Previous research has linked maternal depressive and anxiety symptoms with emotional and behavioral problems in their offspring (Jami et al., 2021; Natsuaki et al., 2014). In Goodman et al.'s comprehensive meta-analysis (2011)higher levels of maternal depressive symptoms were associated with higher levels of child externalizing (mean $r = .21$) and internalizing (mean $r = .23$) symptoms. However, it remains unclear whether these associations denote parent effects or provide evidence of a broad heritable liability for psychopathology.

In a later paper, Gotlib and colleagues acknowledged that associations between parental depressive symptoms and child outcomes could reflect the genetic transmission of risk across generations (2020). However, they also outlined compelling non-genetic pathways through which maternal depressive symptoms could foster poorer developmental outcomes. For example, depressive symptoms could adversely affect parenting, rendering some parents nonsupportive (by omission or commission) or unresponsive to their children's developmental needs. A parent with depressive symptoms could also model maladaptive emotional and behavioral regulation, thereby increasing their children's risk for maladaptive

outcomes. Lastly, they also proposed that child characteristics (e.g., temperament) and paternal depressive symptoms could mitigate or amplify associations between maternal depressive symptoms and child outcomes. Therefore, this framework emphasizes that rearing parents can shape children's outcomes, but genetic risks and children's own characteristics can also redirect development for better or worse. Again, the adoption design is uniquely suited to tease apart rearing parent effects from genetically influenced processes, and to explore the more complex paths that Gotlib et al. anticipated.

Several analyses from the EGDS have examined associations between adoptive parents' depressive symptoms, anxiety symptoms, and/or internalizing symptoms and adoptees' behavioral outcomes. In each of these studies, analytic models included adoptive parents' internalizing symptoms and plausible indices in the birth parents of genetically influenced predispositions (i.e., birth parents' history of psychopathology or negative affectivity). While some studies examined if adoptive parents' depressive or anxiety symptoms predicted growth in child symptoms over time (A. P. Field et al., 2020; Kerr et al., 2013), others focused on cross-sectional associations between adoptive parents' symptoms and child outcomes (Brooker et al., 2011; Committee on Environmental Health, Committee on Substance Abuse, Committee on Adolescence, & Committee on Native American Child Health, 2009; Grabow et al., 2017; Leve et al., 2009; Leve, Kerr, et al., 2010), or longitudinal associations using path analysis (Ahmadzadeh et al., 2019; Brooker et al., 2014; McAdams et al., 2015; Pemberton et al., 2010; Sellers et al., 2020) and a random intercept model (Cioffi, Griffin, et al., 2021). Most EGDS studies have relied upon parent reports of affective symptoms and also, with the primary caregivers of children reporting on their internalizing symptoms and secondary caregivers reporting on child outcomes. A smaller set of EGDS studies have used observational measures of child outcomes (e.g., Bray et al., 2020; Brooker et al., 2011; Leve et al., 2009; Natsuaki et al., 2013; Roben et al., 2015). Despite these methodological differences, the results across analyses converge upon the same findings. Independent of genetically influenced child effects or the effects genes common to parent and child, adoptive mothers' depressive and anxiety symptoms are associated with more adverse child outcomes. To illustrate these conclusions, the next section will summarize findings from EGDS, with a special focus on two studies that examined associations between adoptive parent depressive symptoms during infancy (Pemberton et al., 2010) and from the toddler to middle childhood periods (Cioffi, Griffin, et al., 2021).

Pemberton et al. (2010): adoptive parent depressive symptoms during infancy.: In an early EGDS study that only included Cohort I, Pemberton et al. focused on the infancy period, assessing adoptive mothers' and fathers' depressive symptoms from 9 to 27 months and their children's externalizing behavior at 27 months. As shown in Figure 7, the full model tested also included an index of adverse prenatal exposures (i.e., perinatal risk), infants' genetic risk for depression (i.e., birth mothers' depressive symptoms), and infant fussiness (an index of temperament) at 9 months. Adoptive mothers' depressive symptoms were moderately stable over time and could be modeled as a single latent factor; however, adoptive fathers' depressive symptoms were time dependent. In the final model, adoptive mothers' and fathers' symptoms were related to their children's externalizing behavior at

27 months (Pemberton et al., 2010). Notably, these associations were independent from children's temperament, prenatal risk, and those genetically factors indexed by birth parent measures.

Additional analyses conducted during infancy have included indices of children's negative emotionality as outcome variables, rather than using behavioral symptoms. In one study, adoptive mothers' depressive symptoms at 9 months were associated with children's fussiness at 18 months (Natsuaki et al., 2010), and adoptive parents' anxiety symptoms at 9 months were associated with their children's negative affectivity at 18 months (Brooker et al., 2015). Considered together, these three studies illustrate rearing parent effects on child outcomes.

Cioffi et al. (2021): adoptive parent depressive symptoms during childhood.: Within EGDS, adoptive parents' depressive and anxiety symptoms are also related to their children's behaviors during early to middle childhood. For example, Perez Grabow (2017) noted significant associations between adoptive mothers' depressive symptoms and children's internalizing symptoms at age 7 years. A more comprehensive analysis led by Cioffi used a random intercept cross-lagged panel model to examine associations between adoptive parents' depressive symptoms and children's internalizing symptoms from 18 months to 6 years (2021). The random intercept model can be used to estimate the average association between adoptive mother's depressive symptoms and children's internalizing symptoms across families and over time (i.e., between-family effects). It can also capture within-family effects, including the degree to which changes in a child's internalizing symptoms are predicted by a rearing parent's symptoms at previous time points and the degree to which changes in a rearing parent's symptoms are predicted by children's own symptoms at previous time points. Separate analyses were conducted for adoptive mothers and fathers. The full analytic model also included children's broad genetically influenced risk for psychopathology (i.e., birth mother and birth father p factor), prenatal risk (prenatal depressive symptoms) and a broad index of children's psychiatric symptoms at age 6 years. Last, analyses controlled for children's perinatal risk, child sex, and adoption openness. Results for the adoptive mothers are included Figure 8.

At the between-families level, adoptive mothers' and fathers' depressive symptoms were moderately correlated with children's internalizing symptoms: β's ranged from .39 (adoptive mothers) to .43 (adoptive fathers). In both models, these associations were independent of genetic risks as indexed by birth parent measures, and prenatal risks, and child temperament. Further, at the within-family level, adoptive mothers' depressive symptoms at 18 months predicted changes in child externalizing behavior between 18 and 27 months. This latter finding is consistent with a causal association between maternal depressive symptoms and child symptoms. Last, there were within-age associations between adoptive mother depressive symptoms and child internalizing behavior at 27 months and 6 years, but the underlying directionality of these associations cannot be determined. Significant cross-lagged paths or within-age associations, however, were not observed within the adoptive father model.

The results of Cioffi et al. (2021) dovetail with the results of an additional EGDS analysis that explored predictors of changes in children's internalizing and externalizing symptoms from 18–54 months within a latent growth curve model (Kerr et al., 2013). This study found evidence of modest direct associations between adoptive mothers' depressive symptoms and children's levels of internalizing and externalizing symptoms at 18 months, after heritable influences (as indexed by birth mother depressive symptoms) and prenatal risks were statistically controlled. Neither adoptive mothers' depressive symptoms nor heritable risk were related to *changes* in children's internalizing or externalizing symptoms over time. Because Kerr et al. did not include adoptive fathers in the analyses, the effects of fathers are unknown. Therefore, similar to Cioffi's findings, adoptive mothers' influences on child symptoms arise early in development and have persistent effects on children's level of symptoms over time but may not account for the rate of change in symptoms over time.

Two additional EGDS analyses have examined links between adoptive parents' anxiety symptoms and their children's internalizing or externalizing symptoms during early and middle childhood. Notably, these studies indicate that adoptive fathers play a role in the development of children's anxiety symptoms. A growth model analysis that spanned the preschool period (18 – to 54-months) found that both adoptive mother's and father's anxiety symptoms comparably and uniquely predicted differences in children's anxiety symptoms at 18 months, but not on the rate of change in anxiety symptoms over time (A. P. Field et al., 2020). These associations were independent of children's genetic risk for anxiety symptoms. In addition, Ahmadzadeh et al. (2019) found that adoptive fathers' anxiety symptoms at child age 6 years predicted children's anxiety symptoms at age 8. Given the lack of earlier assessments in this analysis, it is possible that these effects could reflect the impact of earlier stages or that fathers' anxiety symptoms predict changes in child anxiety symptoms during middle childhood. Thus, akin to findings for parental depressive symptoms, adoptive parents' own mental health appears to influence their children's anxiety symptoms.

Summary and implications.

Collectively, these papers provide ample evidence of parent effects via parental depressive and anxiety symptoms. Adoptive parents' symptoms are consistently associated with their children's outcomes, and these effects operate alongside genetic pathways guided by children's heritable characteristics. However, consistent with Goodman's and Gotlib's (1999) framework, additional analyses from EGDS suggest that the pathways from heritable and rearing parent risks are more complex than simple additive models. These studies also indicate that the pathways from adoptive parents' depressive or anxiety symptoms to child outcomes reflect a myriad of intermediary processes, including parenting behaviors as mediators, bi-directional parent and child effects (Ahmadzadeh et al., 2019; Brooker et al., 2014; McAdams et al., 2015), and interactions between adoptive parent behaviors and children's heritable risks (Bray et al., 2020; Brooker et al., 2014; Cree et al., 2020; Leve et al., 2009; Roben et al., 2015). Therefore, although we have isolated and highlighted adoptive parent effects on child outcomes in this chapter, it is important to note that these effects often occur alongside more complex processes in which children's genetic influences affect and interact with parents' mental health. Child effects and interactions between parent and child characteristics will be expanded upon in Chapters IV and V, respectively.

3) The Impact of Interparental Conflict on Child Outcomes—Decades of research have established links between interparental conflict and child adjustment (e.g., Davies & Cummings, 1994; Harold & Sellers, 2018). The mechanisms proposed to underlie these observed associations include: (1) exposure to interparental conflict impinges upon parenting quality, which then contributes to adverse child outcomes (i.e., spillover effects; (Erel & Burman, 1995; Krishnakumar & Buehler, 2000); and (2) exposure to interparental conflict affects intermediary emotional, cognitive processes, and physiological reactivity that increase children's vulnerability to internalizing and externalizing behavior problems (e.g., Davies, Martin, & Cicchetti, 2012; El-Sheikh et al., 2009; Grych, Harold, & Miles, 2003).

Like parenting, however, there is convincing evidence that genetic factors influence marital quality (Spotts & Ganiban, 2015), and parents' genes partially explain covariance between interparental conflict and parenting (Ganiban, Ulbricht, et al., 2009). Regarding child outcomes, previous studies examining the cascading pathways from marital relationships to child development have shown that the same genes that influence (marital difficulties may, if passed on to children, influence children's developmental outcomes. For example, using an adoption study, O'Connor et al. found that links between divorce and children's impaired academic achievement and social competence were due to shared genes between the divorced partners and their children (O'Connor, Caspi, DeFries, & Plomin, 2000). Other studies also suggest the role of genetic factors that are shared by parents and children in accounting for links between parents' marital quality and child externalizing problems (Harden et al., 2007; Schermerhorn et al., 2011). Therefore, genes that run in the family appear to play a key role in understanding associations among measures of parents' marital relationships, of parent-child relationships and of child development. Less clear is how to reconcile these findings with the extensive research literature that conceptualizes marital conflict as an environmental exposure that affects children's emotional and behavioral development (Harold & Sellers, 2018). Capitalizing on its study design features, the EGDS can examine the extent to which the effects of genetic predispositions and rearing environments on child outcomes are intertwined or independent.

To date, several EGDS analyses have examined the degree to which parenting mediates associations between marital problems and child outcomes, known as spillover effects. Our earliest observation of the spillover effect comes from a study of 9-month-old infants (Rhoades et al., 2011). Adoptive parents' marital hostility at age 9 months led to overreactive parenting at age 18 months, which was positively associated with child anger and frustration at age 18 months. This spillover from marital quality and parenting continues to be observed well into toddlerhood (Stover et al., 2012) and the beginning of elementary school (Harold et al., 2013). These results suggest, spillover effects do not depend on genes shared by parents in distressed marriages and the children they rear. Below, we summarize three of these papers that support these conclusions: Harold et al. (2013), Stover et al. (2012), and Ramos et al. (2022).

Harold et al. (2013): do genes that are shared by parents and children explain associations between marital hostility and child outcomes?: Noting that there are genetic influences on parenting and child outcomes, Harold et al. (2013) examined if associations amongst interparental conflict, parent hostility, and child externalizing behavior could be

explained by genes that are shared by parents and their biological children . This analysis combined data from genetically *un*related families within the EGDS sample and genetically related families within the Cardiff IVF study. Including both samples allowed us to examine whether marital hostility to parenting spillover effects on child outcomes depends on the genetic relatedness of parents and their children. If spillover effects were only apparent for genetically related parents and children, it could be inferred that spillover effects are due to genes shared by marital partners and their children. However, if spillover effects did not rely on genetic relatedness, then the underlying process would be more consistent with parent to child socialization effects. Both studies assessed families at similar ages (age 6 years for EGDS, between 5–8 years for Cardiff IVF) and used the same measures to assess interparental conflict and child externalizing behavior. Slightly different measures were used to assess parent-child hostility. Results were consistent with socialization processes, as paths between marital hostility and parenting, and between parenting and child outcomes were similar in magnitude for genetically related and unrelated parent-child dyads.

Stover et al. (2012): interparental conflict and toddler aggression.: An EGDS analysis led by Stover et al. (2012) examined associations between marital conflict, adoptive mothers' hostile parenting, and children's aggression at age 27 months. Composite scores based on the adoptive mothers' and fathers' ratings were created for each study variable. In addition, the analytic model included an index of children's putative genetic predisposition for aggression (i.e., birth mother antisocial traits), adoptive parents' antisocial tendencies and financial stress, and child aggression at 18 months. As in all our studies, analyses also controlled for children's prenatal risk and adoption openness.

Consistent with spillover models of interparental conflict, marital hostility was indirectly associated with toddler aggression via hostile parenting (see Figure 9). As the adoption design rules out passive gene-environment correlation, these analyses again indicate spillover effects as an environmental pathway through which marital hostility affects parenting and child aggression. In addition, these effects were partially driven by the adoptive parents' own antisocial behavior and by child aggression at 18 months, but independent of children's genetic risk (as indexed by birth parent measures) and perinatal risk. Therefore, variance in parent hostility is multiply determined, indicating that there may be several intervention targets to disrupt both marital hostility and hostile parenting.

As described earlier in this chapter, Stover's 2012 analyses were expanded in a second paper that assessed the contributions of marital hostility to changes in child aggression from 27 months to 4.5 years, and from 4.5 years to 6 years (see Figure 4, Stover et al., 2016). Marital hostility was significantly associated with adoptive father hostility, but not adoptive mother hostility. Furthermore, adoptive father hostility mediated associations between marital hostility and changes in child aggression from 27 months to 4.5 years, consistent with the spillover models. As described previously, this pattern of associations was independent of children's genetic risk for antisocial behavior. Surprisingly, this effect was not replicated for changes between 4.5 to 6 years. Therefore, although spillover effects were detected, their relevance for child outcomes may change over time, with toddlerhood being a particularly sensitive period. As children become better regulated during the preschool and early school-age period (Shaw & Bell, 1993), showing lower levels and

less variability in externalizing symptoms than during the toddler period, this age-specific finding is not terribly surprising.

Ramos et al.(2022): associations between interparental conflict and warmth and observed parenting behaviors during early childhood.: The previous papers relied upon parents' self-reported parenting behaviors. A third EGDS paper recently examined spillover processes within Cohort I using observational measures of parental coercion and positive engagement during a puzzle task when children were 6 years of age. In addition, while previous analyses focused on marital conflict in spillover processes, Ramos et al. also considered marital warmth. These analyses also included birth mothers' negative affectivity and child temperament (anger, expressing pleasure). These latter constructs were included to examine if children's genetically influenced tendencies and behaviors, as indexed by birth parent scores, were related to marital conflict and warmth as well as parents' observed behaviors. Last, the analytic model was longitudinal in design, with child anger assessed by parents at age 18 months, latent marital conflict and warmth factors generated by mothers' and fathers' reports at 27 months, and parent-child interactions observed at age 6 years. Marital factors, birth mother negative affect, child temperament, and parenting constructs were examined in a single model, with separate analyses conducted for marital warmth and conflict. (Note: the published paper referred to marital processes as "interparental", but here we use the term "marital" for consistency).

Path analyses suggested that over time marital warmth was inversely associated with adoptive mothers' coerciveness with their children. However, the patterns of associations for fathers differed from previous EGDS studies. Specifically, fathers' positive engagement with their 6-year-old children was positively associated with marital conflict and inversely related to marital warmth. These associations were independent of children's genetically influenced tendencies and temperament also ruled out were the effects of genes shared by parents and children.

Discrepancies between Ramos et al. and the previously described EGDS papers (Harold et al., 2013; Stover et al., 2012) may be based on methodological differences. First, Harold et al. and Stover et al. used contemporaneous assessments of marital conflict and parenting behavior, while in Ramos et al., the assessment of marital constructs and parenting were separated by four years. Therefore, if spillover processes rely upon the literal transfer of emotions and behaviors from one family relationship to another (Cox & Paley, 1997) they may be best captured when the marital and parent-child relationships are assessed close in time. Ramos et al. findings, however, may reflect a different process altogether – compensatory effects. Over time fathers may have adapted to negative features of the marital relationship (i.e., more marital conflict and less marital warmth) by investing more in their relationship with their children.

Summary and Implications

Collectively, analyses conducted with the EGDS have replicated spillover effects from marital relationship difficulties to parenting, and downstream effects on child outcomes (Harold et al., 2013; Rhoades et al., 2011, 2012; Stover et al., 2012), also, finding evidence

of compensatory effects (Ramos et al., 2022). These analyses effectively rule out genes shared by parents and children as the underlying process that accounts for spillover or compensatory effects on the parent-child relationship.

Nevertheless, additional studies from the EGDS also suggest that children may play an active role in associations between marital conflict, parenting, and their own outcomes. For example, child effects were present in Stover's analyses (2012; 2016) and in Ramos et al. (2022) An additional EGDS study conducted by Rhoades et al. (2011) found that marital hostility between adoptive parents was associated with increases in child anger, but only when the child's birth mother scored high on measures of her frustration and anger. It may be that children who have inherited emotional lability and low frustration tolerance from their birth mothers are more sensitive to the ill effects of marital hostility in their rearing parents. Furthermore, Fearon et al. (2015) found that adoptive parents' marital problems in infancy/toddlerhood (9 – 27 months) dampen adoptive mothers' feelings and confidence about their parenting ability, but this association was only evident for families of children whose birth mothers had a history of psychiatric symptoms (Fearon et al., 2015). While it remains speculative, this result may suggest that marital tension makes it difficult for adults to parent any child, but it might be increasingly challenging when children exhibit challenging behaviors. Therefore, the pathways from marital conflict to child aggression may not always be straightforward.

Overall Summary of This Chapter

At the beginning of this chapter, we described challenges to socialization theories of development by a growing body of research indicative of child-based genetic effects on parenting, and genetic transmission of behavioral and psychological tendencies from parents to their biological children. As noted earlier in this chapter, Collins et al. (2000) offered a strong defense of parent socialization effects. Our analyses within the EGDS have enabled us to assess socialization processes in tandem with potential heritable processes in early development. Collectively, our analyses support the notion that both mothers and fathers uniquely influence their children's development, supporting socialization theories. Isolating parenting effects on children from child effects and genetic transmission processes has considerable practical importance. When parenting effects are identified, then interventions may focus on the parent rather than the parent-child relationship. For example, there is some evidence that parental depressive symptoms are related to adverse child outcomes via their influence on parenting. If this parenting effect is shown to be independent of heritable influences and child effects, then interventions focused on parent depressive symptoms alone could have powerful long-term influences on child outcomes. Consistent with this view, a series of studies already suggest that pharmacological or psychotherapeutic treatment of maternal depressive symptoms may not only improve the mothers' depressive symptoms but also the quality of her parenting and her children's development (Cuijpers, Weitz, Karyotaki, Garber, & Andersson, 2014; Stein et al., 2018; Swartz et al., 2008; Weissman et al., 2015). It is important to note, however, that parenting interventions may not always disrupt associations between parental depressive symptoms and child outcomes because depressed parents also influence children's rearing environments through their *inactivity* in

proactively providing economic, educational, and psychosocial resources for their children (Shaw & Shelleby, 2014).

Although our analyses support parent effects on child outcome, EGDS analyses also indicate that developmental pathways are complicated. Parenting behavior and parents' mental health can be influenced by their children's behaviors, and children's heritable predispositions interact with what parents do to further shape development. These processes and implications will be discussed in greater depth in Chapter IV (child effect on development) and Chapters V and VI (gene by environment interactions and development).

Another important consideration is the role that environmental context may play in the magnitude of direct parent effects on child outcomes. As described in Chapter II, the EGDS' adoptive families are educationally and economically advantaged. The magnitude of direct parenting effects could be weaker, stronger or qualitatively different in other environmental contexts. For example, economic adversity could amplify the negative effects of maternal depressive symptoms or child outcomes (Shaw & Shelleby, 2014), or different parenting practices may promote positive youth outcomes across different racial and ethnic groups (e.g., Garcia Coll et al., 1996). Neighborhood variables could also alter the importance of parenting for child outcomes (e.g., Eisman, Stoddard, Heinze, Caldwell, & Zimmerman, 2015; Schofield et al., 2012). In addition, cultural norms can influence the magnitude of associations between parent and child outcomes (e.g., Lansford, Godwin, et al., 2018). Therefore, although we have used the adoption design to identify direct parent effects on child outcomes, parent-child relationships are embedded within a broader social ecology that can influence these effects.

Chapter IV: Child Effects on Parent Characteristics and Parenting

Introduction

Richard Q. Bell published one of the most foundational papers in 20th century psychology.. He integrated evidence from a broad range of studies to challenge a perspective that had prevailed for centuries: parents have profound and direct effects on the psychological development of their children. He presented evidence that, in many cases, it might be the other way around: characteristics of the child could influence their parents (Bell, 1968). Among other studies, he pointed to data from young twins suggesting the heritability of goal directedness or sociability, qualities of children that might influence how their parents treated them. Nine years later, in a highly technical paper, these child effects on parents were named "reactive gene-environment correlation" (Plomin, DeFries, & Loehlin, 1977). That is, differences among children, attributable to their genotypes, could reliably elicit reactions from parents and anyone with whom they interacted. This then results in, or explains, correlations between a child's genotype and the environments in which they develop. Over time this phenomenon came to be called, in genetic literature, *evocative gene-environment correlation* and was expanded into a developmental model (Scarr & McCartney, 1983) suggesting that these evocative effects are likely to persist throughout the lifespan (Scarr, 1992). As noted, in this monograph we refer to these correlations more simply as "child effects." A half century after Bell's paper, this phenomenon remains understudied, despite

several decades of research substantiating the importance of these effects, especially for parenting (e.g., Broderick & Neiderhiser, 2019; Horwitz & Neiderhiser, 2015).

Evidence for the role of children in shaping their environments comes from twin studies. Since the earliest paper by Rowe (1981), studies of twin children have reported child genetic effects on parenting behaviors from infancy to early adulthood (see Broderick & Neiderhiser, 2019 for a review and Klahr Burt, 2014 for meta-analysis). There have also been twin studies that focused on clarifying which genetically influenced characteristics of the child were evoking the parenting they receive (e.g., Narusyte et al., 2007). Overall, these studies have confirmed that child genetic effects on parenting behavior are widespread, with some variation based on reporter and construct.

Our adoption design permits two approaches to identifying evocative child effects. First, it estimates child effects free of the assumptions of the twin design. Twin designs estimate the effect of the entire child's genome as expressed at the age of assessment. A limitation of twin designs for detecting evocative effects is that, in most samples, parents know whether twins are identical or fraternal. If this leads to their initiating more similar treatment of identical twins, evocative effects may be overestimated because the parents will be treating the identical twins more similarly—not because of genetic influences of the twins on parenting—but rather because of parents believe that identical twins should be treated more similarly than fraternal twins. Adoption designs use a weaker tool for genetic estimates: measurement of birth parents (see Chapter II for more details). All estimates of genetic effects in adoption designs are attenuated by age differences between birth parents and children, especially where genes influencing parental behavior may not be the same as those influencing comparable child behavior. Further, because of missing data in the EGDS we must impute data for a majority of birth fathers (Blozis et al., 2013; Marceau et al., 2019; Shewark et al., 2021). In addition, adoption designs cannot estimate—in contrast to twin studies—the effects of interactions among genes.

Despite its limitations, a second advantage of the adoption designs is that it allows us to understand the role of evocative child effects on social processes within the family as a consequence of the transmission of specific characteristics from parent to child. Unlike twin designs, the adoption design affords an "instantaneous longitudinal design," permitting us to identify the earliest manifestations of genetic liability for potential adult psychopathology and genetic assets for favorable adult outcomes. Specifically, we can examine a pathway to child adjustment that helps to clarify the role of the child and the role of the family: birth parent characteristic -> genetic transmission -> child characteristic -> adoptive parent caregiving -> child development.

The prenatal environment is also a potential influence on the child that subsequently influences adoptive parent caregiving and child adjustment. In fact, at least two studies from the EGDS have found that the prenatal environment may be a pathway through which birth parent risks influence the child (e.g., Marceau et al., 2016; Pemberton et al., 2010). Note that although the EGDS design is ideal for distinguishing genetic and prenatal influences from those of the rearing environment, the same is not true for distinguishing genetic from prenatal environmental influences. Specifically, because the birth mother provides the child

with both half their genes and their prenatal environment, definitively distinguishing the two with only birth mother data is not possible. In the EGDS, we also include a subsample of birth fathers that are included in the model alongside indicators of the prenatal environment and birth mother characteristics. Where the association between birth father measures and child measures equals that of the same association for birth mothers we can infer little or no prenatal environmental effects (e.g., Loehlin, 2016; Rice et al., 2018). We can thus more fully examine pathways to child development to include prenatal environment: birth mother and birth father characteristics -> genetic transmission -> prenatal environment -> child characteristic -> adoptive parent caregiving -> child development. In this chapter, we highlight EGDS analyses that examine: (1) evocative effects on parenting via heritable characteristics; (2) Evocative effects on parenting via specific child characteristics.

Evocative Effects on Parenting via Child Heritable Characteristics

Hajal et al. (2015): do child effects evoke a positive response from parents?— The research reviewed previously focuses on children evoking a negative response from their parents. It is also, however, possible that children may evoke a positive response from their parents as has been shown in twin research with adolescents (e.g., Neiderhiser, Reiss, Lichtenstein, Spotts, & Ganiban, 2007). In a first step in examining how children may evoke positive responses from their parents, we considered a characteristic of children that might evoke a positive response from their parents (for a full account of this study see Hajal et al., 2015). We examined the relationship between a temperament characteristic (Cloninger, Svrakic, & Przybeck, 1993) of birth and adoptive parents–reward dependence—and adoptive parents' reports of their overreactive parenting (Arnold, 1993) when the child was 9 months old. Reward dependence reflects an individual's dependence on social reinforcement and may manifest as sociable behavior. We also assessed the quality of rearing parents' marriage based on coding of videotaped interactions during a standard challenge task (Melby, Conger, Ge, & Warner, 1995).

We found a significant *inverse* relationship between birth mother reward dependence and adoptive fathers' overreactive parenting as shown in Figure 10. To be fully appreciated, this association must be examined in the context of the other relationships shown in Figure 10. First, this association was roughly the same magnitude as the inverse relationships between marital relationship quality and father's overreactive parenting. It is also notable that reward dependence of both rearing parents was unrelated to their overreactive parenting, further underscoring the unique evocative child effect illustrated here.

Taken together, these findings suggest that there are child effects that evoke a response from their father, via characteristics that were inherited from their birth parents related to reward dependence. These child effects reduce the likelihood that adoptive fathers will overreact negatively to challenging situations with their infant. The relationships depicted within the adoptive family are cross-sectional but suggest that the child effect operates in tandem with the protective effect of a high-quality marriage. We are unaware of previous studies of these simultaneous buffering systems in families of infants. It is yet another example, if we consider families where parents rear their biological offspring, of parents transmitting to their children qualities that may positively affect their own behavior. In this

case, the children's characteristics break an adverse cascade of negativity in the marital and father-child relationships for which we have suggestive evidence of a parental *self-correcting feedback spiral.*

Stover et al. (2012; 2016), Klahr et al. (2017): Do child effects evoke or mute adverse parenting?—In a second example of our research delineating the influence of child effects on parent behavior, we turned to an index of an adverse outcome: history of antisocial behavior in birth parents as measured by frequency of delinquent behavior using the Elliot Self Report of Delinquency Questionnaire (Elliott, Huizinga, & Ageton, 1985) and a modified version of a clinical interview, the Diagnostic Interview Schedule (Kessler, Andrews, Mroczek, Ustun, & Wittchen, 1998) that measures symptom counts for conduct disorder and antisocial personality disorder. Antisocial behavior in the rearing parents was measured using the Antisocial Action questionnaire (Levenson, Kiehl, & Fitzpatrick, 1995).

In three separate analyses (Klahr et al., 2017; Stover et al., 2012; 2016) we found little effect of birth parent antisocial behavior on adoptive parent overreactive parenting. However, at child ages 18 and 27 months, we found significant associations between the adoptive parents' own antisocial behavior and their overreactive parenting (see Figure 5; Stover et al., 2012). Further, at 18 and 27 months and 4.5 years, we found no effect of birth parent antisocial behavior on child aggression, the presumed mediator in those studies between birth parent antisocial behavior and negative parenting. Interestingly, toddler aggressive temperament was significantly correlated with adoptive mother and father overreactive parenting measured at the same time – at 18 months, 27 months, and 4.5 years (Stover et al., 2012; 2016). These findings suggest that there is a child effect on overreactive parenting for adoptive mothers and fathers, but this child effect is not due to genetic influences from the child as measured by birth parent antisocial behavior.

Evocative Effects on Parenting via Specified Child Characteristics.

Child effects on parenting, especially those at least partially explained by genetic influences, are the result of parents' responding to a child's behavior or temperamental characteristic. Accordingly, one important step in understanding how children influence the way they are parented is to identify child characteristics that evoke parental responses. Studies like the EGDS, which includes extensive assessment of a wide array of child characteristics and behaviors, are well suited to identifying such child characteristics because of the clear distinction between the birth parents, who provide only genes and prenatal environment, and the adoptive parents who provide the rearing environment. The next set of papers describes the progress that the EGDS has made in identifying child characteristics that evoke specific types of caregiving behavior.

Elam et al. (2014): child social motivation effects on parenting.—An opportunity to carry out a more complete analysis is provided by extensive literature on a comparable temperamental characteristic in parents and children: low social motivation. In contrast to social anxiety symptoms or social inhibition, this characteristic refers to a persistent pattern of low propensity to engage in social relationships and preference for solitary activities. In adults, this trait can be measured with the framework of Reinforcement Sensitivity Theory,

first developed by Gray (e.g. Corr, Pickering, & Gray, 1997). Low behavioral activation, as measured on a self-report questionnaire (Carver & White, 1994) has been associated with disinterest in social relationships and inattentive social behavior in adults (e.g. Hundt, Kimbrel, Mitchell, & Nelson-Gray, 2008; Kimbrel, Mitchell, & Nelson-Gray, 2010). An identical pattern of behavior can be assessed through rearing parent reports of behavior in toddlers, particularly their disinterest in engaging socially with parents and preference for solitary play (Olson, Bates, & Bayles, 1982) that anticipates disruptive peer relationships years later (Olson, Bates, Sandy, & Lanthier, 2000). The mechanism for this association is unknown, but EGDS was designed to examine the role of genetically influenced child effects among the causal mechanisms. Although both scales measuring adult behavioral approach and low social motivation in children are, not surprisingly, heritable (Silberg et al., 2005; Takahashi et al., 2007), it is unknown whether there is any overlap in the genetic influences on adults and children, nor even whether there are notable within family correlations between parents and children. Thus, the choice of low social motivation in the parent rests on modest grounds: phenotypic similarity between parent and child manifestations and correlates with social behavior in both generations. Further, we reasoned that a child who is inattentive or unresponsive to their parents might provoke their hostility.

The EGDS is the first study to report a correlation between low social motivation in parent and child; in our case it was between the *birth* mother and child at 27 months suggesting that at least part of the transmission route is genetic (Elam et al., 2014). Birth fathers were not included in these analyses, so the magnitude of genetic factors is underestimated. In a SEM model, Elam and colleagues observed that *birth* mothers' low social motivation was associated with self-reported *adoptive* father and mother hostility towards the child at 27 months, as measured by parent self-reports (Melby & Conger, 2001). These two associations are evidence of an evocative child effect and were mediated by low social motivation in the child at 27 months, which was related to disruptive peer behavior two years later (Elam et al., 2014). Low social motivation in the child at age 27 months mediated the association between birth mother social motivation and both adoptive parents' hostility, as illustrated in Figure 11.

Sellers et al. (2020): Evocative child effects on parenting predicting child ADHD symptoms and aggression.—Might we see a similar pattern of findings in the development of a highly heritable child symptoms? That is, might evocative child effects on parenting play an unheralded role in the genetic transmission from parents to children of symptoms of psychopathology? An inviting target is the set of attentional and hyperactive symptoms often associated with syndromal ADHD, a highly heritable disorder (Faraone & Larsson, 2018) that is commonly diagnosed in children but, in many cases, is also observed in adults. Indeed, when our analyses were planned a decade ago there was ample justification for picking ADHD symptoms in adults to explore not only evocative child effects on the family system, but the role of these effects in parent-to-child transmission of behaviors associated with ADHD, such as inattention and hyperactivity.

In seeking the possible role of evocative child effects in the transmission of ADHD symptoms, our decision had been to use a strategy consistent with newer models of this disorder that emphasized impaired regulatory processes measured on continuous dimensions

that may underly several related disorders (for a current version of this thinking see Martel, 2009). Within this frame, we selected two continuous measures of attentional regulation in adults: an 18-item questionnaire equally divided between symptoms of inattention and hyperactivity (K. R. Murphy & Adler, 2004) and a comparable five item scale measuring attention control (Evans & Rothbart, 2007; Rothbart, Ahadi, & Evans, 2000). Results are presented in Figure 12.

The zero-order correlation between birth mother ADHD symptoms and child ADHD symptoms was −.02 in the larger Cohort I and .19 in Cohort II, illustrating the modest and inconsistent level of associations between parental and child ADHD symptoms. In addition to the results shown in Figure 12, there was a significant indirect path from birth mother ADHD symptoms to adoptive mother hostility, via child impulsivity and activation but no comparable significant path to adoptive mother depressive symptoms. In another indirect path, child impulsivity/activation also predicted adoptive father hostility at child age 4.5 years that, in turn, predicted child ADHD symptoms at 6 years. Multigroup analysis suggested there was no overall significant difference between the models for Cohorts I and II. Overall, these results identify an important evocative child effect on adoptive mothers' hostility and delineate child impulsivity/activation as the evocative characteristic. Equally important, these data convey the critical role of both adoptive maternal and paternal hostility in predicting subsequent ADHD symptoms.

Liu et al. (2020): evocative child effects on parenting via birth parent risk and prenatal environment.—We sought corroboration of these findings with an analysis of broader internalizing and externalizing in the birth parents. Specifically, we created an index for genetically influenced risk that includes composites of birth parent internalizing symptoms, externalizing symptoms, and substance use for birth mothers and birth fathers separately (see Marceau et al., 2019 for a detailed description and rationale). Of note, to partially distinguish genetic from prenatal influences, we did not include in our genetic risk index those psychiatric symptoms or disorders that appeared only in pregnancy for birth mothers. We used, as a separate score, birth mothers' use of illicit drugs during pregnancy. As several studies have shown, maternal drug use in pregnancy can be linked to child outcome based on genetics and fetal exposure (Palmer et al., 2016; Rice et al., 2018). As birth father substance use is included in the analyses, it serves as an approximate control for genetic confounding (see Loehlin, 2016). The main link between birth father substance use and child negative reactivity at 18 months is expected to be genetic. Thus, our measure of illicit drug use by birth mothers during pregnancy captures some of the actual exposure effects. Parental negativity at child age 18 and 27 months was indexed using adoptive mother and father reports of their overreactive parenting and hostility. The main results are shown in Figure 13.

As can be seen in the Figure, there are significant indirect paths from birth mother internalizing and externalizing symptoms to adoptive mother and father parental negativity via child negative reactivity. The indirect path from birth mothers' substance use to adoptive mother and adoptive father negativity via child negative reactivity was also significant but with an effect in the opposite direction of the effects of birth mothers' internalizing and

externalizing symptoms. Note that the likelihood of this path reflecting fetal exposure, rather than a genetic influence, is enhanced by including birth father substance use in the model.

There are two surprises in these findings. The first surprise is the absence of any effect of birth father psychopathology; this null finding may be due to the lower prevalence of internalizing problems in birth fathers than in birth mothers or that much of the effect of birth mother internalizing symptoms is transmitted via the prenatal environment rather than genetically. More surprising is the emergence of birth mother illicit drug use during pregnancy predicting lower levels of child negative reactivity at 18 months, an opposite direction to that of maternal internalizing symptoms. In an adoption design, prenatal exposure can be separated from the effects of parenting by the rearing parents and hence exposure effects on the fetus can influence the rearing parents only through its impact on some evocative postnatal child behavior. But why is this effect in the opposite direction from maternal depressive symptoms? Studies of cocaine exposed infants and toddlers suggest that in exposed toddlers, lower negative reactivity may reflect disengagement, passivity or even sadness. For example, cocaine exposed toddlers fail to show the typical arousal on separation from their mothers, instead showing indifference (Molitor, Mayes, & Ward, 2003). Likewise, cocaine exposed toddlers' reaction to medically required inoculations contrasted sharply with controls: they displayed less anger and distress and more sadness (Roumell, Wille, Abramson, & Delaney, 1997). Earlier in this chapter, we reported that social disengagement in the child can evoke negative parenting. However, in the current analysis, there is a robust association between child negative reactivity at 18 months and negative maternal and paternal parenting 9 months later despite the high stability of negative parenting across this time period. Thus, it is unlikely that birth mother use of illicit drugs while pregnant is leading to children's disinterest in interacting with their parents and preference for solitary play (Olson et al., 1982). The effects on the child of substance use during pregnancy must be different: perhaps a simple blunting of negative reactivity and thus relief for parents of toddlers.

Evocative Effects Leading to Child Outcomes

We have reviewed studies suggesting the birth parent variables demonstrate an influential role for the impact of child characteristics on how they are treated by their parents. So far, we have noted no instances of these child effects being a component of the mechanisms by which parents transmit genetically their traits to their offspring. However, as we have just reviewed, parental temperament has been a reliable influence on child outcomes. Thus, in our most recent analyses, we sought two improvements in measures of birth parents' temperament. First, we included birth father scores and imputed scores that were missing, as was done in the Liu et al. (2020) paper described in the previous section. Second, we measured temperament in birth parents using factor scores derived from confirmatory factor analysis of birth mother and birth father temperament constructs. The creation of these birth parent temperament indices is described in detail in Shewark et al. (2021) and resulted in four factors: *Behavioral Activation*, *Orienting Sensitivity, Agreeableness*, and *Emotional Dysregulation*.

Shewark et al. (2021): child effects on parenting and the role of birth parent temperament.—We reasoned that the child negative emotions of anger or sadness might evoke strong and perhaps different parental responses (Cassano, Zeman, & Sanders, 2014; Oliver, 2015; Snyder, Stoolmiller, Wilson, & Yamamoto, 2003). We measured parent-reported child emotionality at age 4.5 years using the Child Behavior Questionnaire (Evans & Rothbart, 2007; Putnam & Rothbart, 2006), parent-reported parental warmth and hostility (Melby & Conger, 2001) at child age 6 years, and child behavioral problems using the Child Behavior Checklist – Long Form (CBCL) and social competence as revealed in peer play at child age 7 years (McWayne, Sekino, Hampton, & Fantuzzo, 2002). We focus here and in Figure 14 on the multiple indirect pathways from birth parent emotional dysregulation and behavioral activation (see Shewark et al., 2021 for a full account of this study). First, note that parental warmth is not involved in any of these pathways and, despite reasonable expectations (Carver & Scheier, 2013), behavioral activation did not have an inverse relationship with child sadness. However, birth parent behavioral activation was associated with child anger which, in turn, elicited hostility from both parents with adverse consequences for adopted children by age 7, including diminished social competence and increased externalizing. In addition, birth parent emotion dysregulation was indirectly associated with age 7 externalizing and social competence via adoptive father hostility.

These results paint a fuller picture of the role of birth parent temperament in the genetic transmission of liability for problems of adjustment in their children. Parental emotion dysregulation also plays a significant role in the effectiveness of their own parenting (for a review see Rutherford, Wallace, Laurent, & Mayes, 2015). Thus, biological parents rearing their own children genetically transmit characteristics to their children that evoke their negative parenting in them, a parenting pattern that is also influenced by their own difficulties in regulating their emotions.

Fearon et al. (2015): child effects on mother negativity over time.—We have begun to explore what factors may moderate child effects on negative parenting in EGDS. Specifically, we have examined adoptive parents' marital quality as a potential moderator of these effects (Fearon et al., 2015). We used broad measures of birth mother internalizing and externalizing symptoms to measure genetic influences on the child, and growth curve modeling was used to cumulate data on three scales of self-reported parenting for adoptive mothers at child ages 9, 18, and 27 months. This strategy allowed us to distinguish the impact of child effects on the absolute level of adoptive mother negativity (the intercept of the latent growth curves) versus its effect on change in adoptive mother negativity over time (the slope).

Based on previous research on parenting, there are at least three plausible factors that might moderate the negative impact of child effects on parenting (and hence constitute "breaks" on the spiral). These are economic security of the parents (Mills-Koonce et al., 2007), freedom from affective disorder or other psychopathology (Lorber & Slep, 2005) and a secure and satisfying marriage (O'Leary & Vidair, 2005). Any of these three might plausibly reduce negative parental reactivity to a challenging child. The results were quite clear at all three times of assessments −9, 18 and 27 months. Only marital quality moderated adoptive mother response to infants whose birth mothers were higher in externalizing. More specifically,

for infants and toddlers whose birth mothers were higher on externalizing symptoms, the quality of the adoptive parents' marriage was the only one of the three plausible breaks to demonstrate a statistically reliable effect: for higher quality marriages where children were at genetic risk for externalizing disorders, the adoptive mothers' negativity was lowest across all three time points when there were few marital problems, but the highest in the context of high marital problems (for a full description of this study see Fearon et al., 2015).

In contrast, there was little or no effect of marital quality for children at low genetic risk. These data are summarized in Figure 15. Measures of difficult child temperament in this study did not provide evidence of which inherited characteristics of the child evoked the self-reported parenting. Birth parent externalizing was associated with rearing parent hostility, but the intermediate child characteristic could not be identified. The moderation of maternal negativity by marital quality played a role in the further development of the children: we found a significant indirect path linking the interaction of marital quality to birth parent externalizing via rearing parent negativity.

Overall Summary of This Chapter

Our studies of child effects are at an early stage and will become more informative as the children in our study grow older. However, we can draw some preliminary lessons from the findings we report here.

First, measurement of temperament in birth parents, in contrast to psychopathology symptoms, has thus far been an illuminating index of child effects. We have demonstrated four dimensions of temperament that are linked to child effects on the parenting received: reward dependence, behavioral activation, attention control, and emotional dysregulation. Psychiatric symptoms, particularly externalizing and antisocial symptoms, were generally not linked to child effects on parenting. The three instances where there were links between birth parent symptoms and child effects on parenting were when (1) measures of temperament were added to the birth parent index (Sellers et al., 2020), (2) the measurement of symptoms included age of onset and family history of psychiatric problems (Liu et al., 2020), or (3) when a moderator variable was added to the analysis (Fearon et al., 2015). For emphasis, none of our studies were designed to make rigorous comparisons between psychological symptoms or examine the role that the restricted range of psychiatric symptoms may have. Despite this limitation, we might reach a provisional conclusion: the temperament measures we have used define fundamental processes regulating behavioral reactions to challenges, attentional focus, emotional processes and stimulus seeking. Dysregulation of any of these processes are the seeds for potential behavioral and emotional problems in childhood if they are augmented by correlated or moderating environments.

Second, we have only the most preliminary findings when using favorable temperament in birth parents regarding positive outcomes for children. An intriguing finding is the impact of *birth mothers*' reward dependence on the reduction of *rearing fathers'* hostility (Hajal et al., 2015). Generalizing to families rearing their biological offspring, we see the operation of *protective genetics:* children inherit features from one parent that protect them against another. We continue this discussion in Chapters V and VI.

Third, a range of temperament characteristics are transmitted from birth parents to the parenting of adoptive parents via characteristics of the child. The latter includes temperament and emotionality. Specifically, genetic influences on child characteristics evoke a particular parental response (e.g., Liu et al., 2020; Sellers et al., 2020; Shewark et al., 2021). These findings are consistent with findings from studies using other genetic designs (see Broderick & Neiderhiser, 2019 for a review) that examine how parents are responding to their children's behavior. The EGDS findings, however, take these general patterns of findings a step further by identifying which child characteristics are evocative, thus identifying potential intervention targets. We have also found some evidence that different aspects of the rearing environment (i.e., marital relationship) may moderate child effects on parenting in important ways (Fearon et al., 2015), thereby emphasizing the importance of considering multiple relationships within the family and possible genetic influences. This research and ideas are discussed more extensively in the remaining chapters of this monograph.

It is also important to consider the possible socioeconomic factors that may influence the child effects found in EGDS. Specifically, as noted in Chapter II, the adoptive households are economically advantaged. As a result, there may be more opportunity for genetic effects to emerge. Other studies have found that, for example, genetic influences accounted for half of the total variance in cognitive ability in infants raised in higher SES homes as compared to children raised in lower SES homes (Tucker-Drob, Rhemtulla, Harden, Turkheimer, & Fask, 2011). In other words, we may find links between birth parents and adopted children that are associated with rearing environment that may not be present in a different context. We are including more detailed measures of social context in the EGDS, including information that incorporates geographical information for household, school, and activities that will facilitate a more nuanced consideration of the larger context of the rearing environment in future analyses.

Chapter V: Child Effects Moderate the Influences of Parenting on Children's Own Development

In Chapter III, we reviewed evidence for direct parenting effects on child development that were estimated free of the artifact of genes common to parent and child. In Chapter IV, we focused on the reverse, child effects on their rearing parents and parent characteristics. We focused on those child effects that were genetically influenced. In this Chapter, we focus on how child effects, especially those that are genetically influenced, can moderate the effects of parenting on children's development. The focus of this Chapter is summarized in Figure 16.

Introduction

Two sorts of evidence are frequently offered to support the idea that genetically influenced characteristics of the child can moderate the influence of parenting on children's own development. The first is the effect of a child's temperament on the impact of parenting. Indeed, there have been enough prospective studies of this genre so that a recent meta-analysis could summarize the results of 84 separate studies and 235 effect sizes (Slagt,

Dubas, Dekovic, & van Aken, 2016). Of various temperament characteristics investigated, only negative emotionality—or "difficult temperament"—was a consistent moderator of parenting effects on both psychopathology and social and cognitive competence. Although temperament is often regarded as a built-in property of the child, and Chess and Thomas certainly regarded it as just that (1963), genetically informed studies have produced mixed results, with some showing little heritability (e.g., Rhee et al., 2012; Schumann et al., 2017) and others showing more consistent genetic effects (e.g., Vertsberger et al., 2019).

A second type of evidence supporting the moderating contribution of parental influence by a genetic influence comes from studies of the statistical interaction between a specific polymorphism and a measure of parenting on some aspects of child development. A number of these studies focus on the infancy and toddler period, as we do (see for example, Fox et al., 2005; Green et al., 2017; Van Ijzendoorn & Bakermans-Kranenburg, 2006). Early in this line work, Caspi and colleagues reported on the interacting effect on adult depression of variations in the serotonin transporter gene and adult and childhood stress (2003). This report was exemplary in four ways. First, it was built on prior work showing the role of serotonin transporters on stress response mechanisms in the brain (Hariri et al., 2002). Second, the study was adequately powered. Third, it showed the same effect across multiple measures of depression. Fourth, it ruled out the confounding effect of an interaction between a gene that elicited stress and a second gene that moderated people's response to stress. Many similar papers were published to give hope that ascertaining a single polymorphism that—by moderating a person's response to stress—would be a reliable risk factor for depression. However, few of these papers matched the rigor of the original Caspi paper (Reiss, 2016). In addition, they have been underpowered and there have been numerous failures of replication (Border et al., 2019; Culverhouse et al., 2017; Duncan, Ostacher, & Ballon, 2019; Harden, 2021; Munafo, Zammit, & Flint, 2014). Further, many studies suggest that complex traits such as "stress sensitivity" are likely to be influenced by scores, if not hundreds of genes, each with a tiny effect. Indeed, the biological foundation of the Caspi work, the role of the serotonin transporter in amygdala reactivity is, at best, small and often unreplicated (S. E. Murphy et al., 2013). The same is true for experiments testing this polymorphism's effect on stress sensitivity measured behaviorally (R. Miller, Wankerl, Stalder, Kirschbaum, & Alexander, 2013). Thus, the claim that a single polymorphism in the child's genotype has a substantial moderating effect on parental influence seems increasingly uncertain and unwarranted.

More recently, investigators have explored gene by environment interaction with polygenic scores derived from replicated and large samples. Environments include retrospectively reported childhood maltreatment, parenting, neighborhood qualities, and randomized interventions, with outcomes including child depression and conduct problems, as well as smoking and illegal substance abuse (see for example Nelemans et al., 2021; Pasman, Verweij, & Vink, 2019; Shaw et al., 2019). Although promising, these studies have, in general, collected DNA and computed polygenic scores long after the studies have been initiated. Even at that, they have emerged so recently that replications have yet to be conducted, much less reported. We think that a fair reading of the molecular genetic literature on the role of genes in family relationships should underscore its promise, but also

an abiding hunger for more solid information from a broader variety of research designs, including the EGDS.

Quantitative genetics, using twin and adoption designs, has already provided promising evidence that a child's genotype moderates parental influence (Dick, 2011). For example, Feinberg and his colleagues showed in a twin and sibling study that genetic influences on antisocial behavior were higher when the level of negative parenting was higher (2007). Cadoret and his colleagues (1995), using an adoption sample, also showed that genetic risk for antisocial behavior—indexed by severe antisocial behavior in the birth parents—augmented the impact on antisocial behavior of a dysfunctional parental environment (parental mental illness, legal problems or divorce). There is an important distinction between the objectives of twin and adoption studies of interaction between a child's genotype and parental influence. Twin studies aim to capture the moderating role of the entirety of the child's genotype. Adoption studies focus on parent to child transmission and ask does the genetic transmission process of a particular trait, from parent to child, involve the augmentation or diminution of parental environmental influences?

Review of EGDS Studies

Drawing on evidence of specificity in the development of some syndromes, we examine the role of genetic liabilities indexed by maternal anxiety symptoms and, consistent with the work cited above (Hirshfeld-Becker et al., 2007; Kostyrka-Allchorne et al., 2020; Lahat et al., 2014), focus on its links to social and behavioral inhibition in infants and toddlers. In non-genetic studies, behavioral inhibition in children has been linked to maternal anxiety symptoms and children's own later anxiety disorders, especially social anxiety (Clauss & Blackford, 2012; Hudson, Dodd, & Bovopoulos, 2011) We then review our analyses of maternal depressive symptoms and the emergence of children's negative affective, hyperarousal, and poor response to soothing. As noted above, while there is a reliable association between these child characteristics and maternal depression, maternal depression also predicts a wide range of internalizing and externalizing problem behaviors in older children and adolescents. Finally, drawing on evidence of a general psychopathology factor, we examined the association of a broad range of both maternal and paternal psychopathology with the emergence of an equally broad range of problem behaviors in toddlers. In all these studies we control for the confounding variation in adoption openness and prenatal and perinatal influences, including illicit and prescription drug use.

Using Birth Parents' Psychopathology to Index Genetic Influences.

Brooker et al., 2016: Moderation of parental responsiveness on child inhibition from 9 – 18 months by birth parent anxiety symptoms.—At the earliest possible developmental period our study allowed, we studied the relationship between birth parent anxiety symptoms, adoptive parent caregiving, and the change in children's behavioral inhibition from 9 to 18 months. Birth parent anxiety symptoms were measured as a continuous variable (Beck & Steer, 1993). Parenting was measured at 9 months based on observational coding of parental responsiveness during a teaching task. Behavioral inhibition was observationally coded from the infant's reaction to a standardized four-minute exposure to a stranger (9 months) and a scary object (18 months). We found no direct effects of

birth parent anxiety symptoms or parenting on change in behavioral inhibition from 9 to 18 months. But we did find a clear interaction between fathers' responsive parenting and birth parents' anxiety symptoms in relation to children's inhibition. When birth parent anxiety symptoms were low, father's responsiveness yielded the expected result: higher adopted fathers' responsive parenting was related to greater decreases in child inhibition from 9 to 18 months. However, when birth parent anxiety symptoms were high, the reverse occurred: father's responsive parenting was associated with greater increases in child behavioral inhibition. This pattern fits Chess and Thomas' conception of goodness of fit: parenting that was effective for children at low genetic risk appeared detrimental for children at higher genetic risk. We were surprised to see father, but not mother effects, and by the positive relationship between father's responsiveness and child's behavioral inhibition when genetic liability was high.

Natsuaki et al., 2013: moderation of parental responsiveness on child inhibition from 18–27 months by birth mother social phobia.—We then moved to a slightly later point in development to examine stability and change in children's behavioral inhibition from 18 to 27 months. For this study, we narrowed our focus to birth mother lifetime history of social phobia using the Composite International Diagnostic Interview (CIDI; Kessler & Üstün, 2004). We distinguished between birth mothers who met DSM IV criteria for social phobia and those that did not. Both adoptive mothers and fathers were rated by trained observers for the responsiveness of their parenting as observed during a home visit. Raters used the observationally based items from the responsiveness subscale of the Home Observation for the Measurement of the Environment (HOME; Caldwell & Bradley, 1984). We assessed the child's behavioral inhibition by coding the child's response to a stranger and stimuli, designed in part to evoke individual differences in approach behavior (e.g., a robot that makes sounds and automatically approaches the child). We also asked parents to rate their child's symptoms of anxiety using the Child Behavior Checklist (Achenbach, 1992). We found again a significant interaction between birth mother's social phobia and adoptive parents' responsiveness. For both, we found no main effects of birth mothers' social phobia on either the children's observed behavioral inhibition or parent-rated anxiety symptoms. However, where birth mothers met criteria for social phobia, responsive parenting by mothers was inversely associated with both the child's behavioral inhibition and adopted father-rated anxiety symptoms. In contrast, adoptive mothers' responsiveness was unrelated to children's behavioral inhibition or to their anxiety symptoms when birth mothers did not meet criteria for social phobia. This pattern of findings suggest that responsive parenting compensated for children's heritable risk for behavioral inhibition.

Natsuaki et al. (2010): moderation of parental responsiveness on negative affectivity from 18 to 27 months by maternal depressive symptoms.—We found a similar pattern when we examined the relationship between maternal depressive symptoms and the change in infant's negative affectivity from 18 to 27 months. As noted above, in non-genetic studies, negative affectivity (and rapid arousal that is difficult for parents to soothe) in infants and toddlers is reliably associated with maternal depressive symptoms. Negativity affectivity is also a general risk factor for a broad range of subsequent

internalizing and externalizing problem behaviors. We assessed mothers' lifetime history of depression using the Composite International Diagnostic Interview. We measured adopted children's negative affectivity using parents' ratings on the Difficultness subscale of the Infant Characteristics Questionnaire (Bates, Freeland, & Lounsbury, 1979). For analyses focusing on fathers' parenting, we used mothers' ratings of infant temperament and vice versa. Adoptive parents' responsiveness was observationally rated in their home using items from the HOME (Caldwell & Bradley, 1984). Neither birth mother depression nor adoptive parents' responsiveness had a direct effect on change in infant negativity from 18 to 27 months. However, we found a notable interaction between birth mothers' depression and adoptive mothers' responsiveness. As in the analysis of birth mothers' social phobia, we again found that adoptive mothers, but not adoptive fathers', responsiveness reduced the likelihood of an adverse change in the child's negativity, but only when the child's birth mother met qualifications for a psychiatric disorder, in this case a Major Depressive Disorder (see Natsuaki et al., 2010 for a complete account of this study). This finding is consistent with a compensatory or protective role of parenting for children at elevated heritable risk for high negativity.

Using Birth Parents' General Psychopathology as an Index of Genetic Influence

Leve et al. (2009): moderation of structured parenting effects on children's behavioral problems by birth parents' general psychopathology.—As we have noted, there is a growing interest in identifying a common factor that might underly genetic risk for a broad range of psychopathology. Thus, in an additional exploration of moderation of parenting effects in infancy and toddlerhood, we constructed a broad index of psychopathology separately for birth mothers and birth fathers combining alcohol and substance use, antisocial behavior, depressive symptoms, and anxiety symptoms. Against this backdrop of genetic liability for a range of affective and behavioral problems in toddlers, past research supports the importance of parental guidance and structuring (e.g., Denham et al., 2000; Gardner, Sonuga-Barke, & Sayal, 1999; Holden & West, 1989). We coded this form of parenting in a three-minute clean-up task when the child was 18 months old, where the adoptive mother was instructed to get the child to put away toys in a basket they previously had been playing with. We coded the frequency with which mothers provided verbal guidance to the child's clean-up efforts. Mothers also completed the Child Behavior Checklist. We used two temperament measures obtained when the child was 9 months old to control child risk before obtaining our parenting measures. These were infants' distress to limitations and fearfulness as rated by adoptive mothers and fathers on the Infant Behavior Questionnaire (Rothbart, 1981). There was no main effect for either birth mother or birth father psychopathology on adopted child problems at 18 months and the main effects of structured parenting were marginally significant in the expected direction. However, the interaction was significant and similar in magnitude to the interaction of birth parent anxiety symptoms and father's responsive parenting reported above. That is, where genetic risk for a broad range of child problems was high, structured parenting had a favorable effect on child problems; the reverse was true for children with low genetic risk (see Leve et al., 2009 for a complete report of this study). We found the same pattern when birth fathers' psychopathology was substituted for birth mothers' psychopathology as the index of genetic risk. This corroboration not only served as a quasi-

replication of findings with birth mothers' psychopathology but reinforced the hypothesis that the primary child effect was indeed genetic rather than through prenatal exposures. The pattern is shown in Figure 17.

We performed a comparable analysis of interaction between birth parent psychopathology and structured parenting at 18 months using data from the Child Behavior Checklist at 18, 27, 54, 72, and 84 months. We identified four distinct groups of children by their distinct pattern of change across this time. For offspring of birth parents with high psychopathology, there was a high likelihood that structured parenting predicted their being in the group of children who had stable and low scores on the CBCL across this time period. Correspondingly, children with the same risk who received structured parenting at 18 months were less likely to be found among those who had stable high or increasing scores. In addition, offspring of birth parents with low psychopathology who received highly structured parenting at age 18 months were more likely to be found in the high, stable group and less likely to be in low stable group. These data suggest that interactions we have observed in early development may have persistent effects across significant phases of child and family development (Leve et al., 2022).

There were some differences for the pattern found here and that for birth parent anxiety symptoms and fathers' responsive parenting discussed previously (Natsuaki et al., 2013). For birth parent anxiety symptoms, a presumably salutary parenting style had its predicted effect in the absence of genetic risk. Here, structured parenting had its predicted favorable effects only for children high on genetic risk. However, in both cases, the findings are consistent with Chess and Thomas' (1963) concept of goodness of fit.

Cree et al. (2020): moderation of several parenting measures on child externalizing and social competence from 9 to 27 months by birth parents' general psychopathology.—Since the analyses reported in the previous section were published, several investigators—as we have noted—(Caspi et al., 2014; Gluschkoff, Jokela, & Rosenstrom, 2019; Laceulle, Vollebergh, & Ormel, 2015; Lahey et al., 2012; Lahey et al., 2015; Martel et al., 2017). As alluded to earlier, best fitting models often show both a general psychopathology factor and an internalizing and externalizing factor that accounts for some of the comorbidity among subsets of diagnoses. While the general psychopathology(e.g., Neumann et al., 2016), it is unknown how the liability for the general psychopathology factor may first appear in very early childhood nor what environmental circumstances might moderate its earliest expression.

EGDS fit a bifactor model to the psychopathology of birth mothers as measured by the Composite International Diagnostic Interview (Kessler, Chiu, Demler, & Walters, 2005) and the Diagnostic Interview Schedule (Robins, Helzer, Croughan, & Ratcliff, 1981). As expected, we replicated an excellent fit between the bi-factor model and comorbidity patterns of the birth mothers. Importantly, the general psychopathology factor was positively associated with father rated child externalizing and inversely associated with social competence at 54 months. We measured environmental moderators of the influence parental psychopathology. They were measured across 9–27 months; we measured the child's externalizing and social competence at age 5. The three domains were parenting stress as

indexed by a composite of the over-reactivity scale of the Parenting Scale (Arnold, 1993), the Parental Efficacy Scale (Teti & Gelfand, 1991), and the Parenting Daily Hassles Scale (Crnic & Greenberg, 1990). We also assessed marital quality, as rated by adoptive mothers and self-reports of their depressive symptoms. By 27 months, only the parenting stress moderated the expression of the general psychopathology factor as shown in Figure 18.

The Cree et al. study was designed to maximize the chances of finding a pattern of genotype by environment interaction that fit the pattern of differential susceptibility (DS): that children with genotypes that put them at greater susceptibility for psychopathology in adverse environments also placed them in an advantageous position to benefit most from favorable environments. This analysis met the essential criteria for detecting a DS from a genotype by environment interaction. First, children should be exposed to both positive and negative environments. Second, there should be substantial variations in both positive and negative environments. Third, outcomes studied should be both favorable and unfavorable (see Cree et al., 2020 for a complete description of this study). Three features of these findings merit comment. First, these are the first findings of which we are aware that document the substantial role of the general psychopathology factor, as a genetically influenced child effect, in moderating the impact of the social environment on the development of very young children. Second, we did not find evidence supporting the DS hypothesis. Third, the pattern of moderation found is unusual. We observed two patterns of interaction. First, children with birth parents high in general psychopathology were not different from one another in externalizing or in social competence regardless of their parents' levels of parenting stress. At *low levels of* birth parental general psychopathology, variation among children in externalizing was positively associated and social competence was inversely associated with parenting stress. The reverse was the case for mothers' internalizing symptoms where decreases in social competence among children were associated with increasing internalizing of rearing mothers only for children *high* in general psychopathology in birth parent general psychopathology. In other words, for parenting stress, birth parent high in general psychopathology appeared to confer protection from parental stress but a high level of vulnerability to maternal internalizing. It is possible that these two external stress systems — parenting stress and maternal internalizing— have very different mechanisms of impact on the child and hence different genetic moderators. However, our sample may contain (at least) two different types of children; one where low levels of birth parent general psychopathology make them especially sensitive to parental stress and a second group where a high level of general psychopathology makes them sensitive to maternal depressive symptoms. Differences of this kind, among subgroups of children, have just begun to be explored by Belsky, Zhang, and Sayler (2021).

Underlying and Stable Patterns of Self-Regulation and Environmental Sensitivity as Indices of Genetic Influence

Ganiban et al. (2021): moderation of lax vs. structuring parenting styles on child effortful control at age 4.5 by maternal emotional dysregulation and agreeableness.—The capacity of children to voluntarily control their attention and their goal-directed behavior, referred to as effortful control, appears early in childhood. EGDS showed that effortful control at age 27 months predicted, independent of quality of parenting

by the rearing parents, teachers' rated aggression and rule breaking behavior at ages 6 and 7 (for a full description of this study see Reuben et al., 2016). While, as noted above, birth parent executive functioning indexes genetic factors expressed in adopted children using comparable assessments. However, there are good reasons to explore other birth parent indices. First, as already noted, birth parent executive functioning is only a partial index of the genetic factors transmitted to the child. Second, other dimensions of temperament have been regularly associated with executive functioning and may, in birth parents, index additional genetic assets.

Ganiban et al. (2021) used birth mothers' emotion dysregulation and agreeableness as indices of heritable predisposition for effortful control. Emotion dysregulation is conceptually like neuroticism and reflects heightened negative affectivity vs. self-regulation. Agreeableness reflects the degree to which a person forms and maintains positive relationships with others vs. antagonizes others. Theories and previous research link both personality characteristics to effortful control and/or self-regulation. For example, the two components of emotion dysregulation - negative affectivity and self-regulation - are intertwined during adulthood (Digman, 1997) and throughout development (Cioffi, Griffin, et al., 2021; Ferrier, Bassett, & Denham, 2014) and share common genetic influences (Ganiban, Chou, et al., 2009). Likewise, agreeableness is correlated with measures of self-regulation during adulthood (Digman, 1997; Laursen, Pulkkinen, & Adams, 2002), adolescence (Kochanska & Kim, 2020)and childhood (Cumberland-Li, Eisenberg, & Reiser, 2004). Therefore, we reasoned those low levels of emotional dysregulation and high levels of agreeableness would represent heritable predispositions for strong effortful control skills, while high levels of emotion dysregulation and low levels of agreeableness would index heritable predispositions for weak effortful control skills (Spengler, Gottschling, & Spinath, 2012; Vukasovic & Bratko, 2015; Willems, Boesen, Li, Finkenauer, & Bartels, 2019).

The development of effortful control is also influenced by parenting. A meta-analysis summarizes evidence of the association between parenting style and the development of effortful control and includes non-contingent parenting and over-involvement among other caregiving dimensions (Karreman, van Tuijl, van Aken, & Dekovic, 2008; Karreman et al., 2006). This paper raises the question of whether heritable factors and parenting style operate independently or work together to shape the development of effortful control. There are some findings that support interactions between candidate genes and parenting in predicting effortful control during childhood (Kochanska, Philibert, & Barry, 2009; Sheese, Rothbart, Voelker, & Posner, 2012; H. J. Smith et al., 2012) and adolescence (Cho, Kogan, & Brody, 2016; Conway & Stifter, 2012; Sulik et al., 2015; Van Heel et al., 2020) However, these previous studies are limited by the consideration of single genes and did not consider passive gene-environment correlation in their estimates of gene by environment interactions. Therefore, further exploration of the interplay between children's heritable predispositions and parenting is needed. In this analysis, we examined the degree to which parent structuring vs. laxness interacted with children's heritable predispositions to predict effortful control.

We found that birth parent agreeableness and emotion dysregulation interacted with parental use of structure vs. laxness during the toddler period to predict effortful control during early childhood (4.5 years). As depicted in Figure 19, children who were genetically

predisposed to have strong effortful control skills developed higher levels of effortful control when their rearing parents reported very high levels of structure but fared worse when parent structure was combined with low levels of laxness. In contrast, children who were genetically predisposed to have poorer effortful control skills developed higher levels of effortful control when their rearing parents reported styles that included structure combined with low levels of laxness but showed lower levels of effortful control when their rearing parents were highly structuring.

Importantly, the paths from each of these interactions to child externalizing at age 7, via executive function, were significant. We drew two lessons from these findings. First, we again encountered a pattern of interaction, consistent with the goodness of fit model, where a child effect reverses the impact of a self-reported parenting strategy. Moreover, each of these interactions is relevant not only for the emergence of executive function but for the appearance of externalizing problems 2½ years later.

Second, by accounting for differences among children and their primary effect on the impact of parenting, we see parental laxness variable in a new light. In a very large literature on this variable, this finding is rare. However, it is important to note that in our study, most of our parents were highly structuring: the key difference was that some parents combined high levels of structure with moments of laxness. For children who may be predisposed to have difficulties with self-regulation, this adaptation may be particularly important as they might benefit from a lighter disciplinary touch. Similar to our pattern of findings, a handful of studies have suggested that other parenting styles that are often perceived to be optimal may not benefit all children equally. For example, Hughes, Devine, Mesman, and Blair (2020) report that for toddlers, maternal support is inversely associated with externalizing behavior, but only for children who show low levels of negative affect. Likewise, Rioux et al. (2020) found that during early childhood positive parenting (supportive, structuring) predicted fewer hyperactivity-impulsivity/inattention symptoms, but only for children who have higher levels of inhibitory control. Additionally, Newland and Crnic have shown that toddlers with low activity levels benefit from maternal scaffolding—as indexed by assisting children's engagement and accomplishments in defined tasks. Whereas highly active children do not (Newland & Crnic, 2017). Collectively, these studies paint a picture of the effects of parenting being moderated by children's characteristics. In other words, consistent with goodness-of-fit models, different types of parenting are needed to optimize development for children with different temperaments (Chen & Johnston, 2007; Lagace-Seguin & Coplan, 2005; Newland & Crnic, 2017).

Van Ryzin et al. (2015): moderation of responsive parenting on child social competence at age 6 by birth parent sociability.—In our final examination of child effects that moderate the influence of parenting, we examined a favorable developmental outcome in children, their social competence. Was social competence at age 6 predicted independently by birth parent sociability and adoptive parent responsive parenting and—of greater interest—by their interaction? We measured the mean of birth mother and birth father sociability as indexed by the Sociability Scale of the Adult Temperament Questionnaire (Evans & Rothbart, 2007). We again assessed responsive parenting but, in this analysis, measured it as a latent construct combining adoptive mother and father scores on

the responsive scale of the HOME (Caldwell & Bradley, 1984) and observers' global rating of parent responsiveness in their home setting during a clean-up and teaching task when children were 27 months old. The child's social competence at age 6 was a latent construct indexed by separate adoptive father and mother ratings of their child, using the Social Skills Rating System (Gresham & Elliott, 1990) and teacher ratings using the Walker-McConnel Scale of Social Competence and School Adjustment (H. M. Walker & McConnell, 1988). Child's positive emotionality at age 9 months was included in the analysis as the best available variable to account for the child's baseline of affiliative behavior prior to our estimating parental effects. For the third time, we encountered a goodness of fit pattern in the interaction and one that was quite like those found between birth parent psychopathology and structured parenting. For children whose birth parents were high in sociability, highly responsive parenting had a negative effect, whereas for children whose birth parents were low in sociability, adoptive parent responsiveness had a salutary effect. As shown in Figure 8, the significant difference between high and low genetic influence occurred in the presence of unresponsive parenting, thus suggesting genetic influence indexed by birth parent sociability might protect a child from the unfavorable effects of unresponsive parenting.

Overall Summary of This Chapter.

The studies we have just summarized of the moderating influence on parenting of genetically influenced child effects bring us several steps closer to a mechanistic understanding of the importance of gene by environment interactions. First, we affirm previous findings from twin and adoption studies that genetic moderation of environmental influences are plentiful during the toddler period; this pattern reinforces and corroborates a modest number of prior adoption and twin studies in early childhood that have also demonstrated these effects (e.g., Plomin, DeFries, & Fulker, 1988; Tucker-Drob & Harden, 2013; Yan, Benner, Tucker-Drob, & Harden, 2017).

Second, while we have found many examples of gene by environment interactions in infancy and early childhood, it is unclear how they are related to the very sizable large-scale interactions between family social process and genetic influence on the development of adult psychopathology (e.g., Cadoret et al., 1983; Cloninger et al., 1981; Wahlberg et al., 2004; Wahlberg et al., 1997). Are these interactions the start of a sustained process by which genes moderate the effects of parenting or are they relatively brief perturbations of parenting confined to early childhood? Importantly, we did observe that moderation of the effects of parenting by genetic characteristics of the child during the toddler period anticipates the trajectory of child behavior across childhood. In our first follow-up analyses of structured parenting and birth parent psychopathology, observations of parenting observed at 18 months anticipated child behavior through child age 8.

Third, as we noted earlier in this chapter, we used three different strategies for the selection of birth parent variables to identify genetic differences among our children: specific psychopathologic symptoms, a general psychopathology factor and temperament. Although all three strategies yielded promising results, they underscore a larger question: how is the liability for psychopathology transmitted from parent to child? What are the core

deficits most closely linked to genetic factors that—when transmitted from parent to child —create a liability for psychopathology in the child? For example, preliminary evidence from EGDS suggests that there is no reliable association between birth mothers' general psychopathology factor and a comparable construct in their children (unpublished data). Might the first manifestation be an adverse temperament such as negative emotionality and do birth parents with a comparable temperament—without psychopathology—place a child at the same genetic risk as parents with high general psychopathology scores? EGDS is well poised to address questions like these, especially as data become available as our children traverse adolescence.

Fourth, we have in this chapter another example of a positive child effect: birth parent sociability transmits to the child protection from the otherwise adverse effects of unresponsive parenting (by the rearing parents) on their social competence. It is widely recognized that the impact on the child of psychopathology in one rearing parent may be offset by either the mental health or positive involvement of the other parent. The great bulk of the literature, with notable exceptions (Foley et al., 2001; Marmorstein et al., 2012) focuses on maternal psychopathology and the potentially offsetting positive influences of fathers (Brennan, Hammen, Katz, & Le Brocque, 2002; Chang, Halpern, & Kaufman, 2007; Dietz, Jennings, Kelley, & Marshal, 2009; Mezulis, Hyde, & Clark, 2004; Tannenbaum & Forehand, 1994). Two explanations have been offered to explain the offsetting effects on the child of a mentally healthy parent: that the healthy parent provides favorable warmth, support and structure for the child or diminishes the adverse genetic load. EGDS findings bring to the fore a third possibility focusing on child effects: when favorable features of a parent are transmitted, they provide the child the capacity to elicit warmth and support from a rearing parent to diminish the intensity of adverse parenting or to render the child invulnerable to adverse parenting.

Fifth, the form of interaction varied across our analyses. In accord with current interest in interaction patterns suggesting differential susceptibility (DS), genetic factors that enhance positive outcomes and mitigate negative outcomes in adverse environments, we found only one interaction pattern clearly consistent with DS in a study examining birth mothers' major depressive disorder, adoptive mothers' responsive parenting, in relation to changes in child's negative affectivity from 18 to 27 months. However, we found the opposite pattern when examining birth mothers' anxiety symptoms and responsive parenting in fathers in relation to changes in children's behavioral inhibition from 9 to 18 months. We would have expected that, according to DS postulates, that favorable parenting led to positive outcomes in children at higher genetic risk; we found just the reverse. A similar pattern was duplicated in our analyses of parenting stress and birth mother general psychopathology factor: the expected effect was found for adopting parenting stress only for children at low genetic risk.

Indeed, a more consistent pattern in these findings supported a goodness of fit between parenting and genetic influences: for some children, some forms of parenting are beneficial but —for others—the very same parenting is detrimental. We found this pattern in five of the 10 interactions reported in this chapter and in our follow-up analyses from 18 months to 8 years. These findings provide, over half a century later, confirmation of the Chess and Thomas perspective on the role of "intrinsic" temperament in family dynamics. From

a practical perspective, the difference between “differential susceptibility” and “goodness of fit” may be important. Translated to preventive or therapeutic interventions, the findings consistent with differential susceptibility have supported a concept of the “downside of resilience” (J. Belsky, 2014). Children who are invulnerable to adverse environments also cannot benefit from good ones, including those provided by preventive or therapeutic interventions. “Goodness of fit” patterns suggest that interventions can be tailored to match the needs of children who are at high versus low risk for behavioral outcomes.

The concept of DS was originally formulated from studies of the statistical interaction of candidate genes with a broad variety of measures of environmental influence. Recently, it has been a productive paradigm for studying the interplay of environmental influences and other characteristics of children, characteristics that may or may not be primary such as brain morphology and connectivity (Deane et al., 2020; Rudolph et al., 2021), HPA activity (Xu et al., 2019), and sensory sensitivity (Carr, Matthews, Williams, & Blagrove, 2021). We may have found little evidence for this mode of gene by environment interplay because of limits of the adoption design. We reasoned that, if DS is a major mode of intergenerational transmission for children at genetic risk, as indexed by birth parent psychopathology or adverse temperament, such children should do better than other children if their rearing conditions are highly favorable. However, we may not have detected some children with the same genotypes because their birth parents were raised under favorable conditions and hence scored lower on our measures. To the extent we missed these birth parent-adopted child pairs is the extent to which we may have simply found no effect of our indices of genetic differences among our children on parenting. However, this limitation would not account for other patterns that we did discover.

In sum, evidence in Chapter IV and in this chapter emphasize that children bring to the family assets and liabilities that are genetically influenced. We have also provided an example of a liability associated with adverse prenatal exposure. This Chapter strongly suggests that parents vary in their response to these child characteristics and their response may play a crucial role in the ultimate significance of the children’s genetically influenced assets and liabilities. This straightforward idea motivates a summary of our work in Chapter VII.

As we emphasized earlier in the monograph, our sample of adoptive families is almost all White and economically comfortable. How might this skew influence estimates of both genetic and environmental influences we have reviewed in Chapters III and IV? There is a growing literature on the effects of socioeconomic status on estimates of genetic and environmental influences. Turkheimer and his colleagues, for example, reported the importance of genetic influence on children’s IQ is much less for economically disadvantaged families than it is for wealthier families (Turkheimer, Haley, Waldron, D’Onofrio, & Gottesman, 2003). One explanation is that severe privation counters any advantage offered by favorable genotypes. However, a recent meta- analysis showed interactions of SES and genetic influence are restricted to the US but not replicated elsewhere in primarily European countries (Tucker-Drob & Bates, 2016). One explanation is that it is only the extreme poverty faced uniquely by some American families that drives these interactions. Further, an exhaustive meta-analysis shows little differences in estimates

of genetic and environmental influence across racial and ethnic groups (Pesta, Kirkegaard, te Nijenhuis, Lasker, & Fuerst, 2020). However, theoretical perspectives consistent with a social push hypothesis (i.e., Bronfenbrenner & Ceci, 1994; Raine & Venables, 1984), and studies of child psychopathology (S. A. Burt, Pearson, Carroll, Klump, & Neiderhiser, 2020; Hendriks et al., 2020; Schonberg & Shaw, 2007) and on the developmental interplay of temperament and cognitive ability (Finkel et al., 2021) have reported the same patterns of findings as in the Turkheimer study: genetic influences are more apparent in higher SES children or those living in better neighborhoods. In sum, there are enough data to suggest that inferences drawn from our primarily middle class, White sample of adoptive parents may not be applicable to families of quite different economic circumstances.

Chapter VI Dynamic Balance and Reciprocation Between Parenting and Genetically Influenced Child Characteristics

Introduction

In Chapter III, we presented some of our findings on direct associations between measures of parenting and child development. Most of our studies presented longitudinal data and, as we have emphasized, because of the use of our genetically informed adoption design, all these findings were free of genetic factors common to parents and children. Two important confounds were ruled out—reverse causality and common gene effects. Thus, we came closer to defining the causal role of parenting in toddler and child development, albeit firm causal inferences are still not warranted.

In Chapters IV and V, we presented data on two forms of child effects. The first was on associations between genetically influenced child characteristics and self-reported and directly observed parenting. In a few cases, we could report on the subsequent associations of these evocative processes, on the further course of the child's development. Those data initiated our consideration of reciprocity in parent-child relationships: behavior that the child evokes to certain kinds of parenting which in turn, are related to later child behavior. This chapter explores further reciprocal processes between genetically influenced child behavior and adoptive parent caregiving behavior in more detail.

In earlier chapters, we also reported findings on child behavior in relation to parenting, noting several patterns. These included child characteristics that increased or decreased the positive effects of parenting on the child or where a child characteristic countered or buffered the child against adverse parenting. Particularly intriguing were instances where parenting that benefited children with one kind of genetically influenced characteristic was adverse for children with a different genetically influenced characteristic. This last pattern was reminiscent of the findings and conceptualization of Chess and Thomas on "goodness of fit" (1999). These findings also anticipated the focus of this chapter, namely the fine balance between parental and child characteristics in influencing trajectories of child development.

In this chapter, we focus more specifically on the balance between child and parent in the evolution of parent-child relationships and the intertwining of both influences on the child and, in some cases, on the parent. First, we consider evidence of parenting effects in early

childhood on children at higher genetic risk for developing callous unemotional behavior. The evidence for these parenting effects are statistical interactions between parenting by the rearing, adoptive parent, and birth parent characteristics. From a statistical perspective, this evidence is like findings we presented in Chapter V on child effects on parenting. For those findings, statistical interactions were best interpreted as child effects. However, in the examples to be presented here, evidence suggests—in contrast—that they be interpreted as parenting effects.

Second, we review three studies from EGDS where reciprocal effects were studied across time. We looked for evidence that parental influence on child behavior was then reciprocated by a child effect on the parent. Our adoption design permits us to estimate the role of genetic factors in the child on these reciprocal processes.

Parental Moderation of Genetically Influenced Maladaptive Child Characteristics

Hyde et al. (2016); Waller et al. (2016): parental positive reinforcement moderates callous/unemotional behavior in early childhood.—In the absence of intervention, the outlook is often poor for children who show high levels of callous/unemotional behavior. Early in middle childhood, they are more likely to show low empathy, disregard for pain and suffering in others, and impaired positive social behavior and emotions. When these traits are paired with elevated levels of early-emerging conduct problems, children are at elevated risk for developing more serious forms of antisocial behavior that persist into adulthood and criminal behavior (Frick, Ray, Thornton, & Kahn, 2014). Distinctive neural profiles and extremely high heritability distinguish these children from other children who have conduct problems but not callous-unemotional traits (Bolhuis et al., 2019; Jones, Laurens, Herba, Barker, & Viding, 2009; Viding, Blair, Moffitt, & Plomin, 2005; Viding, Jones, Frick, Moffitt, & Plomin, 2008; Viding et al., 2012). A clear understanding of mechanisms favoring or mitigating the development of callous/unemotional behavior could help to reduce the psychological, social, and community costs of its outcome in adult life (see a quantitative estimate of the costs of persistent antisocial behavior in childhood, Moffitt et al., 2011).

The EGDS offered a unique opportunity to study the interplay of genetic and parenting processes in the early development of callous-unemotional behavior. First, the EGDS has measures of severe antisocial behavior in the birth parents, using a short form of the Composite International Diagnostic Interview (Kessler et al., 1998). Second, the EGDS measures of conduct problems as early as 18 and 27 months can distinguish between manifestations of attentional problems, opposition behavior, and callous and deceitful behavior; the last includes parents' reports on the Child Behavior Checklist (Achenbach, 1992) of their child's lack of response to affection and little evidence of guilt after misbehavior or of fear. The prospective validity of these parental ratings has been tested in numerous samples including the EGDS (Waller et al., 2017). Finally, the EGDS has an established observational measure of positive reinforcement of child behavior shown by parents during a clean-up task at 18 months. Findings from other samples using the clean-up task suggest the protective role of parental warmth and support in mitigating the development of callous-unemotional behavior (Waller et al., 2017). However, prior

studies of this mitigating role used only biologically related families that could have been confounded by common gene effects. Specifically, we know there are genetic influences on parental warmth (Klahr & Burt, 2014; Neiderhiser et al., 2004). Thus, these genetic factors might be transmitted to the child and—gene by gene interaction—moderate the effects of genetic factors associated with callous unemotional traits. An adoption design can rule out this explanation.

Figure 21 shows the main results of this analysis. While adoptive mothers' positive reinforcing behavior at child age 18 months is inversely related to both child callous and deceitful behavior and oppositional behavior, severe birth mother antisocial behavior is associated uniquely with callous-unemotional behavior at 27 months. Considering the EGDS as an instant longitudinal design, this finding is the first of which we are aware that callous-unemotional behavior at 27 months may be a very early expression of the very same genetic factors that influence severe antisocial behavior in adults. Figure 21 also shows that the interaction of birth mothers' antisocial behavior and positive reinforcement by the adoptive mother uniquely predict callous unemotional behavior. Moreover, inspection of the interaction of birth mother antisocial behavior and adoptive mother parenting shows that birth mother antisocial behavior is positively correlated with child callous behavior only in the context of low positive reinforcement shown by rearing mothers.

In a follow-up analysis (Waller et al., 2016), we asked two additional questions about the early development of callous unemotional behavior. First, is it likely that antisocial behavior best indexes the genetic risk for the development of callous unemotional behavior, or might personality characteristics and temperament of the parent and child, respectively, also index the risk? Although multiple investigative teams have shown that early manifestations of disruptive behavior initially measured during the toddler/preschool period reliably predict severe forms of antisocial behavior during adolescence and adulthood (e.g., Moffitt, Caspi, Dickson, Silva, & Stanton, 1996; Moffitt, Caspi, Harrington, & Milne, 2002; Shaw, Hyde, & Brennan, 2012; Sitnick, Galan, & Shaw, 2019; Sitnick et al., 2017), antisocial behavior emerges more clearly as a distinct phenotype beginning in late childhood, adolescence, and adulthood. Measurement of dimensions of personality in the parent and temperament in the young child may also bring us closer to understanding the core psychobiological characteristics that parents transmit genetically to their children. Second, we can assess whether positive parenting can buffer the transmission of a challenging temperament from parent to child or the transformation of an adverse temperament from developing into maladaptive callous behavior. Two dimensions of temperament have been linked to the development of callous-unemotional behavior: fearlessness and low social affiliativeness (Waller et al., 2016).

Figure 22 presents these main findings. Biological mothers' fearlessness and low affiliativeness were associated with child callous-unemotional behavior; the path from birth mother fearlessness to callous unemotional behavior was mediated by observations of child fearlessness in the adopted child. As in the previous analysis, positive reinforcement by adoptive mothers mitigated the risk; this model showed that the effect of parenting was not on the transmission of the genetically influenced risk factor of fearlessness from mother

to child, but on the link between child fearlessness and subsequent callous unemotional behavior.

Taken together, these findings shed new light on the early development of callous-unemotional behavior. First, maternal positive reinforcement mitigates the development of callous-unemotional behavior in the early developing child. Second, it does so in part by blocking the development of fearless behavior into later callous behavior. It is possible that mothers who are capable of positive reinforcement with a fearless child do not see their child's fearlessness in a child as undesirable characteristic. Third, in some cases fearless behavior in a child may be an early manifestation of genetic risk for not only callous-unemotional behavior later in childhood but for severe antisocial behavior in later childhood and beyond. Fourth, both fearlessness and low affiliative behavior may index mechanisms by which parents' antisocial behavior is transmitted to their children. However, it should be acknowledged that we did not identify child characteristics that mediated the link between birth parent low affiliativeness and the children's callous-unemotional behavior.

Finally, it is not clear this same pattern of adoptive parent positive reinforcement buffering the progression of child fearlessness to later child callous-unemotional behavior would be evident in the context of lower SES contexts. For example, parents living in lower SES contexts may have less capacity to use positive reinforcement because of added stressors to their daily living or may prefer to use parenting strategies that are more effective in reducing the magnitude of association between fearlessness and callous-unemotional behavior. Differences in socialization practices also are influenced by children's sociocultural context, with similar parenting practices showing dissimilar child outcomes based on cultural norms. This pattern has been repeatedly demonstrated with respect to the association between the use of corporal punishment and children's conduct problems, where the magnitude of this association was reduced across six cultures where corporal punishment was more accepted (Lansford et al., 2005), This association was initially found in an American sample of Black and White children, with the relationship between parental use of physical discipline and child externalizing behaviors significant for White but Black families, within the context of physical discipline being more normative for Black relative to White parents (K. Deater-Deckard, Dodge, Bates, & Pettit, 1996; Lansford, Deater-Deckard, Dodge, Bates, & Pettit, 2004). Based on the influence of sociocultural context and the burgeoning focus on issues related to diversity, equity, and inclusion in the socialization of children (Aldana & Byrd, 2015; Phillips, Johnson, & Iruka, 2022), future research should consider the research question of how the sociocultural environment may moderate gene (i.e., child) by environment (i.e., parenting) in relation to child prosocial and problem behavior.

Bi-Directional and Reciprocal Effects.

The influence of parents and children on one another has been examined on a moment-by-moment basis, usually by direct observation in the home (e.g., Snyder & Patterson, 1995) or in a social interaction laboratory (e.g., Cohn & Tronick, 1988). For example, T. Field (1990) compared the contingent responses to one another of depressed mothers and their 3-month old infants; depressed mother-infant dyads show more contingent negative responses to one another but also more disengagement where responses by one are not soon followed by

responses from the other. Reciprocal or bi-directional effects have also been examined over longer periods of time where measures of child behavior in one year might predict parenting in a subsequent year, which in turn predict child behavior a year or two later (Jansen et al., 2017). For example, Jansen and her colleagues reported that fussy eating in children at 18 months predicted parental pressure to eat at age 4, that in turn predicted fussy eating at age 6. In general, analyses like these, across the span of months or years, use more global assessment of steps in a child's development rather than specific mechanisms of mutual influence. Jansen's data provide few clues about how fussy infants might induce mothers to pressure them to eat. Moment-by-moment interactions, on the other hand, provide clues to mechanisms by which parental characteristics influence child development or vice versa. For example, Snyder and Patterson (1995) observed a distinctive sequence for aggressive boys: a sequence of aggressive behaviors by the child was usually followed by aggressive parental behavior which was then terminated by the child's counter aggressive behavior. Thus, the child's aggressive behavior was reinforced by its ending an aversive circumstance, a definition of negative reinforcement.

Genetic analyses have rarely been applied to either moment-by-moment or longer-term bi-directional effects (for exceptions, see Kirby Deater-Deckard & Petrill, 2004; Tucker-Drob & Harden, 2012). Genetic differences among Snyder and Patterson's parent-child dyads might account for differences between those showing mutual and aggressive coercion patterns and those that did not. For example, there is ample evidence that dimensions of children's temperament, particularly negative emotionality, enhance their adverse response to harsh parenting (Slagt et al., 2016). This pattern is also true for a more recently characterized child characteristic termed "environmental sensitivity" (Slagt, Dubas, van Aken, Ellis, & Dekovic, 2018). There is evidence that these differences, depending on age and method of observation, are heritable (Assary, Zavos, Krapohl, Keers, & Pluess, 2020; Goldsmith & Gottesman, 1981). Likewise, there is emerging evidence that parental sensitivity to aversive child behavior may also show notable genetic influence. As noted, there is ample evidence that genetic factors influence hostile, angry, and conflictual parenting across childhood and adolescence (Klahr & Burt, 2014). Consistent with this pattern, a small sample twin study provided preliminary evidence that parental contingent response to their infant's crying may show genetic influence (Out, Pieper, Bakermans-Kranenburg, & van Ijzendoorn, 2010; Out, Pieper, Bakermans-Kranenburg, Zeskind, & van Ijzendoorn, 2010).

Bray et al. (2020): mother and child contingent behavior: the influences of birth mothers' affectivity and adoptive mothers' depressive symptoms.—Using the unique strengths of our adoption design, we re-examined the pattern of disengagement and non-contingent responding by both children and mothers that was described by T. Field (1990) and has been frequently replicated (for a review, see Goodman & Gotlib, 1999). We focused on sequential mother-child interaction in a 3-minute teaching task and coded positive and negative behaviors in three-second intervals when our children were 27 months old. We measured adoptive mother's depressive symptoms using the Beck Depression Inventory (Beck, Steer, & Garbin, 1988) and estimated some of the genetic risk for toddler negative responding by measuring negative affectivity in their birth mothers using the

Adult Temperament Questionnaire (Evans & Rothbart, 2007), using a summary score for scales measuring the parents' general self-characterizations as vulnerable to fear, sadness, anger, frustration, and discomfort. We found that as adoptive mothers' depressive symptoms increased, the mother-toddler interaction became less contingent (more disengaged), but only for those children whose birth mothers had higher scores on negative affectivity (Bray et al., 2020). These data suggest that, for toddlers, the pattern of transaction between depressed mothers and their children is a not a simple environmental pathway for transmitting maternal depressive symptoms to liability for their children's psychopathology. Rather, depressed mothers raising their biological offspring first transmit genes to their child that make them more likely to participate in a mutual process of disengagement by the time the child is a toddler. Maternal-child disengagement also may be adverse for the mother. Thus, she may be transmitting genes that not only contribute to the child's maladjustment, but through her child's behavior, leads to adverse influences on her own adjustment.

Roben et al. (2015): children's contingent negative responses to mothers' negative behaviors: the influences of birth mother and adoptive mother depressive symptoms.—We examined children's negative responses to maternal negative behavior more directly in another analysis (Roben et al., 2015). Still focusing on the toddler period, we measured birth mothers' and adoptive mothers' depressive symptoms using the Beck Depression Inventory, the former at child age 3–6 months and the latter at child age 9 and 18 months. At 18 months, we measured the frequency of contingent child negative responses to mother's negative parenting behaviors and, finally, mothers' depressive symptoms were measured once again at child age 27 months. We replicated the established association between maternal depressive symptoms and contingent child negativity (see T. Field, 1990; Goodman & Gotlib, 1999). In addition, we found that the higher were levels of child contingent negative responding at 18 months the more likely mothers were to increase their depressive symptoms from child age 18 to 27 months, *particularly when the birth mothers also reported elevated depressive symptoms*. We controlled for adoptive mothers' perception of child's temperament, particularly negative emotionality, to increase the likelihood that it was the child-parent interaction that had a sustained effect across 9 months (Roben et al., 2015). In other words, we demonstrated that the contingent negativity in the child's interaction with their mother at 18 months has an adverse effect on maternal well-being that is facilitated by the child's genes, a finding we could not demonstrate if mother and child were genetically related. That is, there must be a genetically influenced attribute of the children's response to their depressed mothers—or an unmeasured correlate of those child responses—that carries extra weight in worsening mother's depressive symptoms months later. Thus, in biologically-related families, mothers transmit genes to their children that increase their own liability for worsening depressive symptoms—likely through the child's response to mother's symptoms that were less severe before the child's aggravation of them. This *genetically influenced self-defeating feedback spiral* has, to our knowledge, not previously been reported.

Trentacosta et al. (2019): reciprocal influences of parental hostility and children's callous-unemotional behavior.—Does this mechanism generalize to other developmental sequences of parent-child interaction? We turn again to trajectories of

children's callous-unemotional behavior. We did not measure callous-unemotional behavior in rearing parents, but we did measure trajectories of their hostile and negative parenting, in this case using one of the most validated self-report measures (Arnold, 1993) at child ages 18, 27, and 54 months. Evidence of a genetically influenced self-defeating feedback spiral would consist of associations between adoptive mother's parenting and child's callous/unemotional behavior that, in turn, would lead to higher levels of mother's hostile parenting *but only in the presence of higher genetic risk for child callous-unemotional behavior.*

As noted, the features of low interpersonal affiliation and fearlessness are reliably associated with callous-unemotional traits in adults and can be used, as they were here, to index the child's genetic liability (Waller et al., 2016). To measure these traits in birth mothers we used the Behavioral Inhibition System (Carver & White, 1994) and the Harter Adult Self Perception Scale (Messer & Harter, 1986).

The findings for mothers and their children closely parallel those for depressive symptoms, as shown in Figure 24. For children at higher genetic risk, mothers' harsh parenting at 18 months was associated with a worsening of children's callous-unemotional behavior at 27 months. In turn, these children's behaviors were related to increases in mothers' hostile parenting behavior from 27 months to 54 months, but only for children at higher genetic risk. Again, in biologically related mother-child pairs, it is likely that mothers transmit genetic risk to children that heightens the likelihood that children's behavior will worsen their own parenting, another instance of a self-defeating feedback spiral.

Overall Summary of This Chapter

Adding genetic information to more conventional analyses of reciprocal effects between parent and child revealed genetically influenced negative feedback spirals not previously described. To grasp the significance of this finding, it is well to recall that the adoption design is splitting in two the genetic and the social mechanisms of transmission to improve our understanding of associations between parenting and child development. We are discriminating between two pathways that operate simultaneously in families where parents raise their own biological offspring. Thus, these data imply that biological parents transmit genetically to their children a characteristic that intensifies the child's impact on the very parental characteristics that had a prior adverse effect in the child. Unless there are effective repair processes in parent-child dyads engaged in these spirals, they could lead to especially malignant relationship processes, possibly to impairments in the mental health of both parent and child. If they remain continuously influenced by genes transmitted from parent to child, these developmental sequences will likely surface both in conventional studies of heritability and those studies using newer molecular methods such as polygenic risk scores or genome wide complex trait analysis , as genetic main effects (Yang, Lee, Goddard, & Visscher, 2011). However, as we see here, they are attributable to a blend of environmental and genetic mechanisms.

It is intriguing that we found evidence of these self-defeating feedback systems across all three different studies. The two studies focusing on depressive symptoms in the adoptive mothers used different measures for birth parents, in one a more general measure of affectivity and in another, a more focused measure of birth mother depressive symptoms.

Also, these two studies used different measures of contingency. In one, a measure of general behavioral contingency between mother and child that assessed the degree to which either a positive or negative behavior in one was immediately followed by a positive or negative behavior in the other. Thus, a *low* score implies *disengagement or at least, inactivity/passivity.* In the second study, we used a measure of child negative behavior contingent on a prior adoptive mother negative behavior; thus, a *high* score reflects *excessive sensitivity by the child to negative behavior in the mother.*

Our third study differed from the first two in three ways: the reciprocity was on an entirely different time scale, extending across many months. Second, the child behavior was callous-unemotional behavior. Third, the birth parent variable was two measures of temperament that we have already seen as risk variables for callous-unemotional behavior in toddlers. In all cases, we found that genetically influenced risk in the child enhanced the impact of their behavior on a rearing parent, suggesting a novel mechanism that should stimulate future investigation.

We can conceive of positive spirals of increasing competent behavior in mothers and children. Parents may transmit genes to their children that enhance their children's positive influence on just those characteristics in the parents that further enhance their children's competence and, potentially their own. A portion of this mechanism has already been reported in preschool children. Using a twin sample, Tucker-Drob and Harden reported that genetically influenced child cognitive abilities evoked parental cognitive stimulation from age 2 to age 4, whereas parental stimulation at age 2 led to early reading by environmental mechanisms (2012).

All five studies summarized in this chapter provide a more nuanced view of the interplay between child and parent. The study of reciprocity between parent hostility and child unemotional behavior showed, as previous studies have, that callous-unemotional behavior has an adverse impact on parenting. Yet, despite this consistent pattern, some parents can be positively reinforcing to their children, and, at least in early childhood, can temper the genetic risk to the child. This finding conveys a picture of a dynamic balance between child and parent, perhaps involving a reciprocal process we have not yet studied and underscores a need to understand more fully the factors that strengthen parents' positivity in the face of this child-initiated challenge. Although the current findings certainly need to be replicated among more ethnically/racially and socioeconomically diverse samples.

Chapter VII: Major Inference from our Findings: Sketching a New, Genetically Informed Process Model of Parent-Child Relationships

Chapters I-II reviewed the unique analytic opportunities of our adoption design and Chapters III – VI reported some of the major findings afforded by those opportunities. Here, we draw together some primary findings to present a conceptual model. Our goal is not only to provide a framework for synthesizing our findings thus far, but to propose a conceptual model and set of coherent hypotheses for our future research and work undertaken by others.

We asked: what the results of our adoption design add to understanding the child's impact on the family?

The contribution of our study rests on defining the role of the child using *measures of birth parents (BPs).* The correlation between a measure in *birth parents* and a measure of parenting by the *adoptive parents (AP)* strongly implies that the impact of child on parent is primary, a direct result of the child's attributes, and not secondary to the effects of prior parenting by the rearing parent. Likewise, the statistical interaction between a measure in BPs, on the one hand, and the impact of the APs on the child's development on the other hand, strongly implies that again it is a primary characteristic of the child that moderates the impact of parenting on them. Almost the entire corpus of literature on child effects cannot make this distinction between the primary and secondary effects of the child. We hope this additional leverage on child effects sheds new light on family interaction processes and narrows the gap between studies of family development and approaches to preventive interventions. We list here the main advantages of distinguishing between the primary and secondary effects and then sketch a new conceptual model that we hope guides future research on families and both informs and advances the development of preventive interventions.

Delineating more and less favorable responses of parents to intrinsic child characteristics.—As Chapters IV and V summarized, variation among children—as indexed by measures of birth parent characteristics—is associated with variation in parenting by the rearing parent and, subsequently, to children's development. This variation among children is indexed by the following birth parent variables: anxiety symptoms, social phobia, depressive symptoms, general psychopathology, sociability, agreeableness, behavioral activation, and emotional dysregulation (see overview in Chess & Thomas, 1999; e.g., Lebowitz, Marin, Martino, Shimshoni, & Silverman, 2019). Chapter V emphasized how effectively some parents responded to genetically influenced characteristics of the child; where parenting meets the needs implied in these child characteristics, favorable development followed. For example, we reported that those children at genetic risk for behavioral inhibition showed favorable development if their mothers were warmly responsive to their vocalizations (Natsuaki et al., 2013). In contrast, a child at genetic risk for being socially inhibited coupled with a less responsive mother showed less favorable development.

A primary goal of many parenting interventions is to guide parents to meet the challenges of a particular child more optimally. Our model focuses on how certain caregiving practices work well for some children, as defined by birth parent variables, and not so well with others. In some cases, what works best for some children is when parents *desist* from engaging in certain parenting behaviors, even if these behaviors previously have been found to be beneficial for many children. For example, as we reported in Chapter V, parenting may benefit a persistently inhibited child if the parent desists from being overly responsive (Brooker et al., 2016) or, in another instance, children who are already compliant with parenting requests may benefit from parents who do not typically provide proactive structuring in tasks they do together (Leve et al., 2022; Leve et al., 2009). As another example, we reported that children who have a birth mother with elevated

anxiety symptoms and could have a liability for anxiety symptoms themselves, show more behavioral inhibition when the father is responsive to them, an effect not seen with children of birth mothers with low anxiety symptoms (Brooker et al., 2016). In fact, for these children of birth mothers with low anxiety, higher levels of father responsivity were beneficial to the child. The statistical evidence for these anomalous effects of parenting are gene by environment interactions, where the slope for children with one genotype ascends upwards and the slope for children with the opposite kind of genotype slopes downward.

Delineating specific developmental pathways that are initiated by primary child characteristics.—The EGDS has identified several specific BP-->AP pathways to infer a profile of distinctive effects on AP parenting. Relative to previous socialization models that incorporate child effects (Bell, 1968, Sameroff & Chandler, 1975), the adoption research design allows us to go beyond the measurement of negative and aversive child attributes (e.g., negative emotionality, low inhibitory control, highly inhibited) which may reflect prior parenting. Not only has our study encompassed more nuanced, challenging, and positive attributes of the child based on BP attributes, it has attributed their effects to primary child characteristics. Using findings from EGDS, we can begin to sketch a genetically influenced “vocabulary” of child effects that are related to AP parenting.

Our results help to trace the antecedents of child and later-onset psychopathology.—Where the BP characteristic is a dimension of psychopathology and we have identified a child behavior as a mediating link between the BP’s psychopathology and the AP’s parenting, we might be obtaining an early indicator of a mechanism of pathogenesis that involves evocative effects (i.e., BP-->child behavior-->parenting-->child psychopathology). Such child effects have been previously described in socialization models of early-emerging conduct problems (e.g., Patterson, 1982; Shaw & Bell, 1993) without the benefit of delineating genetic pathways that might lead to these early acrimonious parent-child relationships and subsequent child psychopathology.

Reframing the gene-environment debate.—At a broader level, by focusing on genetically influenced child effects, we aspire to reframe the gene-environment debate. Rather than focus on the valence of children’s dispositions and contextual experiences, as exemplified by such perspectives as diathesis-stress, differential susceptibility, and vantage sensitivity, we emphasize advancing our understanding of the processes by which parents perceive the information in their child’s behavior and do or do not respond in ways that profit the child’s development. Our field has previously paid much attention to the patterning of children’s functioning in relation to different levels of biological and contextual strengths/challenges (i.e., how the slopes relate to one another), with the inference that understanding could be significantly buoyed by finding an underlying biomechanism that will explain stress sensitivity, differential sensitivity, or vantage sensitivity. However, rather than focusing on an underlying biomechanism, a search that may or may not have its own yield in the future, we hope to enlist family researchers to take more immediate advantage of genetically informed studies. We think our data help to yield a picture of how primary attributes of the child impact the family that is of immediate relevance to those seeking to advance research on family development and preventive interventions. Specifically, we encourage

family researchers and preventive interventionists to focus on the optimum fit between characteristics the child brings to the family and the family's response.

We now turn to describing our conceptual model, which has three steps. Following presentation of the model, we integrate results from Chapters III to VI into our conceptual framework.

Outline of a Genetically Informed Process Model of Parenting

1) Children's behavior can be conceived as containing information about their genetically influenced propensities that parents can detect.—We conceive of information in simple terms based on the early introduction of information theory into psychology (see, among many examples, Attneave, 1954). Suppose we, as observers, are asked to guess whether *birth* mothers have problems with impulsivity from self-reports of hostility by *rearing* mothers. For example, consider data we reported in Chapter IV (Sellers et al., 2020). We showed a modest correlation between measures of impulsivity and distractibility in birth mothers (BMs) with adoptive mothers' (AMs) hostility. This correlation was mediated by AMs report of child impulsivity. From these data, we can make this prediction and could do so at a better than chance rate, a simple example of information transmission. Borrowing from the elements of information theory, our ability to guess correctly suggests the maternal genotype reduced the uncertainty about causes (from our perspective as researchers) in the stream of the rearing parents' self-reported behavior. In this instance, we are simply inferring and reinterpreting an observed correlation between a BM's characteristic and a rearing mother's self-reported behavior towards her adopted child. However, thinking of an observed correlation in this way leads us to ask how this information is transmitted from BM's genotype to rearing mother's behavior. As we have underscored in previous chapters, our design provides strong evidence that the child is indeed the *channel* by which this information is transmitted. The information theory framework also leads us to ask where there is a specific genetic *signal* in the child's behavior to which rearing mothers respond with hostility. This same study of impulsivity in birth parents provides additional data that brackets one set of possibilities: AM's observation of impulsivity and activation of her adopted child. Our data suggest that these maternal observations of her child's behavior mediate some of the association between BMs' genotypes and rearing parents' self-reported parenting. However, mother's perceptions of her child's impulsivity and activation give us only an extremely broad field of possibilities into which to conduct a more detailed search. That is, what specific characteristics of a child serve as signals for these maternal reports, and is there anything specific about genetically influenced child impulsivity that distinguishes it from environmentally influenced impulsivity?

A final advantage of the information theory framework is a more precise delineation of the concept of "detection." When we speak of parents "detecting" a signal embedded in a child's behavior, we mean any form of discriminative response by the parent that we, as observers, can measure. Contemporary social neuroscience allows us to begin this search with indicators of brain responses to signals from the child (e.g., see a meta-analysis of event-related maternal cortical responses to infant emotions in parents and non-parents,

Kuzava et al., 2020) and extend across the range of responses that—at their most complex and self-aware—are the self-reported parenting reported in this monograph.

What is the genetic signal in the child that evokes a parental response? The first possibility is that *genetic information is embedded in some specific characteristics* of a child's behavior, that there is something distinctive about genetically influenced evocative behavior in the child. To return to our example, we know that there are both environmental and genetic influences on impulsivity in toddlers. However, might there be features of this mediating child behavior that are distinctively associated with just the genetic influences? For example, one of many mechanisms by which genetic "information" impacts parents might be *through a wide scope of influences on their impression of their child*. It is possible, for example, that some parent ratings of their child's impulsivity may reflect the parent's observations of its stability across long stretches of time. Parents may also be influenced by reports of the same behavior to the parent by their children's teachers and by their spouse. For some parents, their reactions to their child also may be influenced by their child's behavior arising at a very young age and/or the similarity of their child's behavior to others in the family. If some parents form impressions of their children from these diverse sources, they are duplicating what researchers do in seeking hints to the genetic origin of some behaviors that are consistent across time, appear early in development, are observed across contexts, and run in families.

Parents may or may not consciously attribute these patterns of behavior to genetic sources. Indeed, conscious genetic attribution may be entirely independent of the existence of specific genetic signals and reflect an attribute of the parent. We know from our own data that parents vary in these attributions. For example, adoptive parents who attribute their child's behavior to genetic causes report more daily hassles in their relationship with their children (Stover et al., 2015), perhaps because these beliefs about genetics reflect their feelings of helplessness to modify their children's behavior. In these same analyses, these genetic attributions were unrelated to child temperament or to adoptive parents' knowledge of the health of the birth parents, suggesting their origin is in the adoptive parent and not the child. Other candidates for specific genetic signals might be embedded in *some specific features of the child's behavior:* its intensity or its rhythm or unique relationship to the child's context.

Alternatively, it is plausible that a *specific "genetic signal" indistinguishably embedded in evocative child behavior* has both environmental and genetic influences. In this case, a parsimonious conclusion is that parental self-reported responses to their children may reflect differences in their children's temperaments. Genetic factors may increase the temperament's intensity rather than emerge as a distinctive behavioral feature. Information is transmitted from birth mothers' genotype to rearing mothers' parenting but along a channel that has other sources of information. The "signal" is hardly specific but rather embedded in a stream of the child's evocative behavior. From this perspective, children with birth parents high in impulsivity simply have more of their own impulsivity. APs are simply responding more to impulsivity in their child if that child's BM was more impulsive, a quantitative rather than a qualitative difference among children that reflects genetic differences among them. This broad alternative means that we, as researchers, typically may not be able

to detect what specific children's behaviors mediate the impact of any specific genetic influence on parental responses. However, as noted above, we should still be able to guess at better than chance levels whether a child's biological parent has high levels of impulsivity.

It remains for further research to determine to what extent these signals are consciously perceived by the rearing parents, regardless of whether they are interpreted by the adoptive parent as genetic in origin. In Chapter II, we noted that self-reported parenting is likely to index a parent's concept of their relationship with their children, and even of their view of themselves as parents. Accordingly, this perspective on parent self-reports underscores the "penetration" of the genetic information: it does not simply elicit reflexive behavior. Rather, a child's genetically influenced behavior is likely to influence the adoptive parents' mental state, even if the parent is unaware of the most salient, genetically influenced child evocative behaviors.

2) As noted, genetic information from birth parents can be detected by children's adoptive parents either by influencing their perceptions of their child (the BP variable being correlated with the AP's report of the child), or by influencing APs behavior towards their adopted child (BP variable correlated with AP parenting).—Chapter IV provided clues about the domains of behavior, as experienced by the parent or observed by us, that influenced these parental experiences and mental states. For example, we reported on the transmission of low social motivation from parents to children (Elam et al., 2014). In adults and in children, this trait is assessed by a person's tendency to withdraw from social contact and preference for solitary activities. We reported a notable association between the tendency for social withdrawal in birth parent and adopted child, evidence that this trait is genetically transmitted. Moreover, the child's social withdrawal evoked self-characterizations by rearing mothers of being angry and critical with their children if the child demonstrated elevated levels of social withdrawal. As noted in Chapter IV, the association of a birth mother's social withdrawal and a rearing mother's experience of herself as frequently angry was only partially accounted for by children's social withdrawal. Further, our measure of the child's social withdrawal, based on both observer and parental ratings, may not accurately index all the genetically influenced behaviors that mediate the association between birth mothers' social withdrawal and rearing mother's hostility. Chapter IV also reported, as noted above, that child impulsivity mediated the association between birth mother impulsivity problems and rearing mother hostility, with child's negative emotionality mediating the association between birth mother's externalizing and both rearing mothers' and rearing fathers' hostility.

There is extensive literature on factors that can narrow parental perceptions of their children's behavior in ways that induce harsh or ineffective parental responses. These include elevated levels of depressive symptoms (e.g., Arteche et al., 2011) and maternal substance abuse (e.g., Rutherford, Williams, Moy, Mayes, & Johns, 2011). Moreover, preventive interventions have been specifically directed at broadening parental perceptions to notice more positive behaviors (van den Boom, 1995) and to interpret what they observe in a more positive light (Bugental et al., 2002).

3) Some parental responses to genetic signals are active efforts to overcome signs of risk or promote favorable outcomes.—Correlations between BP characteristics and AP parenting is robust evidence that a genetic signal has activated a parental response towards the child. How can we, for example, understand the family processes that may underlie the degree of structuring an adopted child needs for optimal development if they have a genetic liability for a range of common psychiatric disorders? Perhaps it was just dumb luck. Some children with this liability happened to end up in adoptive families that could provide much needed structure. Or did some AP parents detect a tell-tale signal of this genetic liability to which they made optimum responses? As we do for the other two steps in our model, we provide more evidence in the next section. When we reconsider Step 3, we provide evidence that favors the view that some parents not only detect a genetic signal, but actively initiate and sustain optimal responses.

Fitting our Data to our Process Model of Genetically Mediated Child Effects.

Step 1. Genetically influenced behavior in the child transmits information from BM's genotype to rearing parents.—Data we have presented in previous chapters provided several examples, in addition to those just reviewed above, of how genetic information may become manifest in children's behavior early in their development. For example, the favorable genetic potential indexed by reward dependence in birth mothers, a measure of high social orientation, can be expressed as positive emotionality in toddlers at 18 months, based on correlations with birth mother reward dependence (Willems et al., 2016). Data reported in Chapter IV suggests that as response to this signal, a father dampens his hostility to his adopted child. We have begun to explore "signals" in the child's behavior of other positive attributes of birth parents' behavior. For example, the genetic potential indexed by birth mothers' high scores on executive functioning is expressed as high self-regulation by adopted children during toddlerhood (Cioffi, Griffin, et al., 2021) and persists into later childhood (Bridgett et al., 2018). Also, the genetic potential indexed by both BMs' and BFs' academic achievement is manifest in children's verbal ability by age 4.5 (Austerberry et al., 2021). Our analyses of parental responses to these "signals" are underway, but other investigators have shown, for example, that genetically influenced orientations to learning in preschool children influence their parents' expectations for their academic achievement (Briley, Harden, & Tucker-Drob, 2014).

In sum, our data indicate several possible signal systems: 1) BP impulsivity problems->adopted child impulsivity; 2) BP psychological symptoms-> child fussiness and negative emotionality; 3) BP social withdrawal -> child social withdrawal; 4) BP low social affiliation -> high child callous fearless behavior; 5) BP social orientation -> child positive temperament; 6) BP executive functioning-> child self-regulation and 7) BP academic achievement -> adopted child early verbal ability.

Of course, each of these seven classes of child behaviors have environmental influences and there may be nothing distinctive about those behaviors within each class that reflect their genetic influence. However, parents may respond to a *profile* of both environmental and genetically influenced signals in their children's behavior. We have identified seven of what must be many more connections between parental genotype and child behavior. For

any family, there are likely to be some child behavioral signals that have strong genetic influences while others may have much stronger environmental influences. Though any behavior may have undetectable differences between its genetic and environmental origin, differences among children in profiles of genetic and environmental influences may allow parents, at some level, to distinguish between the two.

Step 2. Parents detect genetic signals from their children, with awareness or not, and respond with conscious impressions of their adopted child and/or by their parenting.—We have presented two types of data to support this view. First, are correlations between BP variables and AP *perceptions of their child's behavior.* More important, however, are correlations between BP variables and AP *parenting.* As we have discussed, genetic signals that activate parenting have wider implications for the development of the parent-child relationship and the child's development. Our data suggest four modes by which parents respond to genetic signals as evident by their parenting.

The first is by *parental matching.* Where the signal is aversive, parents report hostility or over-reactivity in more challenging situations with their child. Many different genetically influenced child behaviors appear to have a final common path in self-reported negative parenting: these include the children's negative emotionality, impulsivity, and withdrawal, but not sadness. Where the signal is more engaging, such as positive emotionality, indexed by birth mother reward dependence, parents respond warmly to the child. Twin data (Tucker-Drob & Harden, 2012) suggest that parents also respond to early genetic signals of child intelligence. However, readers are reminded that in our data the coarseness of this apparent "common path"—warm supportive parenting versus hostile, over-reactive parenting — may reflect the coarseness of our own measures. Using twin data with older children and adolescents, Reiss and colleagues suggested a more nuanced response of parents to genetic signals from their children, including—for example—whether it was from daughters or sons (Reiss et al., 2000).

A second mode of responding to the signal is child-influenced *parental dampening.* We reported on this observed pattern in Chapter IV where a genetic asset in the child, indexed by birth parent reward dependence, was associated with reduced hostility directed to the child by the rearing father. This child characteristic serves to inhibit otherwise adverse effects that can influence harsh parenting of adoptive fathers, such as poor quality of their marriages and their own antisocial personality. These child effects might occur with or without the father's full awareness.

Third, as noted in Chapter IV, data suggest that biological parents transmit to their child characteristics that apply brakes to adverse family patterns, hence our term *self-correcting feedback spirals* (see Hajal et al., 2015). Preliminary evidence suggests that the pleasure scale on Toddler Behavior Questionnaire (Goldsmith, 1996) taps the genetically influenced characteristic that serves this braking function; items include parent reports of their child's delight and laughter in situations of achievement and positive social interaction (Willems et al., 2016).

A fourth mode of responding, an elaboration of parental matching, is the *self-defeating negative spiral:* biological parents transmit to their children a propensity for responding to them in ways that undermine the quality of their own parenting and, in some cases, also on their own mental health. We were able to delineate a detailed mechanism for at least one of these spirals. Adoptive mothers with elevated depressive symptoms have interactions with their children characterized by lower levels of contingent responses to their children's behavior; this pattern appears to be an environmentally mediated association. However, where the birth mother has a history of clinical depression, the adoptive mother herself is more likely to be adversely affected by this form of interacting with her toddler (Bray et al., 2020). In our data, this pattern shows up in two ways. In the first, higher levels of adoptive mother depressive symptoms were inversely related to her contingent response to her child, especially if the birth mother showed evidence of negative affect. In other words, a decline in contingent responding by the adoptive mother was a response to genetically influenced characteristics of the child. However, we could not identify the critical child characteristics underlying this association (see Bray et al., 2020). A second spiral involved child's contingent negative responding to mother's negative behaviors. As anticipated, higher levels of depressive symptoms in the adoptive mother were associated with greater child negative behavior contingent on mother's negative behavior. This association was environmentally mediated. However, only when BMs were high on depressive symptoms did this pattern lead to a subsequent increase in the rearing parent's depressive symptoms (see Roben et al., 2015). This pattern also appeared in findings we reported in Chapter VI. Callous-unemotional behaviors in toddlers predicted subsequent harsh parenting in their mothers only for children with BMs who scored high on factors of fearlessness and low affiliation.

We can translate these findings to families where birth parents rear their biological children. For depressive symptoms, the *environmental* effect of maternal depressive symptoms is an alteration of her own and her child's contingent behavior. The *genetic* effect of maternal depressive symptoms adds a component—yet unidentified—to this environmentally mediated impact on contingent responding that, as negative feedback, worsens mother's symptoms and her behavior towards her child. For callous-unemotional behavior, the same pattern holds. The environmental effect is the link between parental harsh behavior and an increase in the child's callous unemotional behavior, particularly from 27 to 54 months. Genetic influence adds something—again not yet identified—to the callous-unemotional behavior of the child such that it augments parental harshness. As with depressive symptoms, we saw this effect for mothers but not for fathers.

This unidentified additional contribution of genetic factors to the impact of child on parent is the most secure evidence we have seen so far in EGDS to suggest that there is a specific genetic signal embedded in the child's behavior. We know it is detected, consciously or not, because it has a clear consequence in maternal behavior and symptoms; it degrades them.

Step 3. Some parental responses to genetic signals seem to be active efforts to address signs of risk or promote favorable outcomes.—A clear example is our data on positive parenting and callous/fearless behavior. Our findings suggest a highly agentic parental response to a difficult child already displaying fearlessness and callousness.

A more stable and later-developing version of this cluster of child attributes assessed during middle childhood and early adolescence, termed callous/unemotional traits, uniquely predicts chronic and violent offending in adulthood (Pardini, Byrd, Hawes, & Docherty, 2018). Callous/fearless behavior is highly heritable in toddlers (Flom & Saudino, 2017) and is associated with reduced brain volume in critical areas by age 10 (e.g., Bolhuis et al., 2019). More importantly, twin studies suggest that a genetic component of callous/fearless behavior evokes negative parenting during toddlerhood (Flom, White, Ganiban, & Saudino, 2020). Although additional research is needed to demonstrate continuity between callous/fearless *behavior* in the toddler period and callous/fearless *traits* in later middle childhood and adolescence, it is likely that toddlers showing callous/fearless behavior are at risk for future problematic behavior. Moreover, of the many challenges to parenting, this one seems to arise from particularly well embedded biological characteristics of the child. Some parents can respond in ways that result in warm, reinforcing relationships with these children. How families accomplish this remarkable achievement, a testimony to their agency, is of great interest to prevention science. As our own short-term objective, we need to direct our own future analysis to the precise mechanisms by which some parents can counter these behaviors and others cannot.

As a second example, in Chapter IV, we presented evidence that mothers who are in high quality marriages showed little negative response to children at risk for externalizing problems (Fearon et al., 2015), “failing” to play their part in promoting a downward spiral of parent-child relationships as described in Patterson’s coercive cycle (1982). It is possible that these same mothers, embedded in high quality marriages, may also have provided more positive parenting to these same children, although we have no data yet to support this possibility. It, of course, remains possible that a random process had led some fortunate children with a liability for externalizing behavior to end up in homes with warm marriages. Consequently, they have mothers who are inclined to provide positive responses and suppress their own negative reactions. In other words, these data linking marriage, parenting, and child outcome alone cannot by themselves refute a simple null hypothesis that child and parent characteristics co-occur strictly by chance.

However, other findings suggest that this crucial balance between marriage and parenting is hardly fixed at the time of the child’s arrival in the family system. For example, we know from our findings that marital hostility is associated with *change* in parents’ hostile relationships with the child from 9 to 18 months (Rhoades et al., 2011). That is, a link between negative aspects of marriage and parenting unfolds in infancy and toddlerhood to form an additional challenge to vulnerable children. Thus, it seems unlikely that our results reflect children being assigned—by random or chance processes of conception or adoption—to a family with a fixed balance of marital and parenting process that was present before their arrival. To put the matter more positively, the evolution of a highly satisfying marriage in the early phase of family formation protects parents from engaging in coercive cycles with their young child; our data suggest that a more satisfying marriage clearly benefits children at higher risk for externalizing problems and serious psychopathology as adults. Some responses to the child may be similar to rapid reflexes; some of the sequences we described in Step 2 may be of this kind. Our data on marital satisfaction and favorable parenting serve as more evidence that genetic signals have been detected and responded to

with agency rather than automaticity; Indeed, these responses may reflect a more volitional co-construction of a satisfying marriage that supports a more positive response to those signals from the child.

These preliminary examples suggest two distinct levels of agentic response to genetic signals. The first is *countering* where a parent—for reasons we have yet to explore—provides favorable parenting to a child despite influences from the child that make such positive parental responses more difficult than usual. A second form of agency may reflect a *supportive family system* co-constructed first by the parents and then by the remainder of the family system.

Limitations of our three-step model

Limited focus on only part of our findings.—Our model has a limited focus on genetically influenced child effects and their impact on how children are parented and how parenting affects them. These data are novel and reflect the strengths of our adoption design. However, consistent with generations of research documenting associations between biological parents and their biological offspring, we corroborated many main effects between rearing parents' caregiving practices and child characteristics in early development, w*hile controlling for genes common to parents and offspring*. In many cases, our findings on direct parenting effects, primarily presented in Chapter III, were not surprising. For example, no head will be turned by our report that hostile parenting anticipates later aggressive behavior in children genetically unrelated to their parents (Stover et al., 2016).

Indeed, we replicated in our adoption sample a frequently observed *association between adoptive parental hostility and child aggression early in development*. As expected, because of the greater variability in child behavior during the "terrible twos" relative to the later preschool and early school-age periods, 27 months to 4.5 years was found to be a more sensitive *period of child's vulnerability* (see Chapter III, Stover et al., 2016). This finding did not require an adoption design to discover. Our data add an exclamation point to previous, comparable findings because our design rules out the confounding effect of genes common to parent and child. However, of note and more unique to our adoption design, associations between adoptive parent hostility and child aggression during the toddler and early preschool period were comparable in magnitude to genetic risk, as indexed by BM self-regulation abilities.

In addition, we found that the *impacts of mothers and fathers and children on each other formed a system of influences across time during early childhood.* One part of this transactional system was the distinctive influences of mothers and fathers on child development. Each played an independent role in contributing to child development while holding the influence of the other constant. This pattern held true for measures of parenting (see for example Chapter III, Bridgett et al., 2018) and parent psychological symptoms (see, for example Chapter III, Pemberton et al., 2010). Most important for understanding this system's influences may be our findings studying the influence of both depressive and anxiety symptoms across the family; the triad of mother, father, and child showed manifold influences on one another for both anxiety (Ahmadzadeh et al., 2019) and depressive symptoms (McAdams et al., 2015). Our adoption design helps us understand that these

child effects are unrelated to birth parents' anxiety and depressive symptoms, respectively. Again, few readers with knowledge of the early work on family systems (e.g., Ackerman & Sobel, 1950; Bateson et al., 1956; Bowen, 1960; Jackson, Riskin, & Satir, 1961), let alone continuing clinical work on family systems, will be surprised by these findings. What our study confirms is that many of these inter-relationships among family members can now be much more firmly attributed to environmental processes rather than genes that are shared by members of the same family.

Our findings on the distinctive influences on mothers and fathers emphasize the importance of involving fathers in preventive interventions. A trend in that direction is already well underway (e.g., P. A. Cowan, Cowan, Pruett, & Pruett, 2019; Lavner, Barton, & Beach, 2020; J. Y. Lee, Knauer, Lee, MacEachern, & Garfield, 2018; Pruett, Cowan, Cowan, Gillette, & Pruett, 2019). While mothers tend to spend more time providing caregiving to young children, even when both parents are employed full-time, our findings consistently suggest that fathers add distinct and independent variance to the prediction of multiple types of child behavior. While not always easy to accommodate because of their work demands and/or their openness to parenting-based prevention programs, the current findings emphasize that engaging fathers, along with mothers, in interventions holds much promise based on the comparable amount of variance to that of mothers found across our findings. As stated at several points, our adoption design confirms the importance of fathers' parenting as an environmentally mediated effect on child development. It should also be noted that the generalizability of our findings on the importance of paternal involvement in the socialization of children needs additional replication among samples of less economically and educationally resourced two-parent different- and same-sex parent families, including greater representation of families of color.

In addition, our measures of paternal caregiving were limited to constructs developed primarily for assessing maternal parenting (e.g., sensitivity, scaffolding). However, in the past few decades, more attention has been dedicated to exploring constructs based on unique contributions to socialization that fathers provide, focusing on such activities as encouraging children to engage in more challenging and risky behavior but also being careful to set limits to ensure the child physical safety. These parenting practices, distinctive for fathering, are now widely referred to as *activation parenting* (Feldman & Shaw, 2021). Future studies on paternal caregiving that incorporate measures of activation parenting might reveal even greater influence of fathers on children's social development.

As found in typical studies of biologically related parents and children (e.g., Emery, 1999; Emery, Fincham, & Cummings, 1992; Harold & Sellers, 2018) *we observed associations between marital quality and early child development, particularly the link between marital hostility and child anger*. Additional EGDS data also suggested links between marital quality and child sleep problems (Mannering et al., 2011). But our unique data allowed us to see these findings in the context of the child. For example, we observed a crucial role for a genetic characteristic of the child indexed by birth mother's level of anger and frustration. For children of birth mothers higher on anger and frustration, there was a clear prospective association between marital hostility of the rearing parents and measures of the adopted

child's anger and frustration. No significant association was evident where birth mothers were lower on anger and frustration (Rhoades et al., 2011).

Blanks in the model left by insufficient data.—These holes need to be filled in before our incipient model turns into a more developed and coherent set of hypotheses. For example, we do not yet have clear data on the processes by which parents detect genetic signals. We do not know whether factors that might affect parental detection influence their ability to perceive the signal or how they interpret the signal once perceived. Are these signals consciously perceived or are parents aware of them only after they have more automatically responded to them? Some of the mechanisms of signal detection might be discoverable if we had more observational data, particularly detailed sequential coding of parent and child behavior which, in a sample of our size, would incur enormous costs. Some of our analyses, many reported in this monograph, have benefitted from observational data of both child and parenting behavior, making it easier to delineate mechanisms as outlined in the studies of reciprocal parent-child interaction in Chapter VI.

However, we hope the outlines of our model, described here, might stimulate greater interest in companion studies to map the interrelationships of child genetic influences, observed child and parent-child behavior, and parents' self-characterization of their own parenting. The measurement problem extends beyond low correlations between observed and reported parenting. The same discrepancy between observation and parent reports also holds true for ratings of child temperament (see for example Seifer, 2003; Seifer, Sameroff, Barrett, & Krafchuk, 1994). A more complete map of how and why self-report may be related to observational measures can serve as a bridge to newer observational methods that can test the validity and utility of the model we have described here.

For example, the data we have reported in this monograph—outlined above—suggest that genetic variation among children provides a detectable signal to parents that provides clues to child needs and capabilities. Two new areas of research might be deployed in further testing of the model we propose here, but each depends on a clearer map linking observed and self-reported behavior. For example, a new direction in social neuroscience—as we noted earlier in this chapter—is to explore brain mechanisms involved in parents' responses to stimuli related to their children—such as brief exposure to photos or cries—(e.g., Rutherford, Graber, & Mayes, 2016; Rutherford, Maupin, Landi, Potenza, & Mayes, 2016; Rutherford et al., 2011) . These techniques allow measurements of parental brain responses to stimuli related to their children without relying on their self-report. Even more pertinent to testing part of our model are new paradigms for simultaneous brain scans of parent and child as they interact with one another to detect impersonal sequences of activation of brain circuits known as *cross-brain connectivity* (see a recent review Fulford et al., 2021; Ratliff et al., 2021; Russell & Gajos, 2020).

Finally, our model can be further extended and tested by intervention research that helps parents focus on recorded behavioral sequences with their children (e.g., van den Boom, 1995) or on the specifics of their behavioral response as provided by video feedback (see review in Bakermans-Kranenburg, Van Ijzendoorn, & Juffer, 2003). Studies of this kind will help define much more precisely what components of a child's behavior evokes parental

responses and, using interventions, how changes in what parents perceive may alter their parenting responses.

The limited focus on the child.—In effect, we have considered only a component of a potential and broader genetically informed model of family process. Our emphasis on parenting in the context of the child in this monograph is because the adoption design allows us to make causal inferences about this process, and because we can make the most secure inferences about child effects in our adoption design relative to other genetically informed designs.

However, genetically informed designs are poised to make significant contributions to understanding other aspects of the family system. For example, twin designs have clarified that evocative gene-environment correlations occur in marital couples: genetically influenced traits in one spouse can influence the marital satisfaction of the other partner (Spotts, Neiderhiser, Towers, et al., 2004). These effects are analogous to the genetically influenced child effects on which we have focused in this monograph. However, these influences play a relatively minor role in linking marital quality with the reported mental and physical health of spouses (Spotts, Neiderhiser, Ganiban, et al., 2004; Spotts, Neiderhiser, Towers, et al., 2004; Spotts et al., 2005; Whisman et al., 2018). In other words, the links between marital satisfaction and adult mental and self-reported physical health are mediated by environmental and not genetic factors. Indeed, marital process may be a major reason for divergent trajectories of adjustment between identical twins and between more ordinary siblings.

Genetically informed studies have also focused on child development in the context of the parent. Twin methods (Ahmadzadeh et al., 2021; Neiderhiser et al., 1999; Neiderhiser et al., 2007; Neiderhiser et al., 2004) and newer methods of molecular assessment (e.g., B. Wang et al., 2021) have contributed to understanding how genotypic differences influence differences in parenting.

Genetically informed studies have also clarified links between unsatisfying marriage and impaired parenting (Ganiban et al., 2007). For decades, this link, called the “spillover effect,” was attributed to environmental mechanisms. However, a notable proportion of these observed associations are genetically influenced. Genetically influenced characteristics in the parents play a significant role in both marital dissatisfaction and in negative parenting.

Finally, genetically informed studies have clarified the importance of sibling relationships in child and adolescent development. The apparent effects of siblings on one another, if they have at least one common biological parent, could be attributable to the genes they share. However, many genetically informed studies show that these major effects of siblings on one another are due to environmental mechanisms (McGue & Sharma, 1996; Reiss et al., 2000; Rende, Slomkowski, Lloyd-Richardson, & Niaura, 2005; Samek, McGue, Keyes, & Iacono, 2015; Slomkowski, Rende, Novak, Lloyd-Richardson, & Niaura, 2005).

In effect, the model we initially describe here is part of a growing empirical and theoretical effort to improve our understanding of family process and child development by taking

both social and genetic process into account. ” (high effects, due to chance, in a first report of findings from a relatively small sample), although they are hardly immune from this affliction.

First, these findings are drawn from a large, unbiased sample and many of our findings, particularly those in Chapter III on parenting effects, replicate previous findings. Thus, although small in magnitude, our findings are more likely to themselves be replicated with similar samples.

Second, as suggested by Funder and Ozer (2019), the magnitude of our findings can be evaluated in the context of other findings. To do so, Funder and Ozer recommend the simple correlation, *r,* as a useful indicator of the strength of a relationship rather than the square of *r*. The latter emphasizes what proportion of all the possible sources of variation on a criterion measure is accounted for by any specific predictor. In almost any assessment of this kind, some of the variation in the criterion variable will be due to unreducible error of measurement and some to random or individually specific circumstances of influence. Using r^2 as a metric of effect size will often lead to belittling an important finding because it accounts for only a fraction of this universe of influence. Using a simple correlation enables a different strategy: *the evaluation of effect size in context of other findings.* In our case, we can readily compare the standardized path coefficients in our Figures with meta-analytic *r*s drawn from relevant meta-analyses. The most useful are those by Pinquart. For example, he reported a meta-analysis of 256 published and unpublished studies of the prospective relationship of many different dimensions of parenting behavior with change in child aggression from an initial period of assessment to a subsequent one (Pinquart, 2017). All meta-analytic *r*s were less than .10. Pinquart obtained comparable meta-analytic *r*s for the association of many parenting dimensions and child academic achievement (Pinquart, 2016). Because adoption studies such as ours are rare, there are no meta-analysis of BP correlations with characteristics of the adopted child or with AP parenting.

Finally, and as noted earlier, all our assessments of genetic effects are *expected to be small* for three reasons. First, we are we are assessing genetic propensities in a child only from scores of their birth parents. This substantially attenuates estimates of genetic effects because genotype-phenotype correlations change across development and because by using a specific measure of birth parents we detect only the genetic influence associated with that measure. Second, many of our estimates use data only from birth mothers as birth father data was often not available. Third, adoption studies can only detect the additive effects of genetic influence rather than interactive effects. Twin studies circumvent all these limitations because estimates of genetic influence on any child trait reflect all those influences active at the time of assessment, parental data are unnecessary, and effects of interaction among genetic influences can be estimated. However, as we have noted, twin methods may over-estimate child evocative effects because we cannot readily rule out the confounding effect of parental knowledge of twin genetic similarity. Still, twin data may be a useful estimate of the upper bound of, for example, genetically influenced evocative effects. These effects are, of course, a central part of our analyses. Genetic modeling using twin data, however, does not readily produce a statistic comparable to *r;* it is more comparable to r^2. Klahr’s meta-analysis of twin studies (2014) suggests estimates from 23 to 40% of variance in

parenting may be accounted for by these genetic evocative effects. Thus, the small effects we report here are best thought of as efforts to detect specific, genetically influenced pathways of development within a broader, more influential nexus of gene-environment interplay in child and family development.

A model without specifications for sociocultural context.—Our model is built on data from a sample that, as far as possible, is representative of agency adopted children and their birth and rearing parents in the US. Because of expenses associated with seeking and obtaining a domestic adoption in the US, rearing parents will be drawn from upper income brackets that are primarily White. Thus, we have pointed out the limits on the generalizability of our findings and, consequently, of the model inducted from them. Within our sample we have generally controlled for child sex assigned at birth but not added it as an additional variable to our already complex modeling. Although of interest, we have too few same-gender marriages, but analyses we have conducted thus far have not shown meaningful difference between them and the rest of our sample. We hesitate to analyze our data either by race of child or race of rearing parents, reflecting concern that race is a label that might reflect a range of differences among individuals that are better attributed to differences in economic resources, environmental disadvantage, and prejudice (Noroña-Zhou & Bush, 2021). Still, even in its larval stages, our model can only be improved by a better understanding of the influence of many contextual variables.

Comparisons to Other Models of Parent-Child Relationships

As Chapter I underscored, theories of parent-child relationships extend back millennia. Moreover, there has been no shortage of newly developed models in the recent era of empirical family psychology. When more fully developed, where might our model fit into this very broad area of study? As a start to this reckoning, let’s consider three widely used and explored models in the current literature.

Initially inspired by the seminal work of Bell’s (1968) study of reciprocal effects, the closest line of work would seem to be the rapidly expanding investigation of how children’s temperament influences the impact of parenting on children’s own favorable and unfavorable development. In Chapter VI, we cited some of this work, particularly the detailed meta-analysis by Meike Slagt and her colleagues (2016). A review of 235 effect sizes across three broad areas of temperament produced generally disappointing results. For surgency and effortful control, there were no consistently moderating effects of temperament on parenting. For negative emotionality, there were modest effects in enhancing positive parenting on favorable outcomes and negative parenting on unfavorable outcomes, but only when measured during infancy.

Not surprisingly then, research in this area focuses on temperament characteristics that are more likely to moderate parenting effects. One approach is to define temperament not by the consistency of infant and child behavior across differing contexts, but by assessment of a child’s sensitivity and flexibility to changes in environmental context. A prime example is the dimension of sensory processing sensitivity first elaborated by Elaine and Arthur Aaron (1997). In children, this variable is measured by parent reports of children’s reactivity to a

range of stimuli. Recent work suggests that sensory process sensitivity may be a component or correlate of negative emotionality that is specifically linked to moderation of both positive and negative parenting and child outcomes (Slagt et al., 2018). Another more recent effort delineates a child characteristic poetically named the “dove” temperament, defined by the variation in the child’s curiosity and inhibition across several test situations. These newly conceptualized dimensions also appear to moderate parenting effects (Davies, Hentges, Coe, Parry, & Sturge-Apple, 2021). As a parallel, there is also a growing interest in this line of work in specifying neural systems that are especially “sensitive” to the environment. For example, preliminary studies suggest correlations between adult measures of sensory processing sensitivity and patterns in functional fMRI (Acevedo et al., 2014) and axonal structure, the latter revealed in diffusion tensor imaging (David et al., 2022).

This work, in general, has considered both the conventional and newer conceptualizations of “temperament” as an intrinsic characteristic of the child, although there is copious evidence for environmental effects on the traditional dimensions of temperament; the causal role of environmental variation on newer assessments is unknown. In contrast, our work thus far has been far less successful in identifying the specific characteristics of children that account for our findings on the moderation of parenting by either the child’s genetic liability or potential. However, we have been able to demonstrate that moderating characteristics are not only intrinsic to the child but that their moderating effect on parenting is likely causal. Although current investigations of the interaction of parenting and temperament seek to define the moderating feature of the child and discover neural correlates of these features, our model has focused on the impact of these characteristics on the family, how parents respond to them, and characteristics of parents that might determine their response. Our model will be strengthened if we can more clearly identify the genetically influenced characteristics of our children that have such clear moderating influences. Correspondingly, the investigation of the moderating role of child temperament would benefit from a better understanding of the processes that lead some parents to provide optimal environments for different kinds of children. In sum, investigation of child temperament as a moderator of parenting is increasingly focused on neural and psychological process within the child. As a complement, our model focuses on transactional processes within the family that account for moderation.

We turn to two other models of parent-child interaction, both with a broad focus: an understanding of family processes in which children are embedded and which determine their development. Both models have stimulated an unusually large amount of basic and clinical research, and each serves as a foundation of unique approaches to both prevention and treatment. The first of these is the well-known “coercion model” of Gerald Patterson and his colleagues at the Oregon Social Learning Center (1984). This model arose from detailed, in-home observation of *sequences* of interaction between parent and child. At the core of the model is the concept of contingency. To test the model, observation of child and parent behavior must be sufficiently precise to compute conditional probabilities. For example, if a child starts an aggressive or coercive exchange with a parent, what is the probability that — immediately following—the parent responds with a comparably aversive or aggressive response. Patterson observed that in families of school-age boys exhibiting antisocial behavior, the child often initiated disruptive behavior that was immediately followed by an

aversive parental response. Following that sequence, the parent—finding their own response ineffective—often terminated their response. Patterson reasoned that this termination was itself very reinforcing to the child. (See Patterson, 1984; Patterson, Reid, & Dishion, 1992). In effect, the parent allowed the child to "escape" from an aversive stimulus, an example of "escape avoidance learning" that is difficult to extinguish. Patterson's treatment and prevention approach focused on altering these moment-by-moment contingencies: helping the parent to respond to an aggressive child behavior with a response that not only terminated the child's aggression in the short run but had the long-term effect of modeling and teaching emotion regulation, such as a brief time out. As Patterson continued to develop his model, he recognized that some intrinsic characteristics of children accounted for why some children were prone to start aggressive interaction sequences with a parent while others were not. He allowed for the possibility that genetic differences among children might account for this contrast, focusing on genetically influenced individual differences in negative emotionality and inhibitory control (Patterson, 2002).

In two of its analyses to date, following up similar longitudinal work by Martin (1981) and Shaw and colleagues (1994; 1998) across the infant and toddler periods, EGDS has not been able to duplicate the precise observation of parent-child contingencies that lie at the heart of Patterson's social learning model. Moreover, our focus was not exclusively on aggressive and aversive exchanges. Yet, EGDS has demonstrated a range of genetically influenced child characteristics that, from a very early age, evoke aversive responses from parents. These go well beyond aggressive behavior in the child and include low social motivation (Elam et al., 2014) and impulsivity. Equally important, our data suggest genetically influenced child characteristics that may inhibit adverse parental responses, such as frequent expression of delight and laughter (Hajal et al., 2015; Willems et al., 2016). Thus, it is plausible that our data can amplify the coercion model in two ways: first, by suggesting additional parent-child cycles that may influence the developmental course of aggression in children. Some of these processes may forestall, rather than encourage, the initiation of coercive exchanges. Second, our model may help to clarify how genetic variation and social learning may be coupled in the development of coercive parent-child relationships. Our data do suggest, as Patterson suspected, that genetic variation among children might well account for why some children initiate coercive exchanges and others do not. In addition, our data suggest that social disengagement may be as potent as overt aggression in initiating these exchanges. However, a more precise fit between the two models depends on replicating Patterson's detailed observation of sequences of parent-child behavior within a genetically informed design to answer a fundamental question: does genetic variation influence the contingent, coercive responses of parent and child to each other?

We also can compare our model to a second and very broad line of investigation: the transmission of attachment security from parent to child. In 1985, Mary Main and her colleagues reported an astonishingly high correlation between maternal attachment security, as measured by the Adult Attachment Interview, and child attachment behavior in a maternal reunion setting (1985). Since then, investigation has focused on whether these findings are replicable and, if so, how this transmission might occur. Investigation has pursued the possibility that secure attachment somehow enhances a mother's sensitivity to the cues and needs of her child, a sensitivity that engenders in the child the growth of trust and

security in relationships. Data on the transmission of attachment security and insecurity had, by 2016, accumulated across at least 67 studies of the transmission of attachment, 17 of which contained measures of maternal sensitivity. This number of studies was sufficient to merit a comprehensive meta-analysis (Verhage et al., 2016). This review showed that the magnitude of association between mother's and child's attachment has tended to diminish in more recent studies but remains substantial, and that about half of this association can be attributed to sensitive parenting. An impressive extension of this work on the transmission of attachment is the formation of a consortium of attachment researchers organized to pool data and harmonize measures (Verhage et al., 2020). Pooling data allows greater power of analysis, and the first reports included, among other findings, a clear moderation of transmission by child age. Parent-child associations are higher in older children (Verhage et al., 2018). We will return to this specific finding below.

However, a central problem facing this line of research is that parental attachment status is not necessarily constant across development. One study in small sample of White middle class young people showed remarkable stability of attachment security across 20 years (Waters, Merrick, Treboux, Crowell, & Albersheim, 2000), while another disadvantaged and racially mixed sample showed almost none (Weinfield, Sroufe, & Egeland, 2000). Some data suggest that current life circumstances can shift the level of attachment security above and beyond the effects of a secure parent in early childhood. For example, a positive marriage has been associated with improvement in security of attachment in adults, as measured by the Adult Attachment Interview (Crowell, Treboux, & Waters, 2002), whereas stressful experiences of the college freshman year have been associated with insecure attachments, as measured by a questionnaire (Lopez & Gormley, 2002).

Throughout the course of this research, the role of genetics in the transmission of secure attachment has been dismissed by most attachment researchers largely on the grounds of twin studies of infants and toddlers (Bokhorst et al., 2003; Roisman & Fraley, 2008). However, some twin studies provided compelling evidence of genetic influence on attachment measures (Fearon, Shmueli-Goetz, Viding, Fonagy, & Plomin, 2013; Finkel & Matheny, 2012) but little evidence for the importance of shared environment. The latter is inconsistent with the impact of maternal attachment on children's attachment as maternal attachment status is the same for both twins.

EGDS did not study parent-child attachment using classical paradigms. Nonetheless, its findings might open new questions for attachment research. First, the relatively modest level of association between adult and child attachment, as seen in recent studies and confirmed by meta-analyses, means that there are substantial number of children of secure parents who are not securely attached and some children of insecure parents who are securely attached (Verhage et al., 2018). The new consortium is now pursuing "ecological constraints" on the transmission of attachment status. However, our data suggest that variation in genetically influenced child characteristics might also play a role and could be examined in twin studies or by using polygenic scores as an alternative to the labor-intensive adoption design.

Second, the relative instability of adult attachment has already been shown to be associated with current relationships and stresses of young adults. We could find no study of any

association between challenging child characteristics such as these and the discontinuity of secure attachment across time. A persistently challenging child can be very stressful for parents. Our study has shown that genetically influenced social disengagement, impulsiveness, negative emotionality, and anger can elicit self-reported hostility in parents. We have argued that these self-reports may reflect a broader conception by the parent of their relationship with their child. Whether such self-characterizations are connected to initial attachment security and stability of attachment to the parent is, our EGDS results suggest, worth investigating.

Finally, we have noted that a large meta-analysis confirms that the transmission of secure attachment from parent to child increases with child age. From a conventional perspective this transmission can be seen in environmental terms: the longer a child is exposed to a parent, the more complete is the transmission of attachment security from parent to child. However, by mid-adolescence, evidence is strong that environmental mechanisms of transmission are much less likely. Evidence comes from an adequately powered twin study using the state-of-the art child attachment interview (Fearon et al., 2013). As we have noted, this study showed a moderate genetic influence in adolescent attachment status but no evidence for the influence of environments shared by the twin siblings. If parental attachment status remains a significant influence in adolescent attachment, it would be manifest in a shared environmental effect as a parent's adult attachment status, by definition, should be the same for all siblings in the family.

Considering the role of genetic factors in the transmission of attachment security allows us to stress a very general point once again. Samples of adoptive parents and their children are not hard to recruit. In any large community, there are many families who would qualify for study. Moreover, in our experience, these parents feel relatively isolated by their relative older age and the lingering stigma of adoption. Many are happy to be involved in a study. The expense and effort of an adoption study such as ours is *linking* adoptive families with the biological parents of their children and collecting data from both sets of families. However, this linkage is unnecessary for many purposes. Including adoptive families routinely in family studies remains the most powerful way of verifying environmental mechanisms in the association of parental characteristics or parenting with child development. While molecular techniques are promising, they still capture only a small portion of genetic influences and may fail to capture some genetic similarities between parents and children, and thus overestimate environmental effects.

Overall Summary of This Chapter

Based on a range of our findings in early and middle childhood, we have proposed a new model of family process with three steps: 1) genetic variations in children that serve as signals to parents of their children's needs and potential; 2) the detection—consciously or unconsciously—of these signals by parents; and 3) factors that determine whether parental responses provide the child an optimal environment based on these signals. We have thereby recast our own findings on gene-environment correlation—which we have termed "child effects"—and gene by environment interaction into a theory of family process. By implication, we emphasize the value of a similar reframing of the very large number of

similar studies. Because our adoption design allows us to make strong causal inferences about the role of genetically influenced characteristics of children in the evolution of family relationships, our model is necessarily child centered. However, other efforts at integrating genetic information into newer theories of family dynamics are filling in other pieces of family process involving marital, parenting, and sibling effects. To explore the more general value of our fledgling model, we have considered its utility in three fields of current inquiry in development science: the interaction of child temperament and parenting on child development; the coercion model where behavior of children and parents reinforce a downward spiral of aggression and hostility; and the transmission of attachment security from parent to child.

Thus, our model has attempted to reframe basic genetic concepts into indicators of family process. We have also applied to reexamine other theories of parent-child relationships. Yet the value of our model may rest most heavily on its potential value for action research, particularly efforts to prevent the emergence of psychopathology capabilities and to enhance child strengths. We evaluate our model from that perspective in the next chapter.

Chapter VIII: Addressing Questions of Genetic Influence, Malleability, and Intervention

All developmental science is directed at three questions. What are the factors that shape human development, for better or for worse, across the life span? To what extent are these factors malleable and, hence, potentially responsive to intervention? And if malleable, can we develop interventions that have positive and lasting effects? Chapters III through VI have presented our contribution to addressing the first question. In Chapter III, we presented data on the role of parenting and of the parents' marriage and mood in the development of their children. There were few surprises here; however, our design eliminates the artifact of shared genes between parents and their children and makes inferences about parenting effects on child behavior more secure. Chapters IV, V, and VI began to address the question of malleability. Particularly in Chapters V and VI, we showed how intrinsic characteristics of children could moderate the effects of parenting and the reverse, how parenting could moderate the trajectory of child development even when those trajectories were influenced by genetic factors.

In this chapter, we take a last look at malleability through a window provided by an innovative extension of our adoption design, the inclusion of siblings of our adopted children who were reared by their biological mothers. This design affords unusual insight into the malleability of highly heritable child characteristics. For our first analyses, we have chosen measures of academic achievement. Results presented below confirm the malleability of academic achievement scores and provide a transition to our discussion of possible applications of the main results of EGDS to preventive and promotive interventions in early childhood.

One More Look at Malleability: Comparing the Child Placed for Adoption with Their Siblings Who Are Being Raised by That Child's Biological Mother

We have recently assessed the development of children of birth mothers in EGDS who were not placed for adoption; rather, these children were raised by their EGDS birth parent(s). Comparisons between these children and those placed for adoption provide a unique window into the developmental potential of children who grow up in families with different levels of economic resources: most adoptive families had higher education and economic security than most birth families at the time of placement. As in other adoption studies (e.g., Kendler, Turkheimer, Ohlsson, Sundquist, & Sundquist, 2015; McGue et al., 2007), birth parents in the EGDS sample who placed their children for adoption more often lived in far less favorable economic circumstances than did adoptive parents. However, some birth parents in our study did improve their circumstances through better education and employment opportunities since they were initially recruited to participate in EGDS.

Adding siblings reared by their birth mothers allows us to estimate the overall effect of adoption on many trajectories of child development by comparing parents and children in biologically related families to those in adoptive families. The contrast between the economic and educational circumstances between the two groups is large because parents who place their children for adoption are often younger than adoptive parents, with less time to develop economic stability and higher-paying jobs. For these and other reasons, they are hard pressed financially. In contrast, families that follow the arduous trail to successful adoption are typically advantaged economically and educationally. We found these expected differences in our sample. The median income of adoptive families was $119K, most were college educated, and 91% were married when we assessed their child at age 7 years. In contrast, birth mothers—on average—had a median education of a high school diploma at the time of assessment, a median household income of $14K, and 43.7% were married. This contrast provides unique leverage in evaluating the potential for growth and development in children born into financially stressed families, if they have a chance to grow up in better-resourced households, neighborhoods, schools, and communities.

Two kinds of informative comparisons are possible with this additional sample. All are directed at estimating the development of adopted children had they been reared by the birth parent(s). Being reared in an adoptive family might be regarded as an intervention, with the most straightforward test of its "efficacy" a comparison of means of how the child would have fared if reared by their birth parents with how they are doing at the time of measurement after growing up in a well-resourced adoptive family. One estimate of how the child might have done is the mean, on any score, of birth parents themselves. If the adoption has had a significant effect on the child, we would expect the child's mean score to be closer to the adoptive parents score than to the birth mothers. A second informative contrast is between the adopted children and the children that the birth mother is raising herself, the latter typically being a half sibling of the adopted child, and on certain occasions, a full sibling.

These comparisons, however, can be confounded if children are selectively placed for adoption or have, inadvertently, been recruited by us in a biased fashion. The principal concern is that adopted children in our sample may have a more favorable prenatal history

than do the children being raised by their biological mother. This outcome could happen in several ways. For example, families seeking to adopt a child may be hesitant to complete the adoption if they learn that the birth mother had severe obstetrical complications. Moreover, EGDS excluded infants with notable medical problems. However, detailed prepartum and peripartum data suggest that, in comparison to their biological siblings, the placed children have a somewhat *worse* obstetrical history (A. Burt et al., 2022). Thus, our estimates of the advantages to the child of being adopted will be very conservative.

An initial domain of child development we examined was academic achievement at age 7 years as measured by the Woodcock Johnson Test of Achievement. We selected this index of favorable development for three reasons. First, it is widely believed that academic achievement is highly heritable, and indeed heritability as high as 80% has been reported from twin studies of children (Schwabe, Janss, & van den Berg, 2017; S. O. Walker, Petrill, Spinath, & Plomin, 2004). However, despite widespread beliefs, high heritability reflects only the association of genetic differences with any phenotype for a particular population that is characterized by both specific risks and opportunities. It cannot estimate the developmental potential of children in adverse environments if they grow up in an environment with high opportunity and low risk. Second, academic achievement is a critical correlate of children's psychosocial development; low academic achievement follows from poor psychosocial adjustment and, in prospective studies, anticipates further declines in psychosocial development (see for example J. Liu et al., 2018; Mercer & DeRosier, 2008; Zhang, Zhang, Chen, Ji, & Deater-Deckard, 2019). Finally, as early as Burks' original study in 1927, the adoption method has shown promise in identifying how advantageous environments can substantially improve academic achievement. Most recently, Kendler et al. (2015) identified a large sample of pairs of Swedish full siblings and half siblings, where at least one sibling was raised by a biological parent and the other by an adoptive parent. Using military assessments of the siblings' IQ, Kendler and colleagues found a difference of up to six IQ points favoring adoption reared over biological reared siblings, a difference directly proportional to the difference in educational level between adoptive and biological parents. The EGDS was able to improve on Kendler's design in four ways: we could assess the prenatal history of biologically and adoption-reared children, we know the exact age of adoption of the children, we used the same measures for parents and children, and we have finer grained methods for assessing the environment.

Our analyses were based on 295 pairs of adopted children and birth mothers, 175 pairs of biological siblings and birth mothers, 139 pairs of adopted children and their siblings raised by their common birth mother, and 332 pairs of adopted children and their adoptive mothers. In addition to the Woodcock Johnson, we also estimated the literacy of the home environments of the biological and adoptive mothers, focusing on the number of newspaper and magazine subscriptions and children's books. Our main findings extended those of previous adoption studies. We found 1) that adopted children's reading scores were significantly greater than those of their biological mothers and that these differences were observed for children of birth mothers with both lower and higher reading scores; 2) adopted children scored considerably higher than their siblings reared by their birth mothers, and 3) measures of literacy indexed one of the advantages provided by being reared in adoptive families and the range of resources available to them. Further, unlike the Kendler study

that showed a notable correlation in education between biological and rearing parents, our sample's correlations were close to zero (see A. Burt et al., [2022]; Noroña-Zhou and Bush [2021] for details about these analyses). Hence, selective placement of the adopted child into an adoptive family similar in academic achievement to the biological family did not occur in our sample.

In sum, these initial results suggest that this new sub-sample of children raised by their biological parents will provide valuable insights into the developmental potential of children raised in difficult economic circumstances. It should also provide additional encouragement for promotive interventions that aim to increase favorable developmental lines that, in turn, may provide children with assets to promote their academic and psychosocial development. More specifically, these data should encourage intervention researchers not to shy away from developmental processes just because they have been repeatedly shown to be highly heritable. Moreover, this newer subsample of children allows a search for aspects of adoptive environment that may be important for its efficacy. However, limitations here also should be noted. For example, we used several simple measures to assess the "literacy" of the rearing environment. However, these measures serve only to bracket a potential area for further investigation. We need to know more about the family processes associated with these literacy indices, and why they may matter for children before they become actual targets of change for prevention research. Some of this unknown knowledge may be possible to obtain within the framework of the adoption design, but other research can be conducted with more conventional parent-child designs.

It is also quite likely that differences in psychosocial and academic functioning between biological parents and adoptive parents were not limited to the greater economic resources of the family environment, but also to the greater extra-familial resources provided by living in communities with better-resourced early learning centers and schools, and lesser exposure to deviant peers and adults that would be more prevalent in lower-SES communities, the latter also encompassing levels of exposure to community violence.

Addressing Questions About Preventive and Promotive Interventions

EGDS is designed as a basic research project in developmental science. In evaluating its relevance for practical interventions, we are guided by a particularly influential model focusing on the links between basic research and planned preventive or therapeutic interventions (Onken, Carroll, Shoham, Cuthbert, & Riddle, 2013). This "NIH Model" of intervention research emphasizes a recursive relationship between field trials of standardized interventions and the kind of basic, developmental research we report in this monograph. On occasion, basic research may yield seminal new insights that launch new avenues for early intervention to promote mental health. Perhaps more regularly, the techniques and strategies of basic research may be helpful in the cyclical process of improving interventions. First, findings from basic research may reinforce early lines of prevention research where initial prevention trials have produced promising but not conclusive results. Second, basic research may enhance understanding of why some children or families may not have favorable responses. Third, it may help simplify complex but successful interventions so that they are made more widely available at lower cost. Finally, it may help identify novel targets for

intervention with new understanding of their malleability and of their influence on children's development.

In this section, we first highlight EGDS findings we think may be closer to practical application. Second, we outline major themes in prevention and promotive research and suggest how our findings might address major questions with each theme. Third, we present an approach to the design of interventions and prevention trials that extends from EGDS findings.

Three EGDS Findings of Special Relevance for Prevention Research

Assessing child "needs" and parental responses.—As described in Chapters IV and V, we identified very early appearing behaviors we have labeled child effects both because they are intrinsic to the child and impact their parents' behavior towards them. Based mainly on data presented in Chapter IV, we have speculated that these child effects help illumine differences among children in what they need from a caretaking environment. These include needs for the self-regulation of attention, for the dampening of high negative emotionality, of relief from withdrawal from social interaction, and the risks associated with fearlessness and early appearing callousness. We have also identified genetically influenced child potential that may benefit from encouragement from parents. For example, we have shown that a positive and engaging temperament in the child, linked to birth parents' positive social orientation, can have a salutary effect on the family. Moreover, evidence of a high level of genetically influenced executive functioning and verbal ability appear very early in development. Most of these findings are hardly new, but the adoption design has clarified how early such patterns appear and how closely they are tied to either the child's genotype or—in one instance we cited in Chapter IV—to the child's prenatal environment (Liu et al., 2020). In practice, the genetic basis of these needs cannot be unambiguously defined without using special research designs such as ours or molecular assays whose limits we have already discussed. However, our data suggest new perspectives relevant to prevention, particularly in defining both risk and resilience in children who may be considered for intervention trials. For example, in evaluating the quality of parent-child relationships as both a risk factor and a potential target of intervention, our data suggest increased attention to the active role of the child in shaping those relationships.

Second, an expanded evaluation of both parents—especially if they are biologically related to the child—may provide clues to the child's status independent of the parents' current psychopathology or the strength and weaknesses of their parenting. Our data, for example, suggest that evaluation of child and parent temperament as well as child and parent executive functioning and intellectual and verbal achievement, may provide clues to both liabilities and strengths of the child. Specifically, our data suggest that a history of parental psychopathology may be as important as current psychopathology because it gives clues to genetic propensities in children and may help to define their needs for specific kinds of parenting from their mothers and fathers, such as highly structured parenting for children at genetic risk for psychopathology (Leve et al., 2009).

Understanding of the relationship of specific features of the caretaking environment, on the one hand, and the needs of children, on the other. —It is no surprise that parental warmth and responsiveness are desirable features of parental response to their children, especially during early childhood when children are highly psychologically and physically dependent on parents' caregiving. However, what is surprising is the severity of biogenetic risk that, for example, may be offset by parental positive reinforcement and warmth. For example, our data adds to other research (e.g., Henry et al., 2018; Humphreys et al., 2015) to encourage preventive interventions for early appearing callous/unemotional behavior. Also, EGDS has clarified that *desistance* from typically adaptive caretaking behaviors for most children may be highly beneficial for a well-defined minority of other children. These caregiving behaviors include desistence from consistent and contingent discipline for children at high risk for problems with executive function and desistance from high levels of paternal responsiveness for children at risk for anxiety symptoms.

The importance of including fathers in interventions with two parent families. —While we are hardly the first to report findings supporting the association between father's parenting and child development, we are among the first to demonstrate the importance of fathering independent of common gene effects (for other genetically-informed evidence for the importance of fathering see Class et al., 2012; Guimond et al., 2011; Reiss et al., 2000). Our data suggest that interventions that effectively target the parenting of just one parent might be less effective for families that have two residential parents. Relatedly, including only the residential parent in interventions for which the child spends considerable time at another parent's residence, might also be relatively ineffective without incorporating the second parent's involvement. In families with two residential caretakers, both are important not only in their separate contributions to child development but their impact on one another (e.g., via the marital relationship) and how that impact may moderate the trajectory of the child's development. Indeed, our data reinforce early efforts in the prevention field to actively involve fathers who may play a marginal role in their families and would ordinarily be challenging to include in preventive interventions (see Pruett et al., 2019; Pruett, Pruett, Cowan, & Cowan, 2017).

Possible Applications of EGDS Research: Four Themes from Existing Prevention Research

Ordinarily, when we evaluate the findings of a major research project, we ask two questions. First, did any of its findings raise central questions or provide new perspectives that, in turn, led to fundamental changes in how we think about human development? Second, did its findings have any immediate and practical significance? Specifically, did we generate new knowledge that could be utilized to directly benefit children and families above what was already known? As appealing as the difference between these two questions might be, they are really part of the same query. The second folds readily back into the first. No research project such as ours can be used immediately. At best, it can suggest modifications of existing practice. However, this suggestion is also a question for further research: When EGDS findings suggest modification in current practice, does that modification improve, in some specific way, the welfare of children and families?

To explore this utility, we ask three questions. (1) What are some of the central questions that prevention research currently faces? (2) How have these questions been addressed by interventions that have been rigorously evaluated? (3) Where might findings from EGDS suggest novel approaches to the design of interventions and prevention trials?

As noted in Chapter I, a strong interest in the prevention of behavioral and emotional difficulties in children and adolescents is over a century old (Levy, 1968). In the last four decades, well-defined interventions have been evaluated with rigorous, randomized controls and extended follow-up. To integrate our findings more fully with this broadening and deepening field of prevention research, we can, quite schematically, identify four themes in the rapidly evolving domain of randomized trials that serve as indicators, and provide information about interventions that have been rigorously evaluated

First, some of the earliest and highly successful prevention trials in this genre, for whom we now have convincing and very long-term follow-up, have important common features (see for example F. Campbell et al., 2014; F. Campbell & Ramey, 1994; Heckman et al., 2017; Olds et al., 1998). Specifically, the selection criteria for inclusion in the studies and interventions consistently included families at very high risk for a broad range of adverse outcomes for both adults and children, using such indicators as poverty and single teenage mothers. In addition, these preventive interventions started in very early childhood. Finally, recognizing the complex biosocial risks impinging on these families, *these interventions were both broad and sustained.* For example, the preventive intervention in Olds' Nurse-Family Partnership (Olds, Kitzman, et al., 2004; Olds et al., 2014; Olds et al., 2002; Olds, Robinson, et al., 2004) study started during pregnancy and extended through the first two years of the child's life, with up to sixty home visits by a registered nurse who attended equally to the medical and psychosocial needs of the mother and her child while broadening the young mothers' network of social support and effective contact with community agencies. Likewise, Campbell and Ramey's Abecedarian intervention (1994) began in infancy, used a specially developed preschool—rather than the home—as a focus of intervention, but provided not only intensive cognitive, motor, and social training but sustained support for parents in their involvement in school activities as well as effective contact with community agencies. Importantly, the school program—which was continued into the elementary school years—also was the site of good health care for the children in the experimental group. In effect, these early preventive interventions have altered the life course of children's development and, even when they become adults, the intervention had two-generation effects that benefitted their own children (see F. Campbell et al., 2014; Heckman et al., 2017; Heckman & Karapakula, 2019a, 2019b; Olds et al., 2014).

A second, more recent theme is interventions focused on risk mechanisms that have been clarified in well-designed longitudinal studies. Thus, *interventions have become briefer as they focus more sharply on specific, modifiable risk mechanisms*. In turn, these newer and briefer interventions have become more "scalable," more suited for evaluation by much larger samples, and more easily disseminated for practical use when successful trials have been completed (e.g., Prinz, Sanders, Shapiro, Whitaker, & Lutzker, 2009; Shaw et al., 2019; Slep, Heyman, Lorber, Baucom, & Linkh, 2020).

Third, there is a growing interest *in promoting favorable development by strengthening children's assets.* Strengthening assets of children at risk has been part of preventive efforts since the days of Jane Addams and Jacob Riis, and also from the outset of rigorous randomized trials (Daly, Nicholls, Aggarwal, & Sander, 2014). However, more recently, helping children achieve skills, abilities, and improved outlook has become a focus of preventive interventions, albeit with an increased focus on mediators and moderators of such outcomes. This shift reflects a growing understanding of how children's assets may give them broad protection from the ill effects of early adversity. Examples include preventive efforts to improve specific or multiple facets of school readiness, including efforts to promote self-regulation in children from low-income families (McCoy, Jones, Roy, & Raver, 2018) improve socioemotional development by promoting attachment security in maltreated children and children of depressed mothers (Toth, Gravener-Davis, Guild, & Cicchetti, 2013; Toth, Rogosch, Manly, & Cicchetti, 2006) and verbal ability in early childhood (see Salmon, O'Kearney, Reese, & Fortune, 2016).

Building on the body of intervention research that has focused on moderators of effects or specific populations that might benefit most from preventive interventions, a fourth and more recent trend has been the utilization of *genetic information in evaluating for whom interventions are most effective.* Multiple approaches use genetic information in prevention trials. First, concurrent with other efforts in studying interactions with single polymorphisms and the environment, various laboratory experiments and more sustained prevention trials added genotyping to their data collection (see a recent review, van Ijzendoorn & Bakermans-Kranenburg, 2015 and a critique, Dick, 2018). However, firm conclusions about the merits of genotyping to identify at risk children are unwarranted for four closely related reasons. First, in studies to date almost all genotyping has been conducted *post-hoc,* preventing rigorous tests of genetic effects through sample stratification before randomization and pre-registration of planned analyses. Second, many of the studies are seriously underpowered to detect interactions. Third, meta-analyses of these data cannot accurately estimate the effect of unreported analyses that failed to produce gene by intervention interactions when many of these analyses may have been done—but not reported—for each study. Fourth, GWAS studies, conducted after these early studies using single polymorphisms, are showing more convincingly that many genes influence complex outcomes, with each polymorphism having a miniscule effect. Thus, the rationale for selecting the specific polymorphisms in the single gene by intervention studies may no longer be valid, with some possible exceptions (see review by Byrd & Manuck, 2014 on MAOA x child maltreatment for antisocial behavior in males). Despite these major caveats, findings from these studies have suggested that polymorphisms ordinarily associated with risk for behavioral problems under adverse environmental circumstances may identify individuals who are most sensitive to the effects of interventions, a pattern that would be consistent with the theory of differential susceptibility (van Ijzendoorn & Bakermans-Kranenburg, 2015). In fact, some argue, based on these early single gene studies, that many children can be divided into those who are more responsive to environmental influences, including preventive interventions, and those who are less responsive (J. Belsky, 2014).

More recently, GWAS studies permit computation of polygenic scores (PGS), computed from data obtained from very large validation samples. PGS have now been incorporated,

but still *post hoc*, into prevention trials that have been more adequately powered to detect interactions between intervention and genotype (Kuo et al., 2019; Musci, Masyn, et al., 2015; Musci, Uhl, Maher, & Ialongo, 2015; Shaw et al., 2019). PGS have several advantages: they are usually derived from replicated and very large samples and they account for a more substantial proportion of variance in maladaptive outcomes in cross-sectional and longitudinal studies than do single nucleotide polymorphisms. Moreover, they generally have shown reassuring replicability. Although some results lend more credence to the DS hypothesis (Shaw et al., 2019) than others, PGS by intervention analyses show an attenuation of genetic risk in an intervention group, rather than identifying a group of genetically influenced unresponsive children, or the effects of a preventive intervention limited to only a low risk genetic group. (Kuo et al., 2019; Musci, Masyn, et al., 2015; Musci, Uhl, et al., 2015). Notably, Musci and colleagues, in their study of the prevention of smoking, unaccountably interpret their results as confirming the differential susceptibility hypothesis even though the most favorable results from their preventive intervention were found for those with *low* genetic risk according to a well-validated polygenic risk score for tobacco use.

Where Might Findings from EGDS Suggest Novel Approaches to the Design of Interventions and Prevention Trials?

Adoption designs would seem to offer exceptional opportunities for studying the interplay of genetic influences and intervention. They can do so in two ways. First, they are excellent model systems for anticipating how genetic factors might moderate intervention effects or which environmental factors might moderate genetic influences. Second, adoption designs can assess both prenatal and postnatal environmental influences. Adoption at conception designs (e.g., IVF studies) capture the first of these influences, such as intrapartum maternal depressive symptoms and adoption at birth designs, the second unbiased by genetic confounds (Leve et al., 2018). Third, adoption designs can test selective genetic effects that might moderate those influences. These designs can ask whether child characteristics associated with specific measurable characteristics of birth parents might moderate the effect of parenting by the rearing parents. For example, do measures of antisocial behavior in the birth parents moderate the impact of warm, responsive parenting on the adopted child? Findings on this issue could have real world implications for foster and adoptive parents where a child has been removed from the home because of witnessing and/or directly experiencing family violence and other forms of maltreatment by the biological parents.

The adoption design also allows an estimate of genetic influences that have life span significance because it identifies genetic effects that are common to adults (the biological parents) and children from infancy onward. For example, if academic achievement in birth parents is associated with verbal abilities in adopted children, during the toddler period we can assume that the genes influencing toddler language ability overlap with those influencing academic achievement in adulthood. Environmental moderation of these heritable characteristics may be particularly salient for early preventive interventions that aim to have a lasting effect on child, and eventually adolescent and adult, health.

Another use of twin or adoption designs is to directly test the efficacy of interventions. Surprisingly, there is almost no published use of these powerful designs for any kind of prevention or intervention research. Arnold Gesell pioneered the co-twin control method to study corrective and preventive interventions in very small samples of MZ twins. This design elegantly eliminates the effects of an intervention on genetic influences or environments that twins share. Thus, when the twin receiving the intervention shows gains in comparison to the untreated co-twin (Gesell, 1929), the main effect of the intervention can be convincingly demonstrated evidence of its causal effect independent of both genes and environments that twins may share. In principle, this approach is the experimental equivalent of comparisons between the development of children reared by biological parents with lower SES and those raised by more fortunate adoptive families. Unfortunately, this method has been used very sporadically, for example, to study the preventive effects of pre-menopausal hormone administration (Ahtiainen et al., 2012).

Intense, sustained, broad, multi-method interventions: EGDS can help the shift to more specific targets and briefer interventions.—As noted earlier in this chapter, many of the early and highly successful preventive intervention trials provided sustained and broad assistance to young children at risk. Since the inception of these interventions, several advances, uninformed by genetics, have supported less intense, broad, and sustained interventions that can be scaled up for widespread dissemination once efficacy and effectiveness have been established. EGDS can contribute to the process of refining, shortening, and focusing these interventions through three related processes buttressed by its unique design.

First, EGDS can identify *nodes in the network of risk and protective variables within the family system.* These are key processes that appear to be highly relevant to trajectories of child development because these nodes serve as a final common pathway of influence by: a) mediating a broad range of other factors that impact development and/or b) because they regularly moderate such influences. EGDS provides extra leverage in identifying several of these nodes because of its improved ability to study the impact of family environmental variables free of the effects of shared genes between parents and children.

One example is *marital quality.* Several prevention programs have already focused specifically on marital quality. These approaches draw heavily on clinical observations of family systems of Ackerman, Jackson, Bowen, Whitaker, and Minuchin as reviewed briefly in Chapter I. Their observations anticipated that a strong marriage would simultaneously enhance parent-child relationship and child and adult development. (C. P. Cowan et al., 2011). EGDS findings encourage extended focus on this key family process because of its importance as a main effect in early childhood and its role in moderating adverse genetic influences on child trajectories. Importantly, unlike the generations of studies showing that marital quality was related to child adjustment in nongenetically informed designs (see Emery, 1999; Jouriles, Bourg, & Farris, 1991), EGDS' findings consistently demonstrate the importance of marital quality on children's early adjustment, after accounting for some genetic contributions from biological parent to the adoptee, and with adoptive parents sharing no genetic affiliation with their children.

Another example is *negative emotionality*, a dimension of temperament that when measured during the first two years, is a consistent predictor of both externalizing and internalizing problem behavior during early and middle childhood (Emery, 1999; Jouriles, Murphy, et al., 1991; Sanson, Oberklaid, Pedlow, & Prior, 1991; Shaw, Owens, Vondra, Keenan, & Winslow, 1996). EGDS not only clarifies the role of negative emotionality in the evolution of psychopathology, but also its impact on executive function and language development. EGDS' data suggest that a critical feature of negative emotionality is its impact on family process, particularly adverse parenting (Liu et al., 2020). In important instances, EGDS' data suggest these child trajectories are moderated by parenting quality, an estimate not confounded by genes shared by rearing parents and children.

A third example is *affective symptoms in both fathers and mothers*. As summarized in Chapter III, internalizing symptoms in rearing parents—both anxiety and depressive symptoms—anticipate the development of internalizing and externalizing symptoms in their children. In many cases, these are statistically direct effects that are independent of comparable effects of the same measures in birth parents and of genes common to rearing parent and child. In other cases, these effects are amplified by various forms of genetic risk in children. Moreover, we have also shown that parenting mediates some of the effects of parental depressive symptoms and the potential effects of child characteristics on their rearing parents' affective symptoms. All these findings strongly emphasize parental affective problems as a third node in the array of our findings.

These nodes, as we have noted, provide additional encouragement for the design of preventive interventions. Alternately, they also suggest the selection of control groups. For example, the presence of affective symptoms in either mother or father may be highly prevalent in some groups of families at risk of psychological problems in their children. For these families, an exclusive focus on parental affective symptoms might be an effective preventive step for their children. A prevention trial could then address this question: how much more effective is a more complex intervention protocol, above and beyond the treatment of parental affective symptoms? Indeed, data do suggest that psychotherapy of maternal depression has salutary effects not only on mothers' symptoms but on their parenting and marital quality (Cuijpers et al., 2014). Further, reduction of maternal depressive symptoms may be a mechanism, among others, by which some forms of early childhood preventive interventions achieve their effect (e.g., Reuben, Shaw, Brennan, Dishion, & Wilson, 2015; F. L. Wang, Feldman, Lemery-Chalfant, Wilson, & Shaw, 2019).

Modelling potential environmental interventions.—Through its more precise estimates of environmental effects, importantly including the potential buffering roles of maternal *and* paternal caregiving and the marital or couple relationship (Bridgett et al., 2018; Marceau et al., 2019; Reuben et al., 2016), findings from EGDS can be used to identify targets and even timing of preventive interventions for specific combinations of parent and child attributes. While most of our findings confirm the wisdom of a broad scope of preventive intervention that aim at parenting and marital processes, EGDS samples a range of such parenting and couple relationship variables and has begun to examine the specific timing of their effects. For example, in this chapter we have mentioned a number of these family process factors, including the important role of both mothering and fathering. As

noted, of special importance to prevention is identifying subgroups of children who benefit from specific attributes of parenting. For example, we reported the highly specific effects of structured parenting on children with a broad liability for psychopathology (Leve et al., 2009) and the equally specific effect of desistance from child-centered fathering for children at genetic risk of anxiety disorders (Brooker et al., 2016).

Identification of signs of very early appearing manifestations of adverse genetic or prenatal influences.—EGDS can also provide insights into prevention targets by advancing our understanding of the manifestations of adverse genetic or prenatal influences early in childhood. For example, patterns of emotion dysregulation and anger expression in toddlers are an expression of genetic risk and can anticipate externalizing behavior four years later (C. Liu et al., 2018). Importantly, because EGDS can account for the independent contributions of genetic and prenatal influences independent of postnatal rearing environment in predicting emerging child adjustment, these findings can uniquely inform identification of targets for prevention and intervention programs.

EGDS findings: a brief note on promotive interventions.—Using the new extensions of our adoption design—the comparison of children raised by their biological mother with children who were placed for adoption by those same mothers—yielded clear evidence of the malleability of academic achievement. As already noted, heritability of a trait or a skill is a poor guide to its malleability, however, it is already well established that planned interventions in early childhood can significantly improve both cognitive skills and academic achievement (see a comprehensive review, Kautz, Heckman, Diris, Ter Weel, & Borghans, 2014). Our findings certainly reinforce such efforts. However, this new extension of our design may provide new information to inform the development of promotive interventions. For example, academic achievement reflects, in almost equal measure, IQ and favorable personality features such as conscientiousness, self-esteem and locus of control (Borghans, Golsteyn, Heckman, & Humphries, 2016). Our design has the potential for clarifying which components might be most affected by growing up in more favorable circumstances. Also, we can explore more fully what environmental differences between our two groups of children matter most for the salutary effects of adoption.

Overall Summary of This Chapter

As noted, complex, broad, and sustained preventive interventions—developed early in the history of the rigorous science of prevention science, anchor the field because high quality and long-term follow-ups of these interventions have shown sustained effects that have not only clinical implications but undergird important agendas for crafting major social policy initiatives and substantial investment in funding. However, based on more rigorous and long-term developmental studies—and major technical innovations in intervention research (such as motivational interviewing)—it has been possible to shorten the length of interventions so that they can be tested on much larger samples and, if found to be effective, make them more readily scalable for large scale dissemination. Several of these interventions have now published findings utilizing experimental designs with follow-ups 7 to 12 years later, encouraging the view that these long-term effects are reliable (Shaw et al., 2019; Zajac,

Raby, & Dozier, 2020). EGDS has much to contribute to further development of these promising interventions.

PGS—as already noted—have made a start in identifying differences in children that lead to successful outcomes. As noted above, genetic information helps identify a primary path leading from the child's genes to response to treatment being a property of the child. Thus, response to treatment can represent a child effect, although there are many other contributing causes to failure of children to respond to interventions. However, the initial use of polygenic scores in prevention research has focused on identifying children who do not respond to interventions. While the construction of the intervention may be solidly based on theory and prior evidence, it is likely not suitable for all children. Our data discourage the use of the term "dandelions" or its equivalent to describe children as relatively impervious to variations in their environment or to planned interventions. Rather, our data suggest a search for modifications of those interventions that are particularly suited to a genetically identified subgroup.

For example, EGDS showed that an important genetically influenced difference among children is their need for a structured environment in completing a clean-up task. Based on these differences, an unfolding process can be shown following from various combinations of children and environments. Children who fare the worst are those where the environment does not provide for their genetically influenced needs (Leve et al., 2009). Likewise, EGDS underscored genetic differences in children's capacities to evoke variations in environmental response but moderated by the environment. Thus, children at risk for externalizing behavior (a risk comparable conceptually to the polygenic score for aggression used in the Shaw et al. studies) evoke negative maternal responses only in families with low marital quality (Fearon et al., 2015). In sum, EGDS contributes hypotheses as to why some children may show failures to respond to what should be a salutary environmental change via a well-designed intervention. Thus, to carry this supposition further, children with higher genetic risk for aggression may benefit from a particular intervention because it provides the necessary structure in their home environment; children low on this score are not necessarily "dandelions"—unresponsive to interventions. Rather, they may thrive on parenting that provides positive reinforcement of their appropriate behaviors, rather than providing a high level of structuring.

In effect, a perspective growing from our research shifts the burden for successful intervention further from the child and more firmly onto the shoulders of the genetically informed preventive interventionist. That is, we steer clear of labeling some children as "dandelions" whose genetically influenced attributes make them unlikely to benefit from preventive interventions. Rather, our findings strengthen a drive towards redesigning genetically informed and more specific preventive interventions that match quite specific needs of groups of children and help families to effectively respond to those needs. More specifically, we can—for example—envision prevention trials with at least two different forms of preventive intervention and at least two different types of children whose are identified with the aid of genetic tools. Children and their families in each of the two groups would be randomly assigned to two specific interventions with the expectation that the intervention that is specifically tailored to the needs of children in one group would be more

effective than the alternate intervention. Although our own data are still far too preliminary to undergird such a design, we can imagine an intervention that helps fathers desist from child-centered care for children at risk for anxiety disorders. (A roughly similar intervention has already been designed for children with clinically significant anxiety symptoms, see Lebowitz et al., 2019.) We might also imagine an intervention that aided parents to provide more structure in task-related interactions with their children who have a broad liability for psychopathology. Our expectation would be that children and families in the anxiety prone group would fare better in this “desistance intervention,” whereas children in the broad psychopathology group would do better in the “structured parenting” intervention.

Though promising, caution is advisable. A design like the one we are proposing, called Project Match, was developed for adults with alcohol problems in the 1990s (Project MATCH Research Group, 1998). The design called for three different treatments, each thought to be particularly effective for subgroups of alcoholic patients. For example, where the alcoholic syndrome included a high level of anger, a motivational enhancement intervention was predicted to be most effective, whereas a form of cognitive behavior therapy was predicted to be more effective when alcohol problems were accompanied by co-morbid psychopathology. Almost none of the predictions of Project Match were obtained, almost certainly because of an incomplete understanding of both the matching variables and the mechanisms by which the different therapies achieved their effects. Our data help to map a path to a better outcome for this approach to interventions. We anticipate advances along two lines. First, genetically informed measures, in concert with other measures, may help to define groups of children with clear intrinsic needs: liabilities that need amelioration by specific processes within families and specific potentials that require enhancement by families, to prevent the development of problematic child outcomes. Second, precise family assessment, also improved through accounting for genetic effects, may help to define what psychological and social factors in the families of these children enable them to provide the protection and enhancement most responsive to their child’s needs. Our continued efforts, in concert with colleagues also using genetically informed research designs, may bring us closer to a successful matching design.

Chapter IX: Summary

Centuries of religious thought, simple observation, and more recent scientific research have underscored the importance of parenting in all phases of child and adolescent development. However, in recent decades there have been improvements in estimates of genetic influences on child development. This development has raised fundamental questions about which is more important: parents or genes? Efforts to discover a balance have passed through three stages.

First was the simplest: efforts to parse how much influence might be attributed to parenting on various measures of child development and how much to attribute to genetic influences.

A second phase recognized that this balance was not a zero-sum game. Indeed, in this monograph, we reviewed four major findings contributed by many investigators—before we started our own work—that yielded a more intricate picture. First were findings

that genetically influenced characteristics of the child could affect the parenting they received. Second, those genetic factors that evoked parenting responses were also those that influenced child development. Thus, many observed associations between parenting and child development could be partially attributed to this two-pronged effect of the same genes. Third were many instances where the links between parenting and child development could be partially explained by genes of the parent that partially shaped their parenting and when passed down to their children, partially guided their offspring's development. This finding also buttressed genetic explanations of observed associations between parenting and child development. Finally, we cited examples in previous research of how genetic factors in the child could alter the impact of parenting on the child's development.

Within this framework, we reported on many salient findings found in EGDS. For example, in Chapter III, when using our adoption design to control for the effects of genes common to parents and children, we confirmed previous findings of the relationship between parental hostility and child aggression. Further, we confirmed past findings that a child might be most sensitive to these effects between ages 2 and 3 during the "terrible twos." Beyond parent hostility, we found that hostility between parents themselves independently contributed to child aggression. We also extended previous findings supporting the intertwined influence of both mothers and fathers on child development. We also reported, in Chapter IV, many examples of genetically influenced child effects on the parenting a child received. For example, we reported on several different manifestations of how a child's genetic makeup evokes negative parenting: specifically via child negative emotionality, impulsivity, and withdrawal, but not sadness. Finally, in Chapter V, we explored extensively the role of genetically influenced child characteristics in moderating parent effects on child development. For example, we showed that children whose birth parents showed many symptoms of psychopathology (e.g., anxiety, depression) benefitted from structured parenting; the same kind of parenting had adverse effects on children whose birth parents had few or no symptoms of anxiety or depression. Likewise, parental responsiveness to the child reduced the likelihood of high child behavioral inhibition only in the context of having a birth mother with social phobia.

In this monograph we sought to transition to a third phase of research on parenting and genetics. We have aimed to go beyond parsing the role of genetic and environmental influences on child development and even beyond documenting the intricate roles that genes may play in accounting for observed associations between parenting and child development. We have taken elementary steps in integrating genetic and parenting data into a third stage of research, a process model of family interaction and development. To aid in this process, as we noted in Chapter I, we have set aside the statistical language—the "interactions" and the "correlations"—that has dominated research planning and thinking during the second phase of research. Then, beginning in Chapter VI, we used our genetically informative design to examine the unfolding reciprocal parent-child relationship over short and longer periods of time, as well as the relationship between the marital and parent-child subsystems. Using these findings, we have underscored processes such as *negative feedback spirals* where parents transmit genetic factors to their children, which not only make them more sensitive to adverse parenting, but reflexively further impair rearing parents' mental health and parenting. We have also provided a striking example of *parental countering* of adverse

child effects on parents by parents providing supportive caregiving that reduces the child's liability for psychopathology. Also in this domain is a clear example of the inter-relationship of marital satisfaction and parenting: parents in satisfying marriages respond less adversely to a child with a genetic liability for psychopathology. We have termed this phenomenon as *systemic support* to emphasize that this favorable parental response reflects relationship satisfaction in a wider social system. Importantly, this systemic effect clearly improved the outcomes for children despite genetic liabilities for psychopathology. These three concepts clearly move beyond the statistical mainstays to help us use genetic information to better understand the relationship between parents and their children and how this relationship may affect the development of both.

Our process model posits a genetically influenced signal system within the family. In our work, we focused on genetically influenced signals from the child, but according to other studies, siblings and spouses also originate comparable signals. Many aspects of the child's behavior serve as signals: their impulsivity, their negative emotionality, their social withdrawal, their callous-unemotional behavior, their fearlessness, their emotional self-regulation, and their early verbal ability. We know parents detect these signals by either their characterization of their children or by their behavioral or self-reported response to their children when either or both are correlated with birth parent characteristics. Just as there is a vocabulary of genetically influenced signals from the child, so too is there is a vocabulary of parental responses. For example, parents match their child's behavior and respond in kind, they dampen otherwise hostile responses to the child, or engage in positive or negative feedback spirals where their own responses amplify characteristics of their children and then, reflexively, their own.

Perhaps most importantly, parents differ in their response to signals from their children. A good example is parents who respond to children at low genetic risk for psychopathology by limiting the structuring of their parenting. Clearly, the next step for our research is to understand the circumstances that favor this optimal parental response. Correspondingly, some parents recognize the need for high structuring in parenting children with a broad liability for psychopathology. We can guess from our data that a satisfying marriage helps sustain that desistance, but we have not yet tied together our data on marriage and structured parenting.

We recognized limits in our model. It failed to encompass all the results we have presented, and it is in need of more data to verify its plausible but unproven hypotheses. It focuses on the active role of the child, whereas genetic data from other studies also examine the active role of marital partners and siblings in the family system. It is limited to the relatively privileged and mostly White sample on which it was based. Nonetheless, even in its early stages, it suggests revisions to existing perspectives on parent-child relationships. One example is the coercion theory, first developed by Patterson and colleagues. Our model suggests that genetic factors may account for differences in children's probability of initiating and possibly maintaining coercive exchanges with a parent. Further. it underscores other cycles that begin with genetically influenced social withdrawal or impulsivity and possibly play an important role in parent-child cycles leading to greater child withdrawal or aggression, respectively. For attachment theory, our model suggests that genetics

may play more of a role in the transmission of secure attachment from a parent to a child than attachment theorists have posited, particularly for older children. Further, genetically influenced differences among children in negative emotionality—or in other genetically influenced evocative characteristics—may impact parental attachment security and subsequent child development.

We also explored the relevance of our model for prevention, particularly its emphasis on intervention for parents in two-parent families and improving assessment during early childhood by providing greater emphasis on child characteristics that require specific types of parenting. Optimal parental response may include parental desistance from utilizing strategies that may be beneficial for some, but not all, children (e.g., structured parenting). Further, our data pointed to nodes in the network of influence in child development that might merit special attention in the design of prevention trials. These points of emphasis include focusing on negative emotionality in the child, parental depression and aggression, and a satisfying marriage. Published prevention trials are already focusing on parental psychological problems and, to a lesser extent, marital satisfaction. Our data suggests these foci might be combined with intervention directed specifically at child negative emotionality.

Appendix A: Tables

Table 1.

Unique features of the EGDS adoption design.

EGDS adoption design features	Behavior genetic terminology	Monograph terminology
EGDS children are raised by parents who are not biological relatives, and therefore the adopted children do not share segregating genes with their rearing parents	Passive gene-environment correlation	Effects of genes common to parents and children
In an adoption study, correlations between birth parent characteristics and adoptive parent/family characteristics reflect the impact of the child's genetic or prenatally acquired characteristics, operating directly on the adoptive family environment	Evocative gene-environment correlation	Child effects on parenting
Birth parent characteristics may increase some children's likelihood of a favorable or maladaptive outcome as a function of their rearing environment; in addition, some rearing environments may be beneficial to children, depending on their genetic characteristics.	Gene by environment interaction	Child effects moderate the influences of parenting on children's own development
In an adoption study, the prenatal environment is contributed by the birth mother, and is thus intertwined with genetic influences, rather than post-natal rearing environmental influences. Careful measurement of the prenatal environment can increase inferences about genetic influences	N/A	Prenatal environment
EGDS birth parents are assessed multiple times, from a few months post-partum to a decade or more later. This reduces measurement error in indices of genetic influences	N/A	N/A

Table 2.

Assumptions of the adoption design.

Adoption Design Assumption	Description	Measurement and Analytic Approach
Selective placement	Adoption agencies might systematically selectively place children into rearing families that are similar to the linked biological parents, or that reflect systematic efforts to counter the possible environment provided by biological parents with adoptive families unlike them	Measure key personality or contextual variables in birth and adoptive parents that are relatively stable and unlikely to change over time/due to environmental influences. Then examine birth parent-adoptive parent correlations. Significant correlations would be evidence of selective placement
Adoption openness	Birth parents might have contact with the adoptive parents or the adopted child. This contact could influence the behaviors of the adoptive family, causing similarities between the adopted child and their birth parents that are due to postnatal environments rather than to genetic or prenatal exposures	Repeated assessments of contact and openness from both adoptive and birth families, longitudinally. Then include a construct of adoption openness in analytic models to control for possible effects of adoption openness
Expectancy effects	Adoptive parents might gain knowledge of qualities of the birth parents, even if there is no direct contact, that can influence their expectations of the adopted child's behavior and characteristics. This could inflate estimates of genetic influences on child characteristics when adoptive parent report is used	Repeated assessments of knowledge from both adoptive and birth families, longitudinally. Include this aspect in the adoption openness construct. Other analytic strategies are using observational data and teacher-report data of child characteristics, and directly measuring adoptive parents' expectations and beliefs about the level of influence that genetics have on specific child characteristics
Research team bias	Research team members who have knowledge of both the adoptive and birth parents within a family could inadvertently influence the behaviors and expectations of research participants.	Eliminated this potential threat by using separate research team members to evaluate birth parents and to evaluate rearing families and the adopted child, within a given family.

Table 3.

The sample demographics by roles.

	Adoptive Mothers			Adoptive Fathers		
Variables	**Cohort I**	**Cohort II**	**Combined**	**Cohort I**	**Cohort II**	**Combined**
Mean age at the adopted child's birth ± *SD* (yrs)	37.8 ± 5.5	37.2 ± 6.0	37.6 ± 5.7	38.3 ± 5.8	37.6 ± 5.7	38.1 ± 5.7
Race(%)						
White	90.6	93.8	91.8	90.4	88.9	89.9
Black	3.9	3.3	3.7	4.7	5.8	5.1
Hispanic/ Latinx	2.8	1.0	2.1	2.2	2.1	2.2
Multiracial	1.1	0.5	0.9	1.1	1.1	1.1
Other[a]	1.7	1.4	1.6	1.6	2.1	1.8
Mean educational level at study start[b] ± *SD*	5.8 ± 1.3	6.0 ± 1.2	5.9 ± 1.3	5.6 ± 1.5	5.6 ± 1.4	5.6 ± 1.5

	Adoptive Mothers			Adoptive Fathers		
Variables	**Cohort I**	**Cohort II**	**Combined**	**Cohort I**	**Cohort II**	**Combined**
Less than a high school degree	0.0	0.0	0.0	0.0	1.2	0.4
GED degree	0.3	0.0	0.2	0.3	0.6	0.4
High school degree	11.5	10.3	11.0	18.2	13.4	16.6
Trade school	4.3	3.6	4.1	6.7	7.0	6.8
2-year college or university degree	6.6	4.6	5.9	3.5	3.5	3.5
4-year college or university degree	43.7	38.5	41.8	37.8	47.7	41.1
Graduate program	33.6	43.1	37.0	33.4	26.7	31.2
Mean educational level[b] last reported ± *SD*	5.9 ± 1.2	6.0 ± 1.2	6.0 ± 1.2	5.7 ± 1.5	5.7 ± 1.4	5.7 ± 1.4
Less than a high school degree	0.0	0.0	0.0	0.0	1.1	0.4
GED degree	1.1	0.0	0.7	0.6	0.6	0.6
High school degree	7.1	10.2	8.3	17.1	11.3	15.2
Trade school	4.6	2.9	3.9	4.2	4.5	4.3
2-year college or university degree	9.4	6.3	8.3	5.6	6.2	5.8
4-year college or university degree	41.0	38.3	40.0	34.0	46.3	38.1
Graduate program	36.8	42.2	38.8	38.5	29.9	35.6
Married at start of study (%)[c]	98.6	97.6	98.2	100.0	100.0	100.0
Married at last report (%)[c]	84.0	87.9	85.4	92.7	96.6	94.0
Median annual household income						

	Adoptive Mothers			Adoptive Fathers		
Variables	**Cohort I**	**Cohort II**	**Combined**	**Cohort I**	**Cohort II**	**Combined**
Study start	$70K~$100K[d]	$100K+[d]	$100K+[d]	$70K~$100K[d]	$100K+[d]	$100K+[d]
Last reported	$100K~$125K	$125K~$150K	$100K~$125K	$125K~$150K	$125K~$150K	$125K~$150K
	Birth Mother			Birth Father		
Variables	Cohort I	Cohort II	Combined	Cohort I	Cohort II	Combined
Mean age at the adopted child's birth ± *SD* (yrs)	24.1 ± 5.9	24.8 ± 6.3	24.3 ± 6.0	25.5 ± 7.2	27.0 ± 8.5	26.1 ± 7.8
Race(%)						
White	71.5	69.9	70.9	74.4	65.9	71.0
Black	12.0	16.8	13.7	8.8	17.6	12.4
Hispanic/ Latinx	6.7	5.1	6.1	8.8	8.2	8.6
Multiracial	4.7	4.6	4.7	4.8	5.9	5.2
Other[a]	5.0	3.6	4.5	3.2	2.4	2.9
Mean educational level at study start[b] ±*SD*	2.6 ± 1.3	2.6 ± 1.3	2.6 ± 1.3	2.6 ± 1.3	2.8 ± 1.3	2.7 ± 1.3
Less than a high school degree	25.7	30.1	27.2	26.4	20.0	23.8
GED degree	14.4	12.9	13.9	16.0	12.0	14.4
High school degree	41.8	39.8	41.1	41.5	49.3	44.8
Trade school	10.7	9.7	10.4	10.4	12.0	11.0
2-year college or university degree	3.4	2.7	3.1	1.9	0.0	1.1
4-year college or university degree	3.7	4.8	4.1	1.9	5.3	3.3
Graduate program	0.3	0.0	0.2	1.9	1.3	1.7
Mean educational level[b] last reported ± *SD*	3.6 ± 1.6	3.1 ± 1.5	3.4 ± 1.6	3.3 ± 1.4	2.8 ± 1.4	3.1 ± 1.4
Less than a high school degree	9.3	18.8	12.6	6.0	23.3	12.6

	Adoptive Mothers			Adoptive Fathers		
Variables	**Cohort I**	**Cohort II**	**Combined**	**Cohort I**	**Cohort II**	**Combined**
GED degree	13.5	13.3	13.4	20.5	12.3	17.4
High school degree	31.5	40.9	34.8	41.0	42.5	41.6
Trade school	15.9	6.1	12.5	12.0	12.3	12.1
2-year college or university degree	14.7	11.0	13.4	12.0	2.7	8.4
4-year college or university degree	10.8	8.8	10.1	6.8	5.5	6.3
Graduate program	4.2	1.1	3.1	1.7	1.4	1.6
Married at start of study (%)[c]	7.0	4.6	6.2	19.1	7.2	14.0
Married at last report (%)[c]	47.3	46.0	46.8	53.4	48.8	51.5
Median annual household income						
Study start	<$15K	$15K˜$25K	<$15K	$15K˜$25K	$15K˜$25K	$15K˜$25K
Last reported	$25K˜$40K	$25K˜$40K	$25K˜$40K	$25K˜$40K	$25K˜$40K	$25K˜$40K

	Target Child		
Variables	Cohort I	Cohort II	Combined
Race (%)[c]			
White	56.8	50.5	54.5
Black	11.4	16.5	13.2
Hispanic/ Latinx	12.2	15.5	13.4
Multiracial	18.8	16.0	17.8
Other[a]	0.9	1.5	1.1

Note:

[a] Includes Asian, Native Hawaiian/Pacific Islander, American Indian/Alaskan Native, and Unknown.

[b] Mean education level is calculated with a 7-point scale ranging from 1 (<high school degree), 2 (GED), 3 (high school degree). 4 (trade school). 5 (2-year college).6 (4-year college). to 7 (graduate progran).

[c] Includes marraige and living together in a committed marriage-like relationship.

[d] Statistically significant difference between cohorts at $p < .01$.

[*] Race of the child was reported by adoptive parents.

Table 4.

Measures.

		Infancy & Toddlerhood				Childhood				Pre and Early Adolescence	
	Constructs	**5 mos**	**9 mos**	**18 mos**	**27 mos**	**4.5 yrs**	**6 yrs**	**7 years**	**8 yrs**	**9 yrs**	**11 yrs**
Genetic influences	Psychopathology (symptoms and diagnosis)	●		●		●					●
	Drug and alcohol	●		●							●
	Delinquency and criminal behaviors	●		●		●		●			●
	Temperament & personality	●		●							●
	Executive functioning & Intelligence	●		●		●		●			
	DNA	●				●		●			
	HPA regulation					●					
	Physical health	●						●		●	
	Health-related behaviors (e.g., eating, sleep, physical activities, risky sexual behaviors)	●		●		●		●		●	
	Well-being (life satisfaction)	●		●		●		●			●
	Height & weight	●		●		●		●		●	●
	Sociodemographic information	●		●		●					●
Prenatal environment	Prenatal drug use	●									
	Prenatal complications	●									
	Prenatal anxiety and depression	●									
Rearing environmental influences	Psychopathology		●		●	●	●	●	●	●	●
	Drug and alcohol use		●	●		●	●	●	●		
	Delinquency and criminal behavior		●								
	Executive functioning & Intelligence					●	●	●			
	DNA			●			●				
	Temperament and personality		●	●		●		●			●
	Physical Health		●					●		●	
	Well-being		●		●	●	●	●			
	Health-related behaviors (e.g.,							●		●	

		Infancy & Toddlerhood				Childhood				Pre and Early Adolescence	
	Constructs	**5 mos**	**9 mos**	**18 mos**	**27 mos**	**4.5 yrs**	**6 yrs**	**7 years**	**8 yrs**	**9 yrs**	**11 yrs**
	eating, sleep, physical activities)										
	Parenting		●	●	●	●	●	●	●	●	●
	Couple relationships		●	●	●	●	●	●	●	●	●
	Resources (e.g., financial stress, income, home chaos, health insurance coverage)		●	●	●	●	●	●	●	●	●
	Sociodemographic information		●	●	●	●	●	●	●	●	●
	School and teacher						●	●	●		●
	Language and literacy				●	●	●	●			
	Executive functioning & academic achievement				●	●	●	●			
	Temperament and personality		●	●	●	●	●	●	●		●
	Peer relationships and social skills				●	●	●	●	●		●
Child characteristics	Relationships with school and teachers						●	●	●		●
	Psychopathology			●	●	●	●	●	●	●	●
	HPA regulation					●	●	●			
	Physical health		●	●	●	●	●	●	●	●	●
	Health-related behaviors (e.g., eating, sleeping, physical activity)		●	●	●	●	●	●	●	●	●
	Height and weight		●	●	●	●	●	●	●	●	●
Confounds	Openness to adoption	●	●	●	●	●	●	●	●		●
	Adoption process	●	●	●	●						

Appendix B: Figures

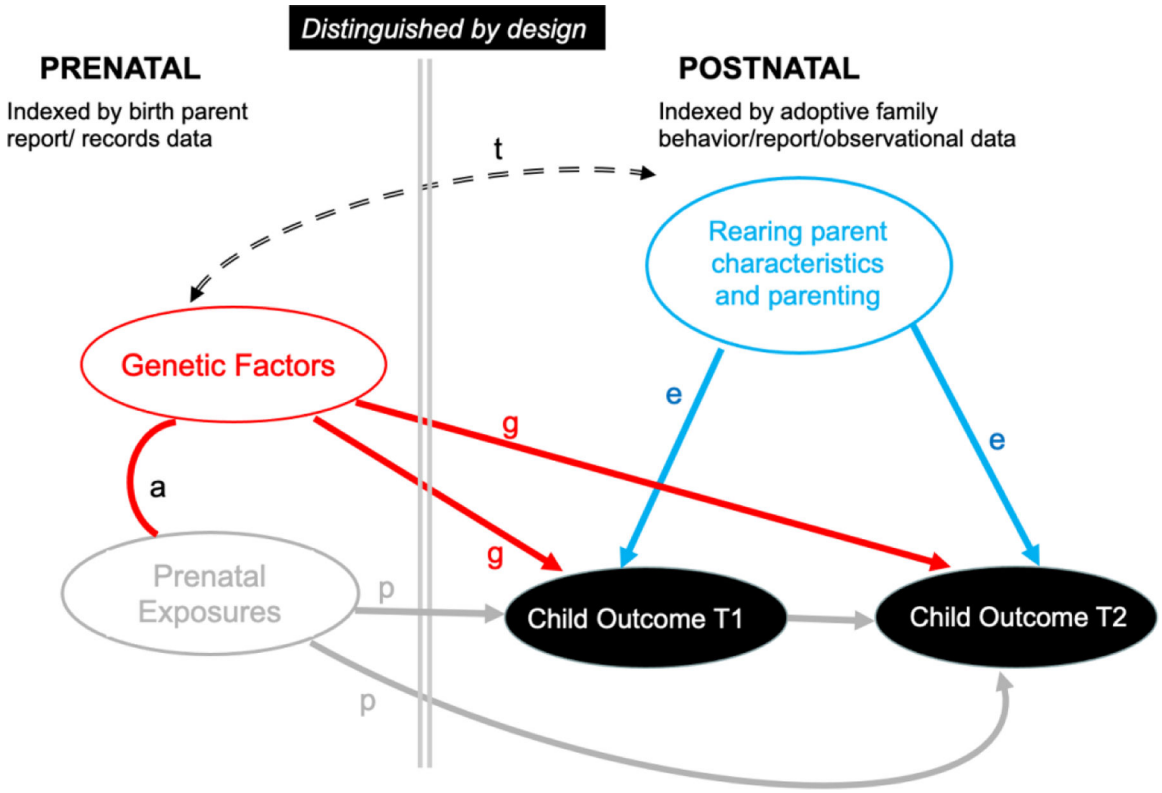

Figure 1A.

Conceptual illustration of a parent-offspring study that incorporates both birth parent and adoptive parent participants

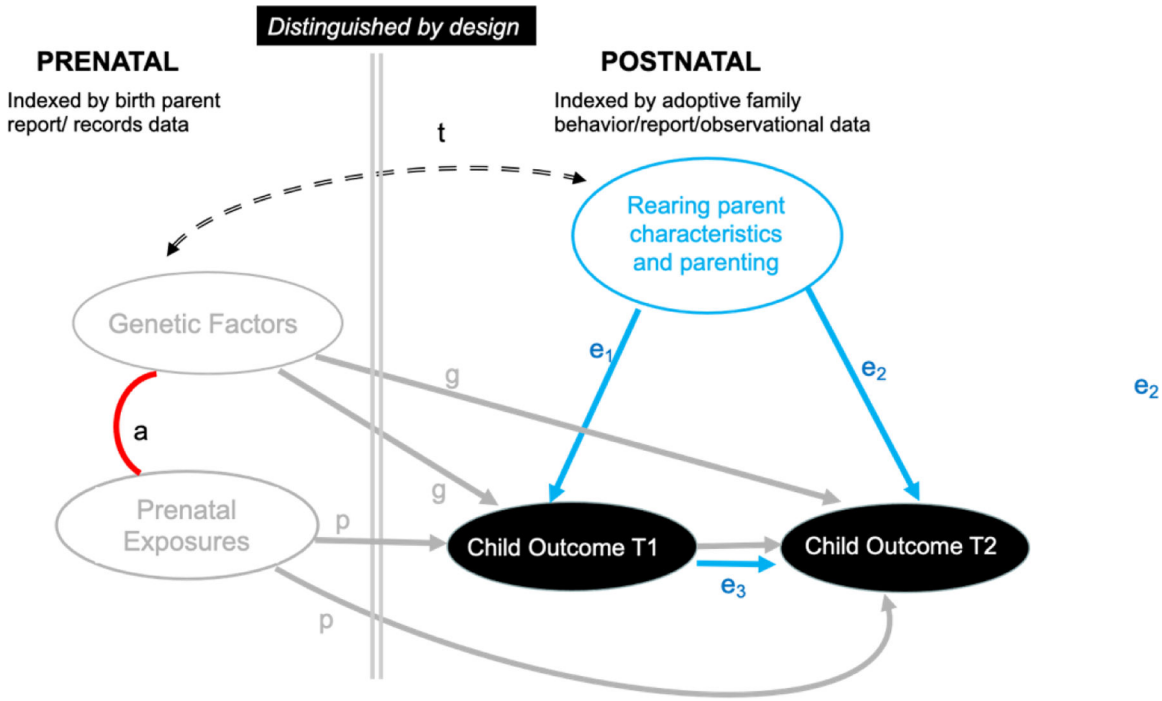

Figure 1B.
Conceptual illustration of a parent-offspring study with genetically related participants only

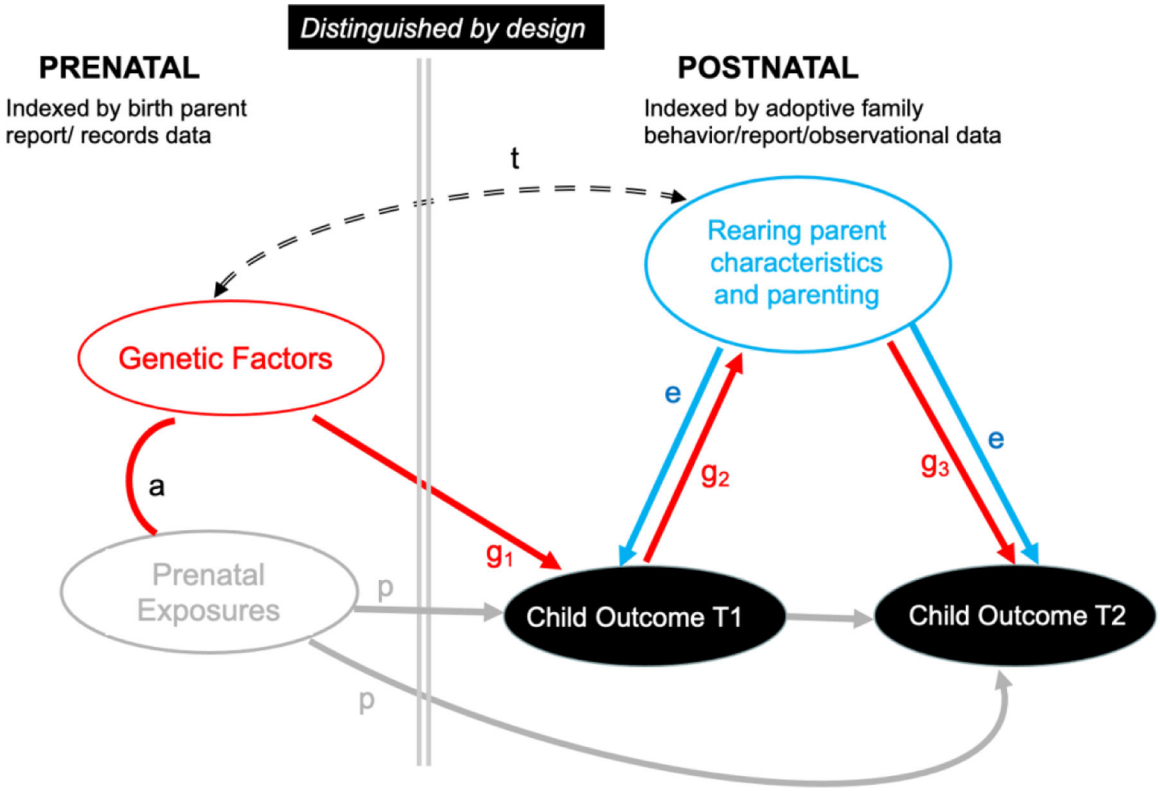

Figure 1C.
Conceptual illustration of the evocative pathway from birth parent to child to rearing parents made possible with the adoption design

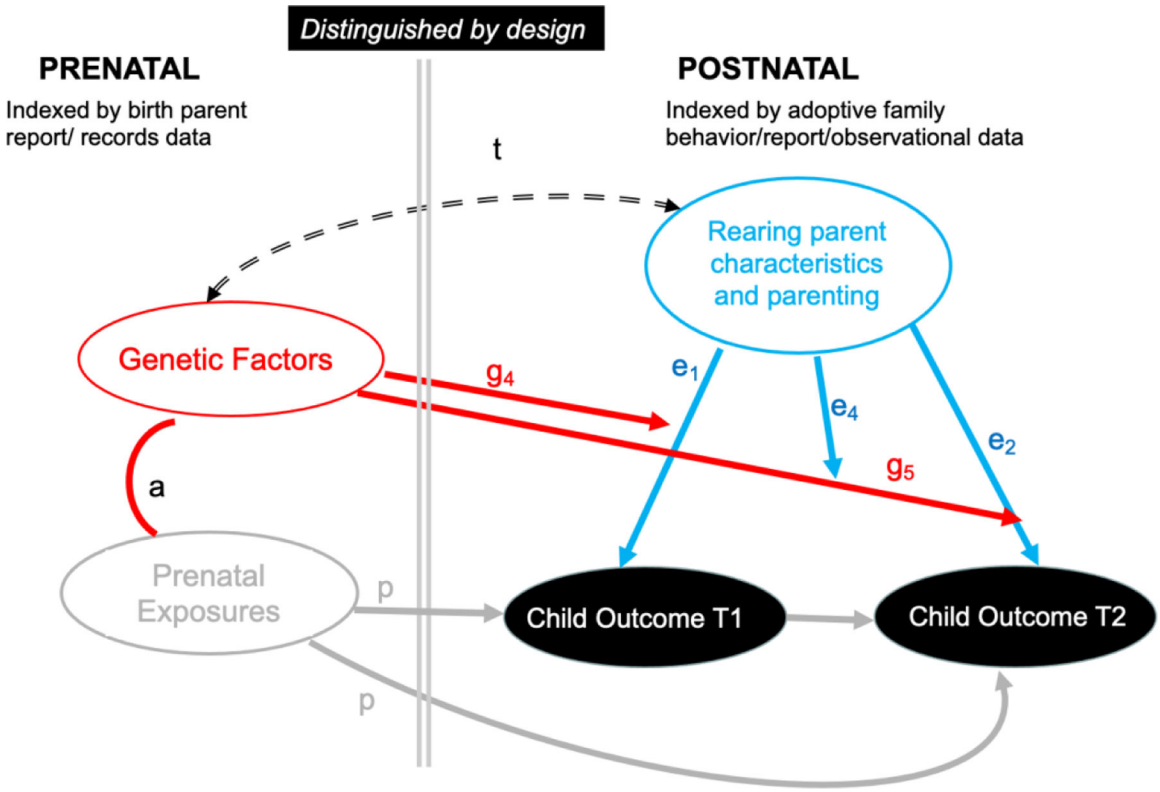

Figure 1D.
Conceptual illustration of genetic moderation of environmental pathways in the adoption design

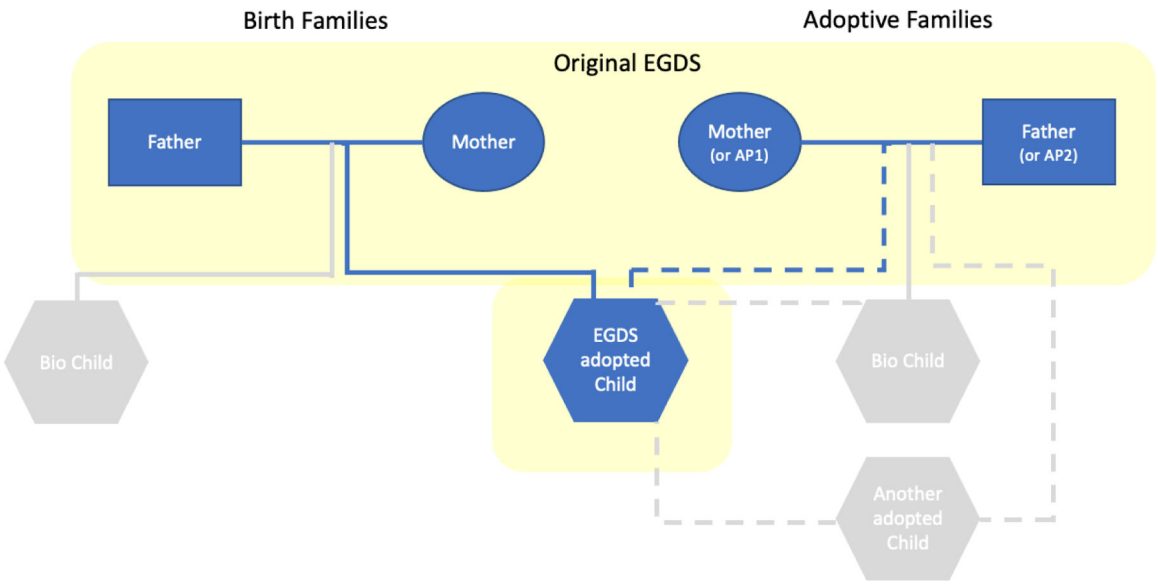

Figure 2.
The design of Early Growth and Development Study

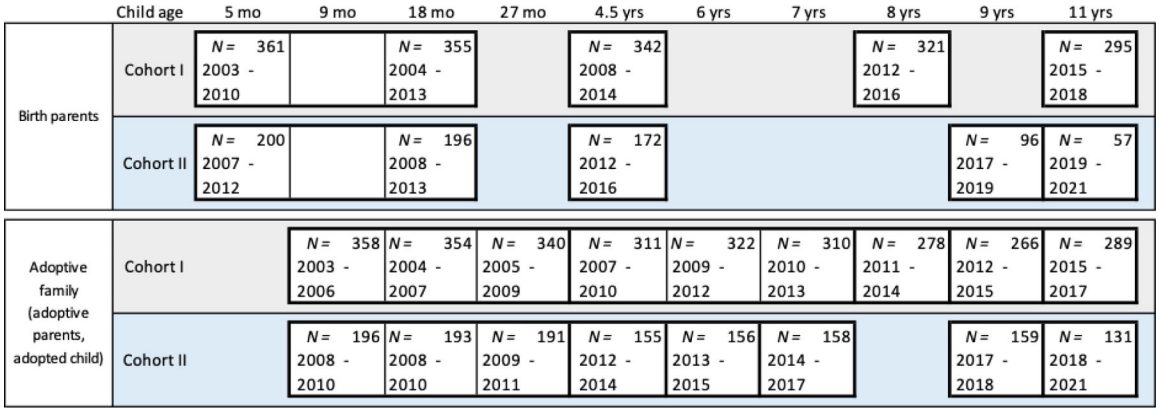

Figure 3.
The timeline of EGDS assessment.
Note. Assessments are currently ongoing; N represents the cases that completed data collection in winter 2022.

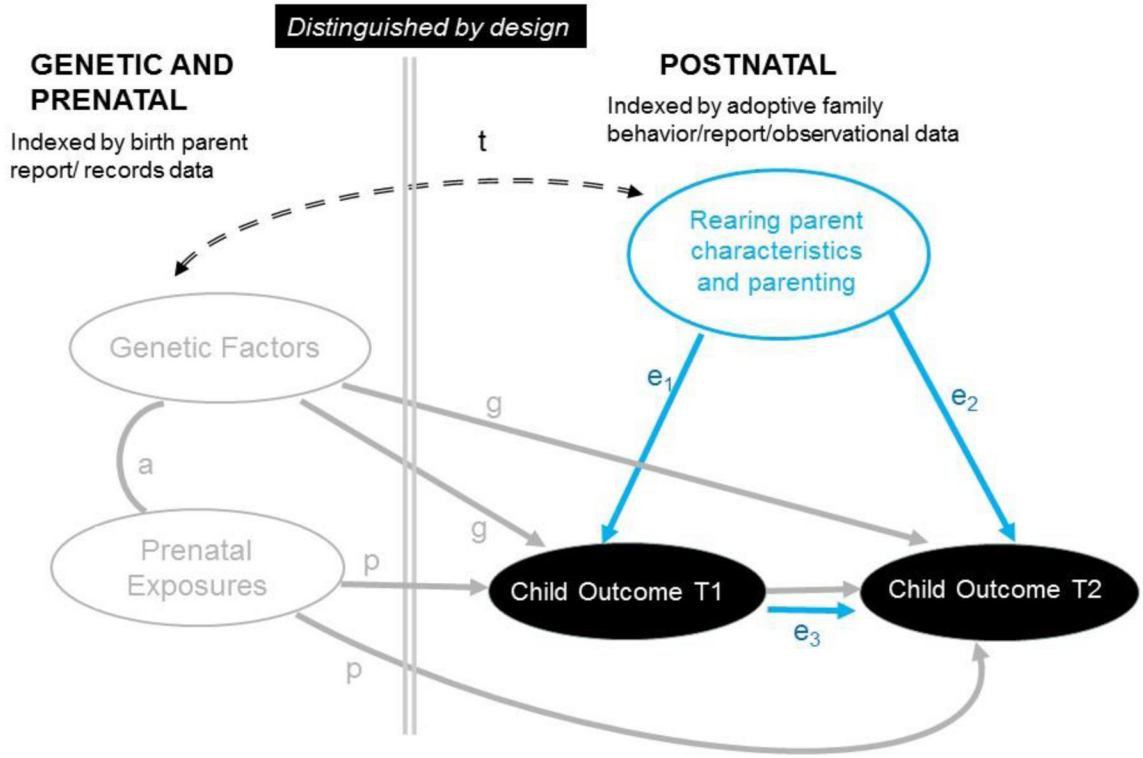

Figure 4.
The adoption design as a tool for isolating the effects of rearing parent characteristics and parenting behaviors on child outcomes over time.

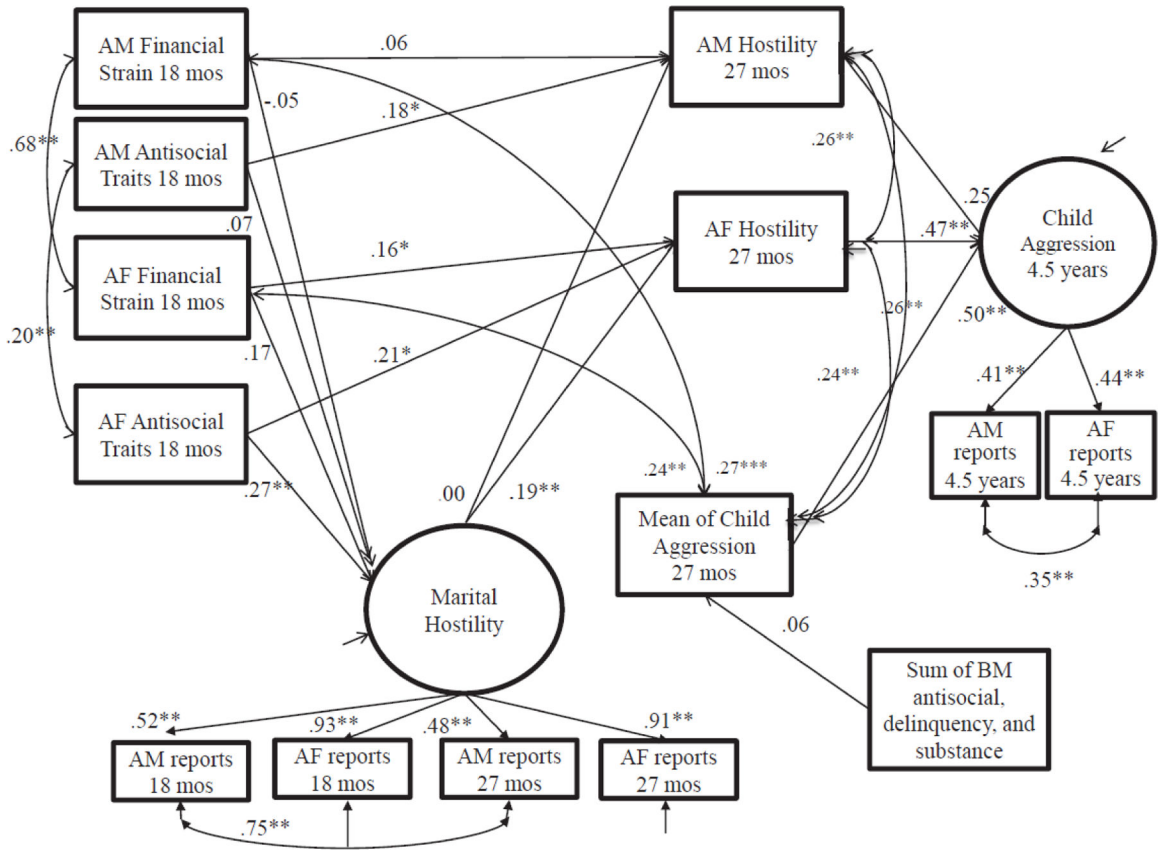

Figure 5.

Adoptive Mother (AM) and Adoptive Father (AF) hostility during the toddler period and children's aggression during early childhood, controlling for the effects of marital hostility, adoptive parent antisocial behavior, and children's heritable and behavioral risks for aggression. From Stover et al., 2016.

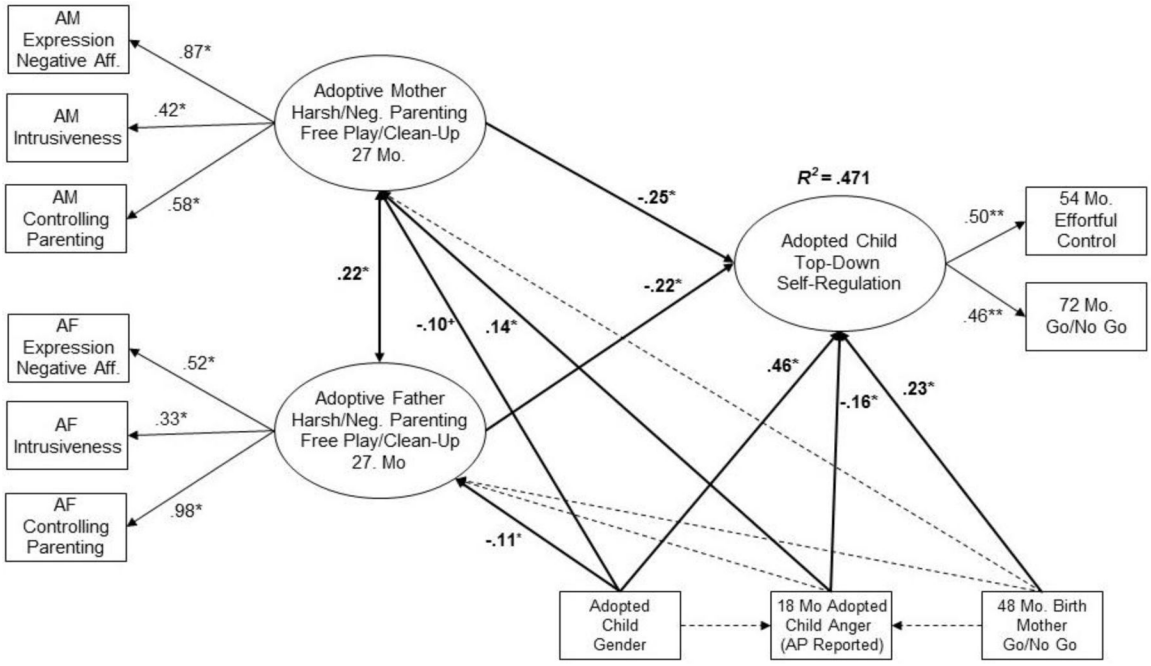

Figure 6.

Adoptive parent harshness and child self-regulation, controlling for adoptive child gender, anger, and heritable influences. From Bridgett et al., 2018.

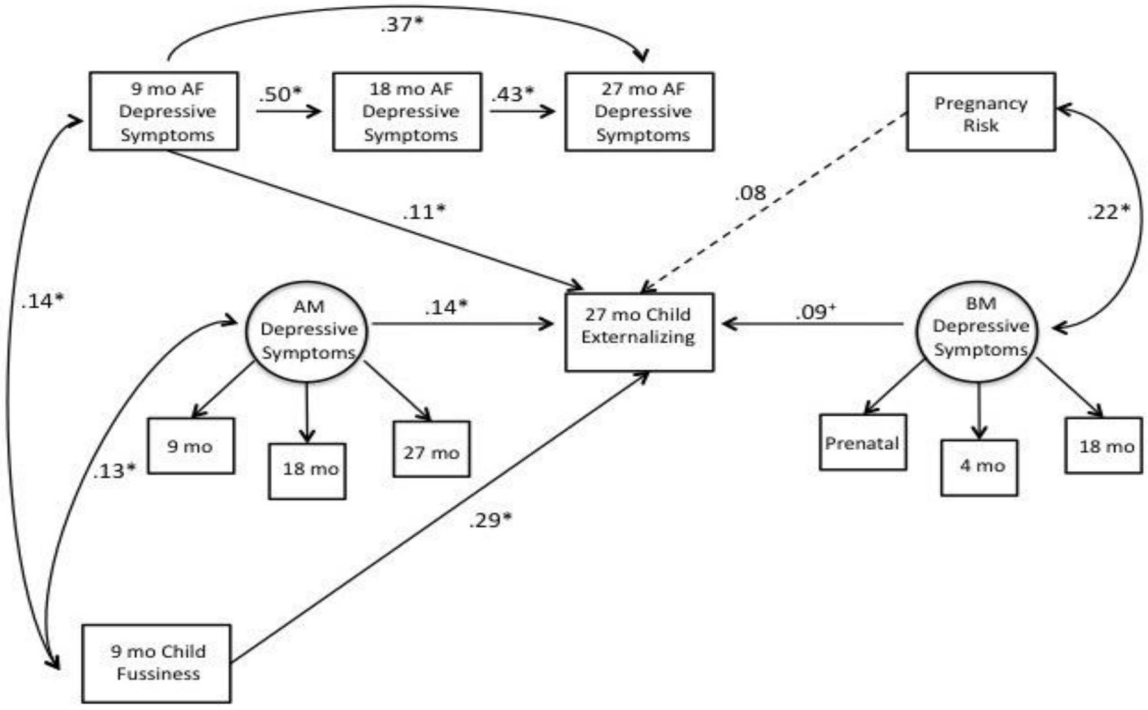

Figure 7.

Associations between Adoptive Parent Depressive Symptoms and Child Externalizing behavior at 27 months, controlling for Heritable and Prenatal Risks and Child Fussiness. From Pemberton et al., 2011.

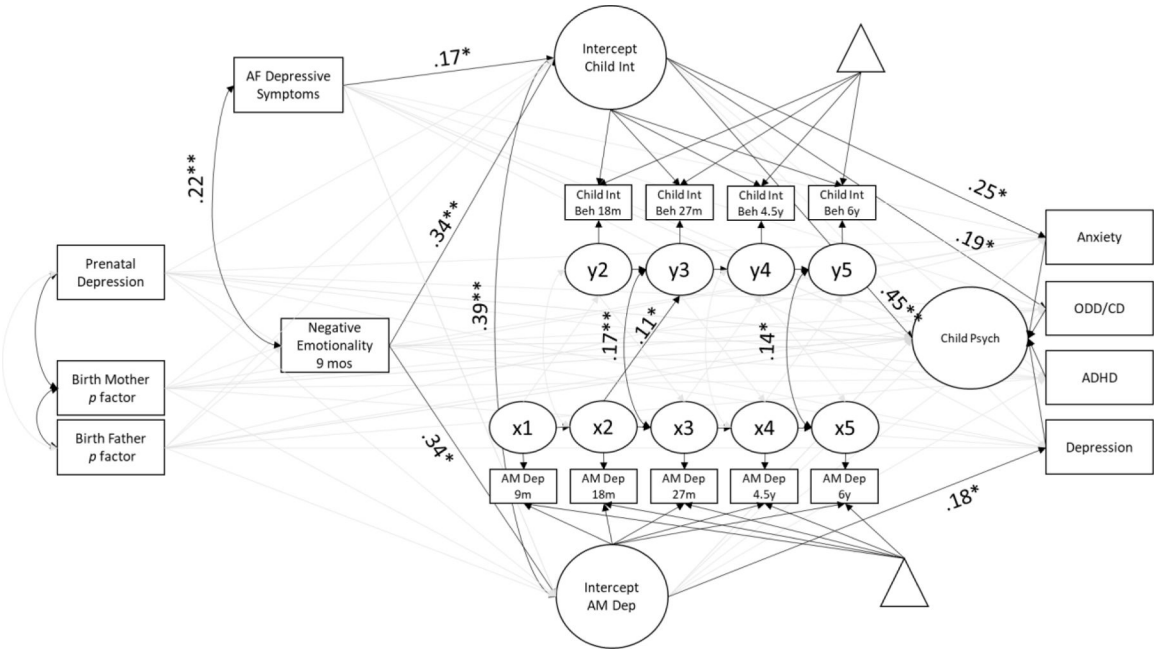

Figure 8.

Associations between adoptive parents' and children's depressive symptoms from Infancy through early childhood. From _S1_Reference86Cioffi et al., 2021.

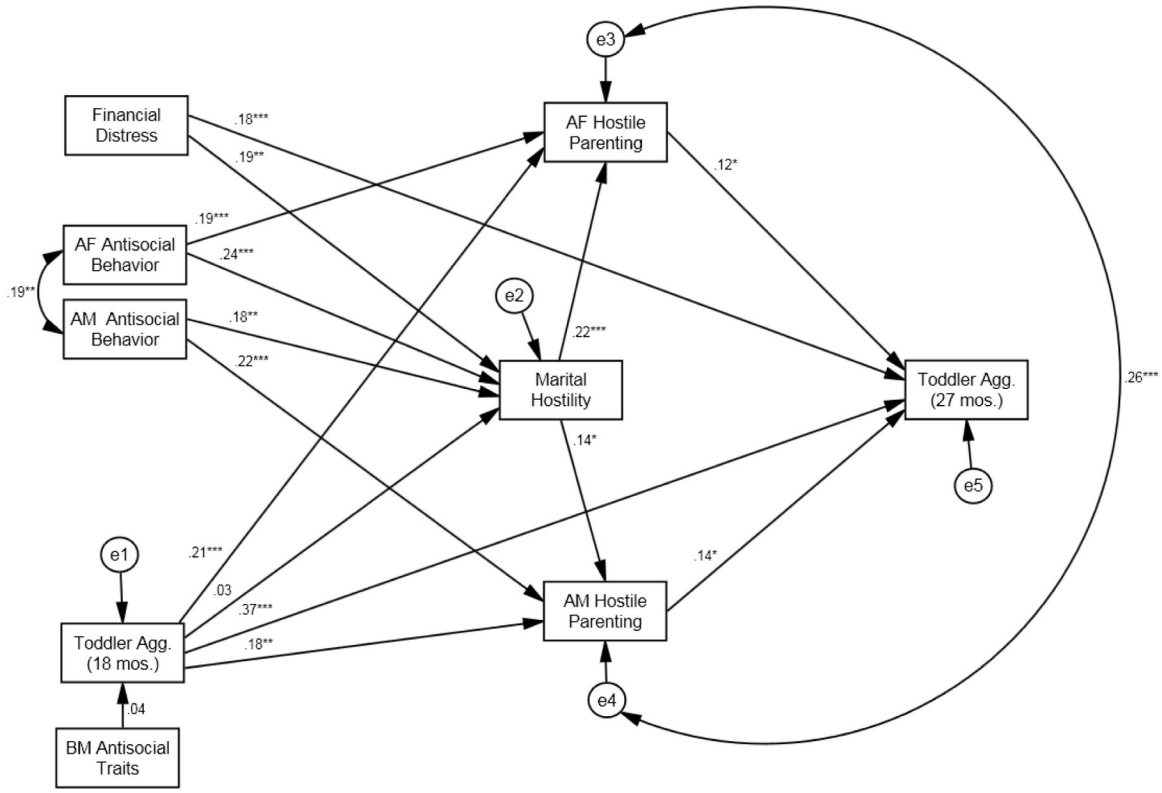

Figure 9.

The Contributions of adoptive family financial stress and adoptive parents' antisocial behavior, toddler aggression, and heritable antisocial risk to the spillover of marital hostility to parenting and child aggression. From Stover et al., 2012.

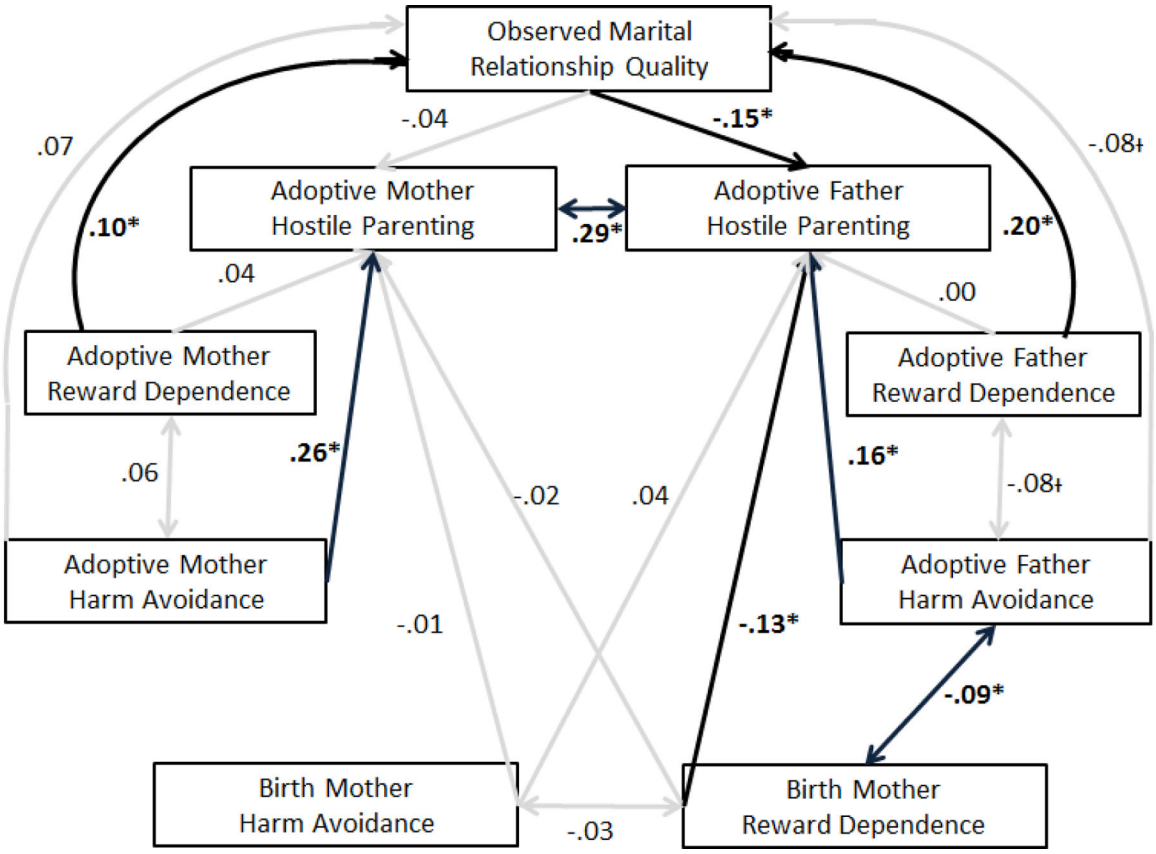

Figure 10.

The association between birth mother reward dependence, Adoptive parent reward dependence and marital quality and hostile parenting of adoptive mothers and fathers. From Hajal et al., 2015.

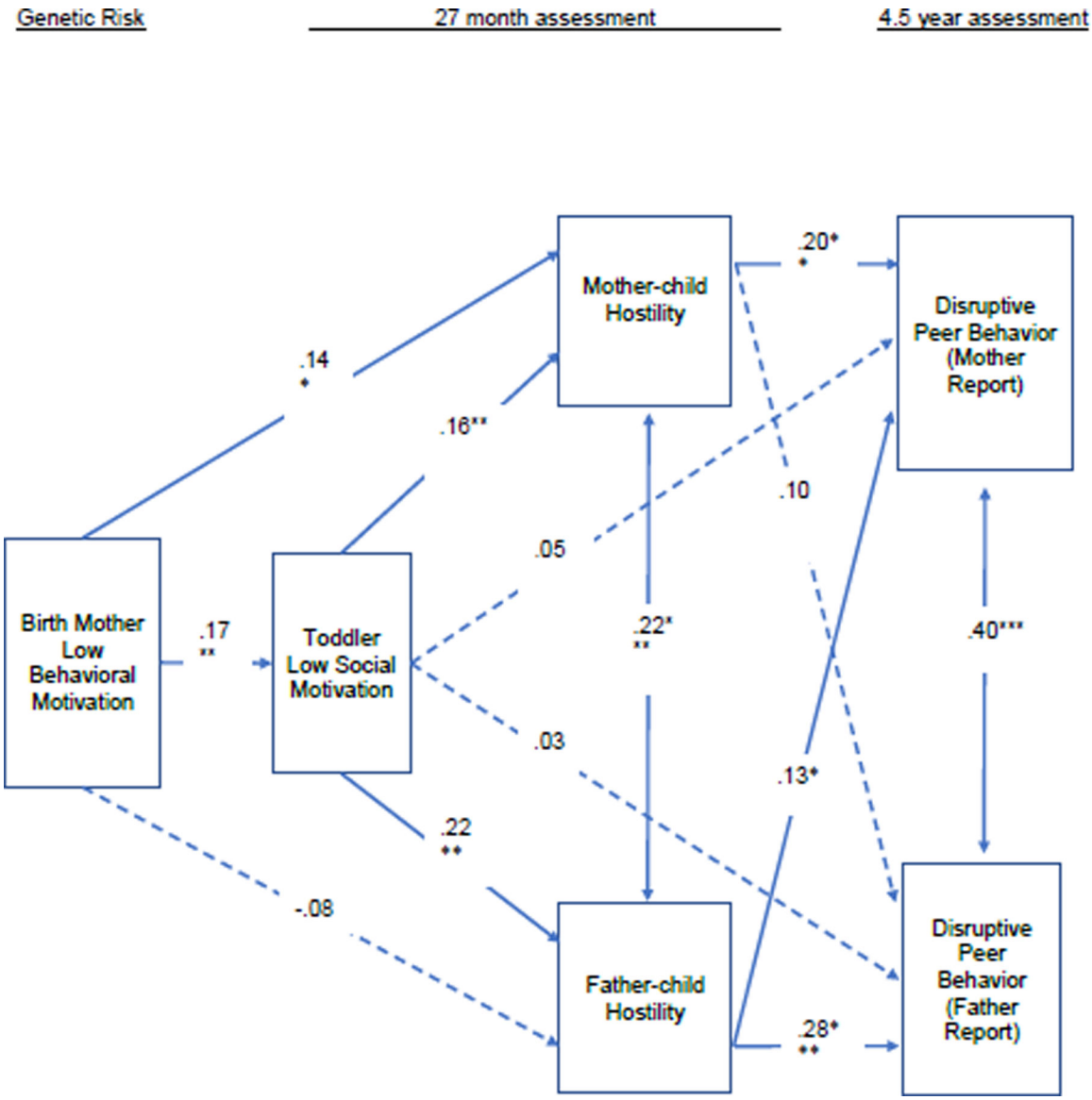

Figure 11.

The association between birth mother low behavioral motivation, toddler low social motivation and adoptive mother and father hostility predicting parent reports of disruptive peer behavior at age 4.5 years. * $p < .05$. ** $p < .01$. *** $p < .001$. From Elam et al., 2014.

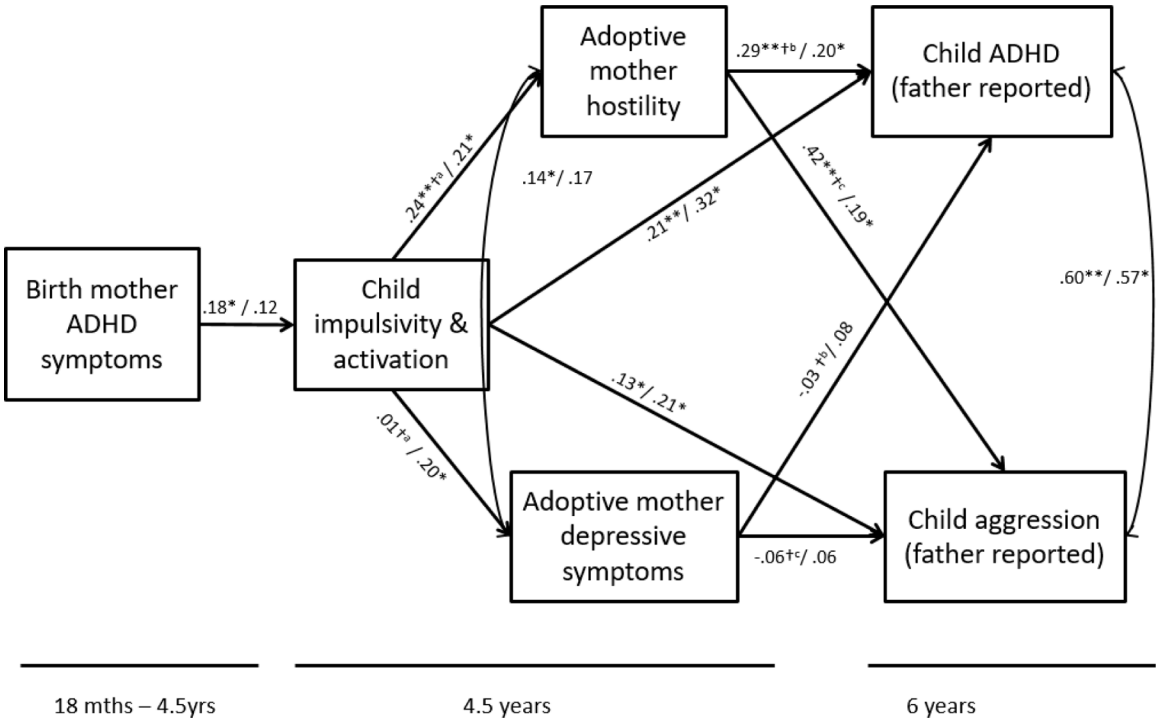

Figure 12.

Associations, for Cohorts I/II, among birth mother ADHD symptoms composite, child impulsivity, maternal hostility and depressive symptoms and child ADHD symptoms and aggression. *p<.05, **p<.01 + significantly different pathways. From Sellers et al., 2020.

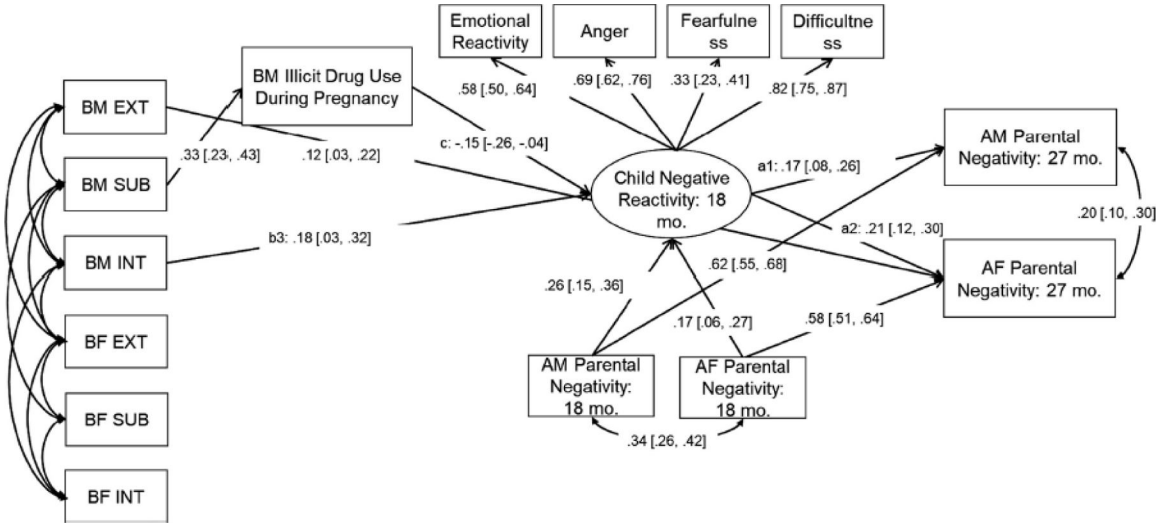

Figure 13.

Child effects on adoptive parents' parental negativity via child negative reactivity: the role of heritable and prenatal factors. Standardized parameter estimates and 95% credible intervals of parameter estimates. AM = adoptive mother; AF = adoptive father; BM = birth mother; BF = birth father; EXT = externalizing problems; INT = internalizing problems; SUB = substance use. The parameter estimates of the covariance among birth parents'

psychopathology scores were omitted due to space. Nonsignificant parameter estimates are not shown. From Liu et al., 2020.

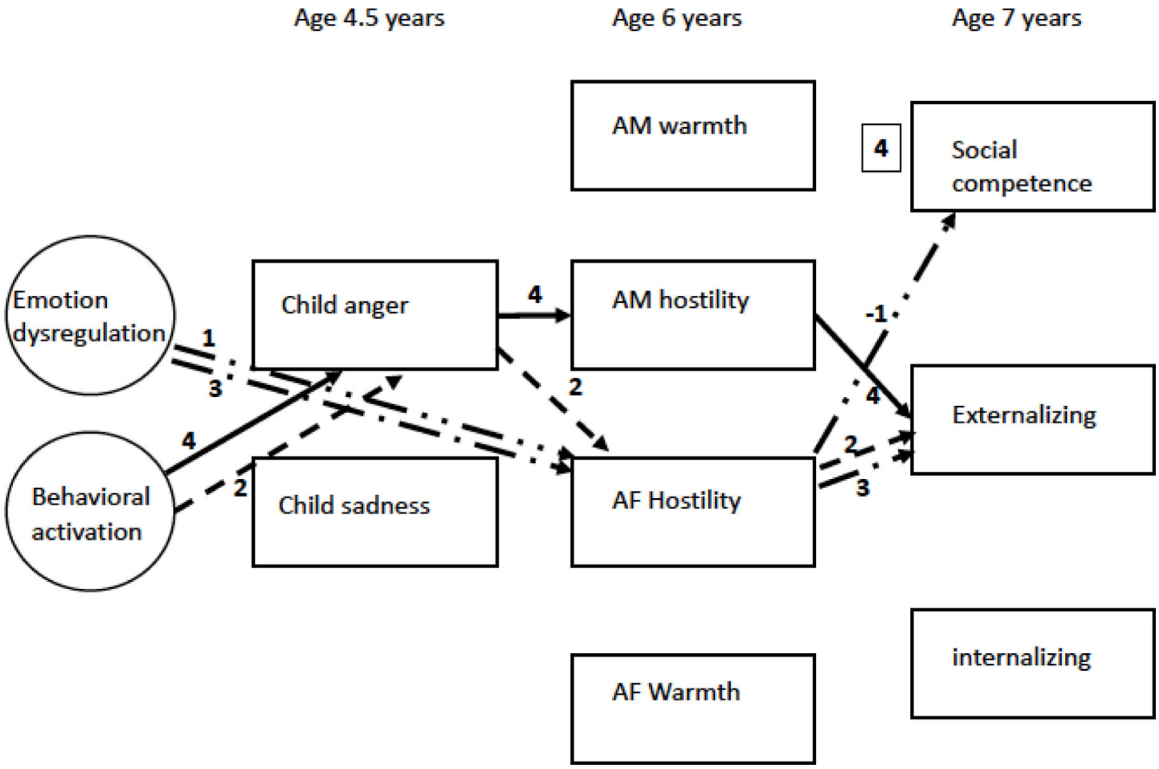

Figure 14.

A diagram of significant indirect paths linking birth parent temperament with child social competence and externalizing via primary child effects on parental hostility. Adapted from Shewark et al., 2021.

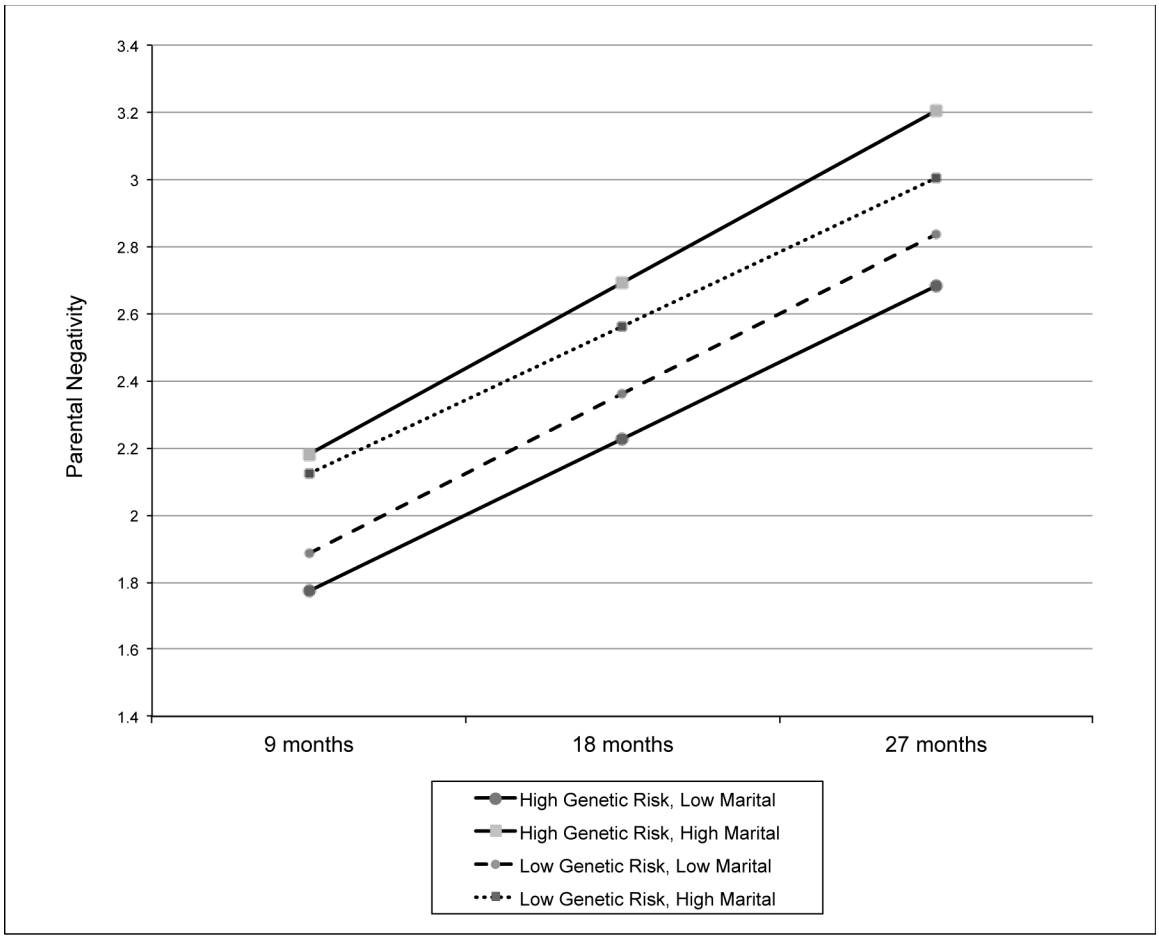

Figure 15.

The interaction between genetic risk and marital problems of the rearing parents on maternal negativity. From Fearon et al., 2015.

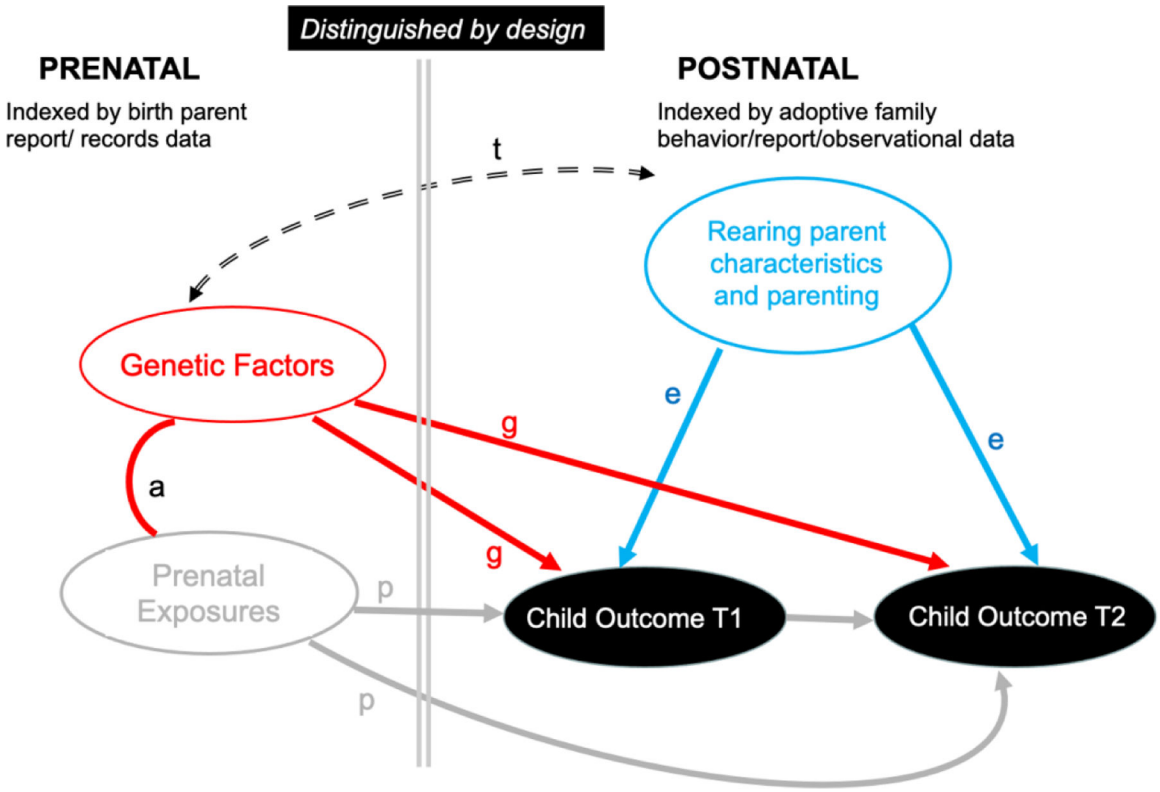

Figure 16.

A schematic illustrating the analyses to be reported in this chapter: the effects of genetically influenced characteristics of the child on the influences of rearing parents.

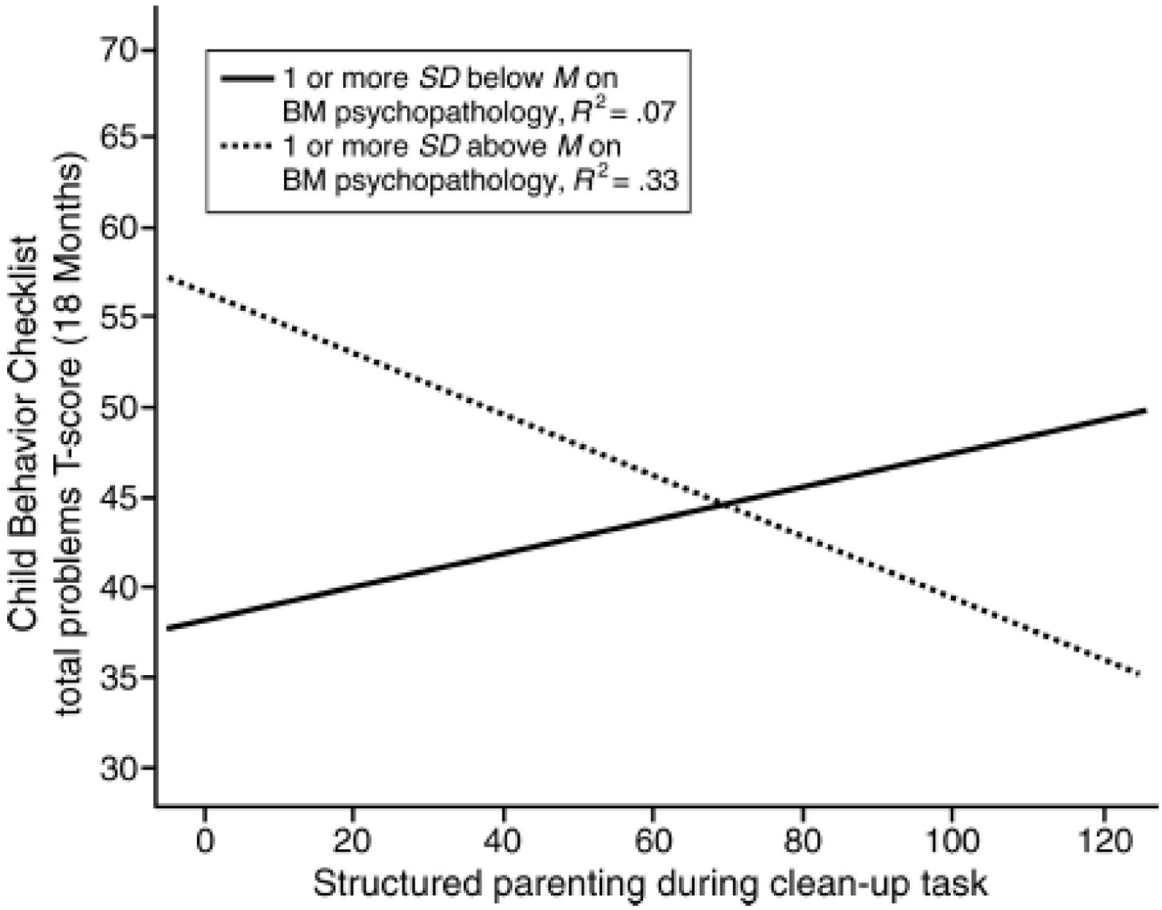

Figure 17.

The interaction between genetic risk for a broad range of psychopathology and structured parenting. Structured parenting is inversely related to child problems for those at high genetic risk but positively associated with child problems for those at low genetic risk.

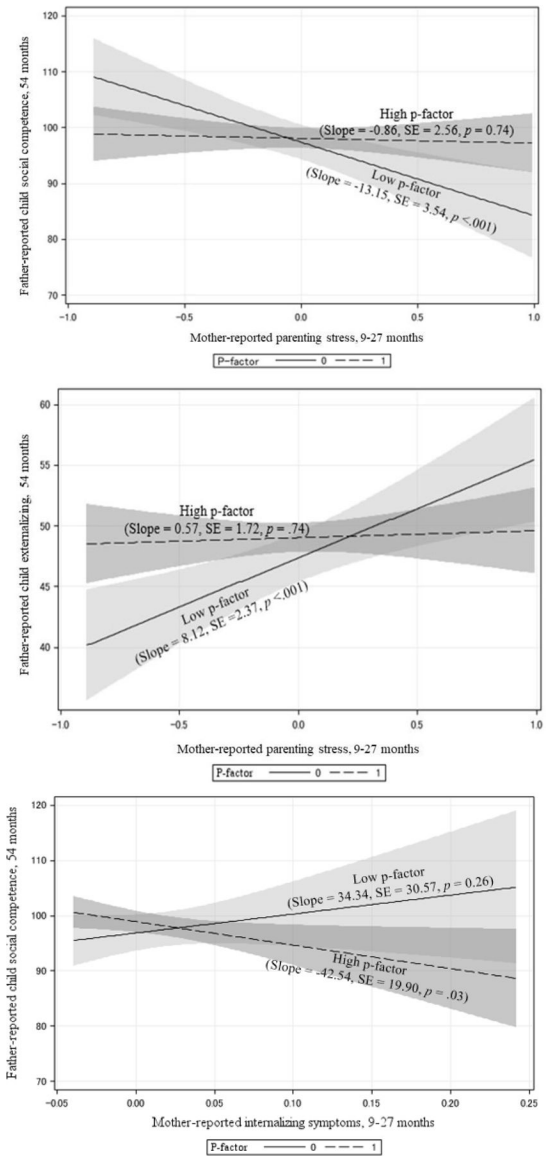

Figure 18.

Top: The interaction mother-reported parenting stress and the little p factor on father-rated child social competence. Middle: The interaction mother-reported parenting stress and the little p factor on father-rated child externalizing. Bottom: the interaction of mothers' reports of their internalizing symptoms and little p on father reported child social competence. From Cree et al., 2020.

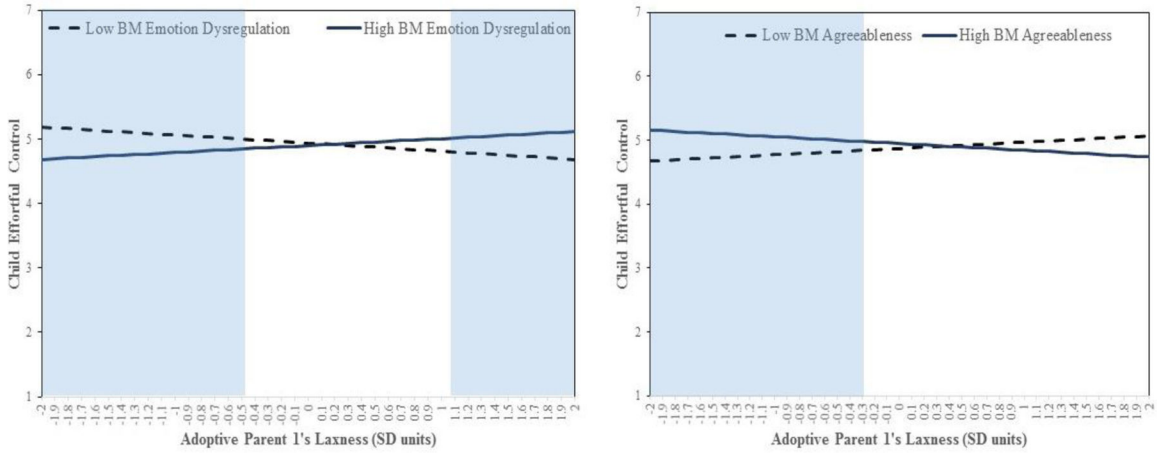

Figure 19.

The interaction of maternal laxness (vs. structure) with birth mother personality (agreeableness, emotion dysregulation) on child effortful control. From Ganiban et al., 2021.

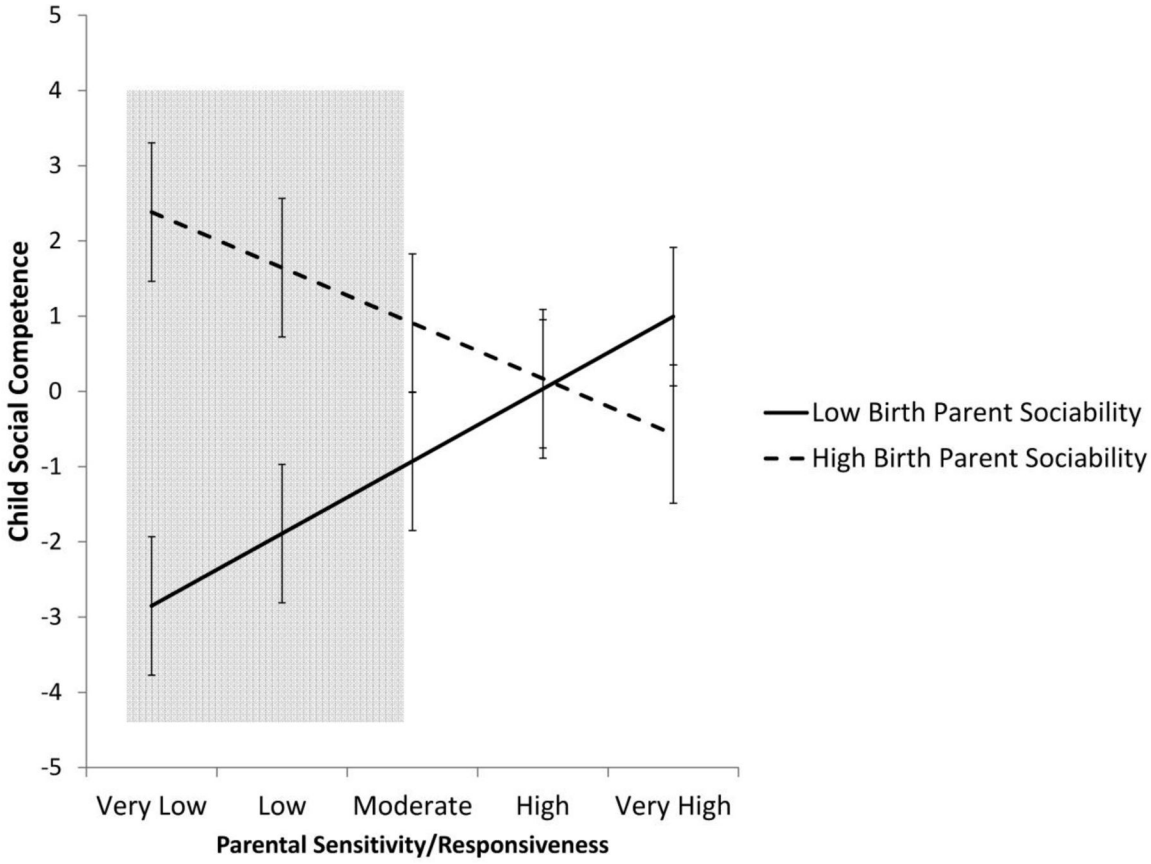

Figure 20.

Interaction between parental responsiveness and birth parent sociability and its association with child social competence at age 6. From Van Ryzin et al., 2015.

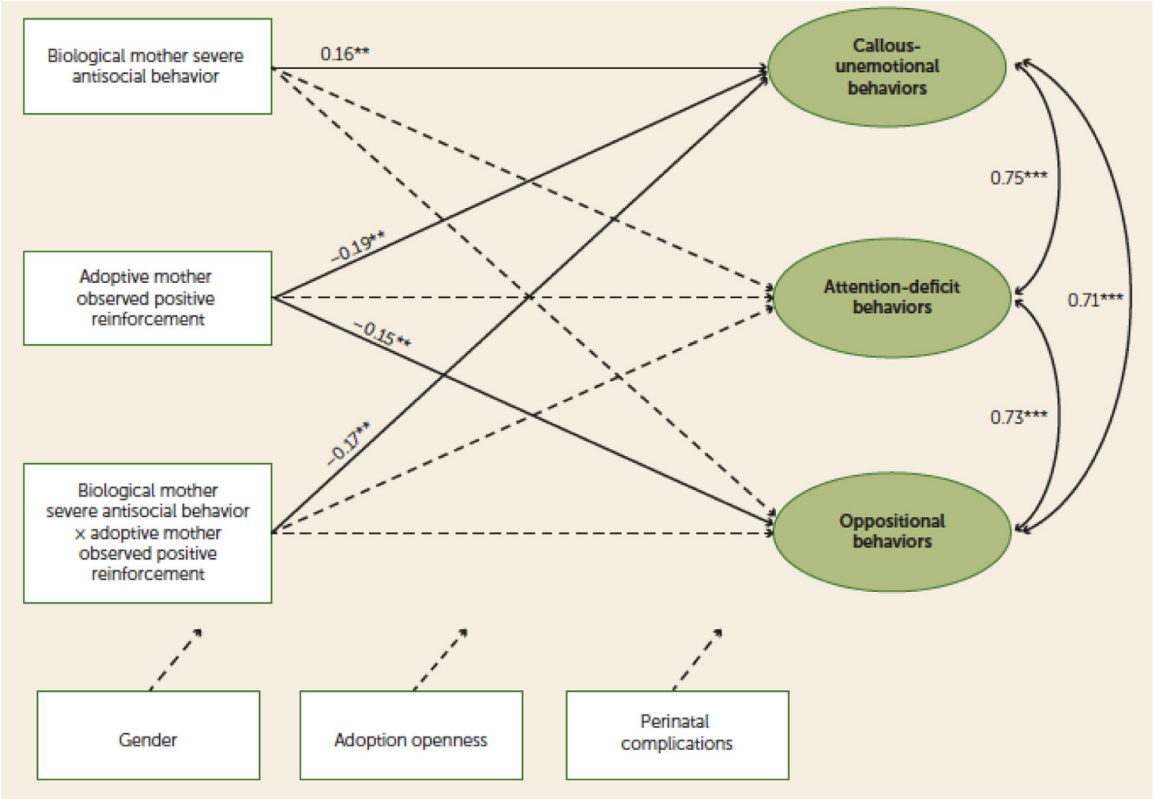

Figure 21.

The relationship of birth mothers' antisocial behavior, measured at child aged 3 months, adoptive mothers observed positive reinforcement at child age 18 months and their interaction on three measures of child behavior at 27 months. In addition to child gender, adoption openness and perinatal complications, children's callous and deceitful behavior at age 18 months is held constant.

[a] Severe biological mother antisocial behavior predicts adoptive child callous-unemotional behaviours, but the effect is buffered by adoptive parent positive reinforcement.

*p<0.05. **p<0.01. ***p<0.001.

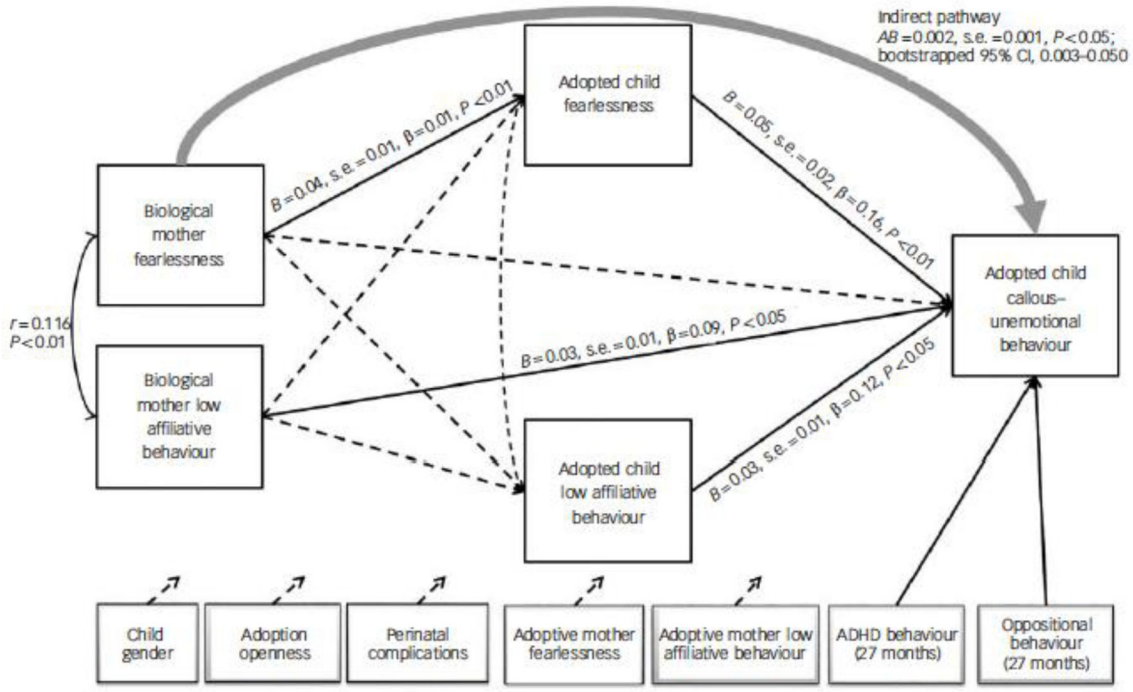

Figure 22.

In the association of biological mothers' temperament with child temperament at 18 months and callous unemotional behavior at 27 months. The broad, grey arrow indicates the indirect effect of adult personality to child temperament to child callous unemotional traits. These analyses control for child gender, adoption openness, perinatal complications, adoptive mother personality, child ADHD and oppositional behavior.

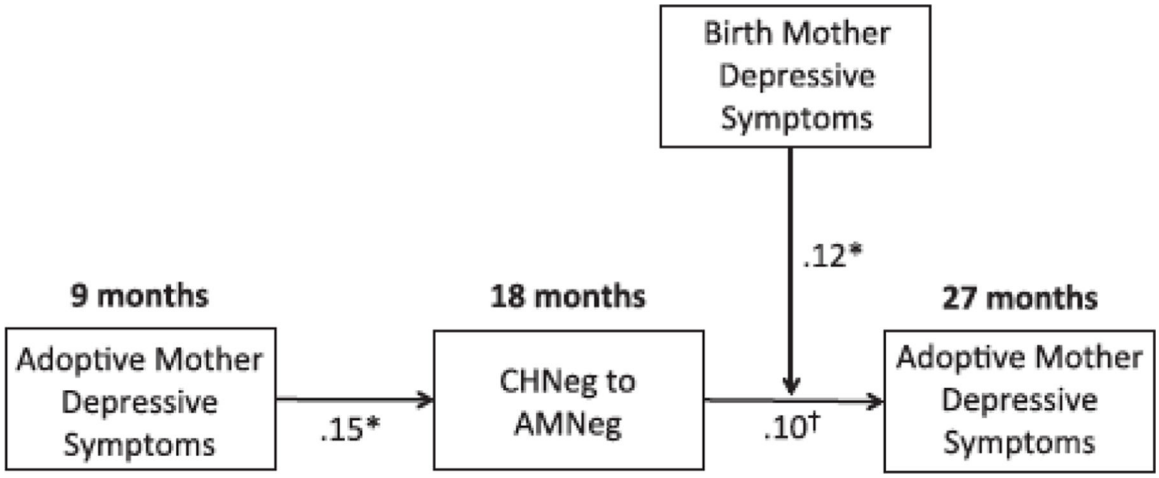

Figure 23.

Genetically influenced self-defeating feedback: Separate influences from adoptive mothers and birth mothers imply that in biological related mother child pairs, mother's parenting acts to enhance a genetically influenced child behavior that, in turn, exacerbates her own depressive symptoms.

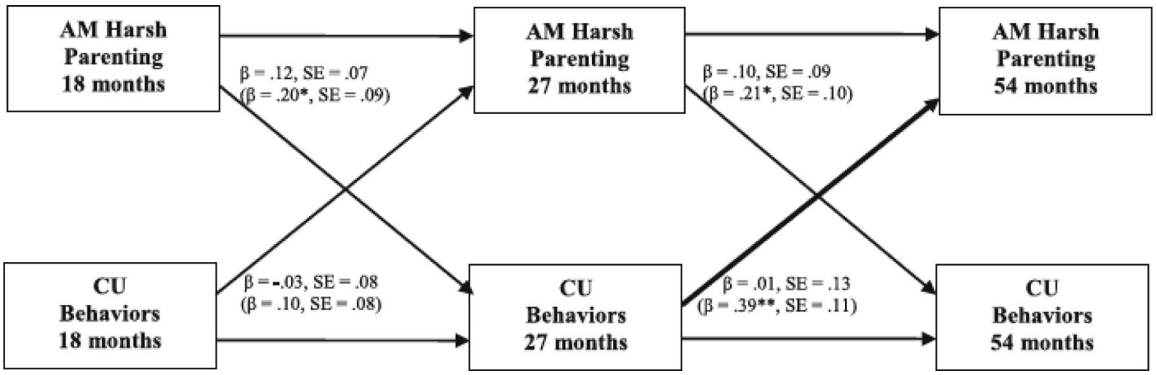

Figure 24.

A genetically influenced self-defeating spiral for callous unemotional traits in children and hostile parenting. Coefficients outside of parentheses are those for the low genetic risk group and those within parentheses are those for children at genetic risk as indexed by maternal low social affiliation and fearlessness.

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