Advances in Development and Psychopathology:
Brain Research Foundation Symposium Series

Patrick H. Tolan
Bennett L. Leventhal
Editors

Disruptive Behavior Disorders

Springer

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Foreword

The *Brain Research Foundation Symposium Series* was born in Chicago, Illinois, at a meeting of the Board of Trustees of the Children's Brain Research Foundation.

We had finished our regular business when our Chairman, Norm Bobins, asked in his characteristic way, "Isn't there something more we can do?" This led to a spirited discussion and ultimately the following thought: What if we brought together leading scientific lights from around the world to focus on a topic of great importance to the field of neuroscience research, with the goal of sparking ideas and collaborations that might not otherwise exist? There could be a series of these symposia, each focusing on a different topic, and the results could be shared in a published work that might serve as an inspiration and research tool for other scientists, perhaps generating even further ideas and collaborations.

Our Board enthusiastically embraced the plan. Drs. Bennett L. Leventhal and Patrick H. Tolan were tasked with the responsibility of organizing the first symposium. The topic—"Disruptive Behaviors in Children and Youth"—was chosen, a venue was selected, and invitations to scientists were sent out.

We had expected a positive response from the scientists we had invited, but were not prepared for the high level of enthusiasm that our invitation provoked. That enthusiasm was carried over into the symposium itself. As one scientist commented at the reception held the evening before the symposium, "I receive many invitations to scientific meetings, but this is one I could not possibly pass up!"

In May 2010, the Children's Brain Research Foundation merged with the Brain Research Foundation, the oldest organization in the United States devoted to funding research on a broad spectrum of neurological disorders, and the combined organization enthusiastically committed itself to the symposia project. At the time of this writing, plans are being made for our second symposium. Consistent with the Brain Research Foundation's vision of funding breakthrough research in the field of neuroscience, the topic chosen for the next symposium is "Gene-Environment Interactions in Developmental Psychopathology and Their Role in Intervention Research."

We are deeply grateful to Drs. Leventhal and Tolan for their dedication and initiative in organizing the *Brain Research Foundation Symposium Series*, to Terre A.

Constantine, Executive Director of the Brain Research Foundation, and her staff for their excellent work on the project, and to our President, Nathan T. Hansen, and our Board of Trustees for their unwavering support. Thanks also to Springer Science + Business Media for publishing this series. But most of all, thanks to all of the scientists who participated in this first symposium and brought it to life.

We are very pleased that this volume is being published in 2013, which marks the Brain Research Foundation's 60th year. We hope that you find this, and future volumes in the series, to be of value.

Chicago, IL, USA

Richard M. Kohn
Member and Secretary of the Board of
Trustees of the Brain Research Foundation

Participants in Brain Research Foundation Symposium, October 2008

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Chapter 1
Introduction: Connecting Brain Development, Disruptive Behavior, and Children

Patrick H. Tolan and Bennett L. Leventhal

Problematic behaviors in children and adolescents have plagued families, educators, and clinicians for millennia. In fact, they are the most common reasons for referral for mental health services (Costello & Angold, 2001). Among these behaviors, oppositionality and aggression are often the most disturbing and disruptive. While they have proven to be clinically complex, research advances in neurobehavioral sciences have created new opportunities for understanding these problems. Yet, many challenges remain and must be addressed for the needed progress in this area of investigation.

While such problematic behaviors may be a part of several clinical syndromes, when not explained by other specific conditions (e.g., anxiety, mood, psychotic disorders, etc.), they become Disruptive Behavior Disorders (DBDs; DSM-IV-TR, 2000) when they are a recurrent pattern of behaviors that interfere with development and adaptation (Moffit & Scott, 2009). In DSM-IV clinical parlance, the DBDs include attention deficit/hyperactivity disorder (ADHD), oppositional defiant disorder (ODD), conduct disorder (CD), and DBD, not otherwise specified; while each of these conditions is significant in its own right, persistent oppositionality and aggression pose especially vexing problems that are the subject of this volume.

Thus, for this discussion, "disruptive behavior" represents a clinical syndrome that is characterized most significantly by engagement in repeated acts of aggression toward others that are often accompanied by little or no regard for the effects of such behavior on others nor the value of complying with directions, requests, and

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expectations for conformity from parents and other authority figures (Kazdin, 2005). It is generally considered that there is a neurobiologic substrate that contributes to these conditions (Moffit & Scott, 2009).

While of concern to society for centuries, attention to behavior problems as a perturbation in development reflecting potential disturbances in underlying psychological and sociological processes did not emerge until the late nineteenth century (Costello & Angold, 2001). As child welfare emerged as a recognized societal responsibility along with interest particularly in the United States about socializing immigrant youth, what was expectable behavior (acceptable and healthy) commanded medical and sociological opinion and drove social and political policy. This interest can be seen in the development of the first juvenile court in 1899, designed to provide oversight of parental behavior and the activities of wayward children, as well as a way to mandate interventions deemed necessary to remediate youthful threatening and disruptive behavior (Scott & Steinberg, 2008). Since that time, this intersection of concern about behavior that seems to reflect a lack of control, and is harmful to others, has been of great interest to child development scholars. Both at first glance and upon extensive study, these problem behaviors seem to reflect scientific and social challenges, each characterized by being at the intersection of scientific study of child development and social concern about aggressive and harmful behavior. These include the relative importance of societal and biological influences, the child as an emerging organism that is malleable and redirectable on the way to becoming a fully shaped person, the responsibility for individual behavior in the context of social mores, what distinguishes pathological processes from misbehavior and other deviance, and understanding the processes that lead to disruptive behaviors.

Each of these concerns has been and is still of keen interest to scientists, parents, a full-spectrum of health care personnel, criminology professionals, and policy makers. This symposium and the derivative chapters in this volume represent a focused attempt to provide additional understanding of this complex area of research and practice. Our goal was to summarize the current knowledge related to key questions in scientific understanding of disruptive behavior problems and to suggest important next steps in furthering knowledge that can guide action. The principal model underlying this effort is framed within the application of advances in understanding brain development as well as the growing specificity and complexity of developmental theory relevant to disruptive behavior problems.

Disruptive Behavior Disorders: Conduct and Oppositional Defiant Disorders

While in the official nomenclature, DBD includes ADHD, for the purposes of this volume, we will use DBD to refer to ODD and conduct disorder (CD). ADHD is frequently comorbid with CD and ODD and, along with other "externalizing disorders," may even share common developmental pathways; ODD and CD are often

seen as directly related if not continuous or synonymous (Moffit & Scott, 2009). However, ODD and CD are usually differentiated from ADHD by the predominance of behavior problems in the phenotype. Thus, in psychiatric, educational, and legal arenas, ODD and CD are typically more closely aligned than differentiated from ADHD. However, for the purposes of the symposium and this volume, we have constrained our focus to ODD and CD and the disruptive behaviors that characterize these two disorders.

While ODD and CD represent the current psychiatric nosology for a specific set of behavior problems, the "labeling" also draws from other disciplines, such as education with respect to the social–behavioral problems that disrupt classroom learning and from the legal system which attends to these behaviors which incur liability due to the harm to others which may constitute criminal actions. Currently under revision, the American Psychiatric Associations Diagnostic and Statistical Manual (DSM-IV-TR, 2000), which is used to officially designate diagnoses of psychiatric disorders, emphasizes the close relationship and overlapping symptoms of conduct disorder and ODD. As noted throughout this book, the primary behaviors that constitute the critical signs and symptoms for each are aggression, noncompliance, defiance, and low concern for the effects of such behavior on others. ODD is characterized as a less severe form of disruptive behavior and is typically identified earlier in development. Symptoms of ODD include a pattern of negativistic, defiant, noncompliant, and argumentative behavior, lasting for at least 6 months, and causing significant impairment in social or academic functioning. Typically ODD behaviors are first exhibited in preschool age children. Conduct disorder (CD) is typically first identified in school age children, or adolescents. CD symptoms include: (1) aggression to people and animals, (2) destruction of property, (3) deceitfulness or theft, and (4) serious violation of rules. The nuances of these diagnostic criteria are discussed in more detail in several chapters of this book. These conditions are relatively common with prevalence estimates ranging from 5 to 25 %, with both about twice as prevalent in males as females.

While related and conceptualized as having some developmental order (meaning ODD typically evidences earlier and may precede CD), only about 25 % of children with ODD ultimately develop CD. Also, often conceptualized as chronic conditions and, in some cases, life-course persistent disorders, most children diagnosed with ODD or CD do not show such a pattern. While children and adolescents with a diagnosis of ODD/CD may not have a persistent disorder, the presence of disorder does predict behavior problems and limitations in functioning that are broad and lasting (see Loeber, Capaldi, & Costello, 2013).

As with many psychiatric disorders, some elements of the cardinal diagnostic phenotype represent more extreme, more frequent, and/or developmentally unusual versions of what may be common behaviors seen in the general population. When less extreme or at lower rates of occurrence, such behaviors do not indicate any particular tendency, subclinical version, or emerging disruptive behavior problems. The differentiation is when the pattern is substantial enough and the acts harmful and disruptive enough to offend the sensibilities of others, disturb social order in groups and settings in which the child is present, and/or represent physical or other

harm to others. This feature suggests that there is important understanding of the disorders in focus on how these behaviors are related to brain development and functioning and important environmental influences on their expression, in typical functioning persons and in those with elevated but not clinical level exhibition as well as part of the syndromes of ODD and CD. This volume and the symposium on which it is based applied a brain-related ecological development perspective utilizing scientific findings of typical, at-risk, and clinical populations.

Disruptive Behavior Problems: A Brain-Related Developmental Science Focus

It is brain-related developmental phenomena that make disruptive behavior problems an optimal focus for this first Brain Research Foundation Symposium on Development and Psychopathology and this resulting volume. It is a vexing problem to determine what and when aggressive and noncompliant behaviors, evident among most persons at some point in development, albeit in less severe and less frequent form, become evidence of a brain disorder that constitutes a psychiatric illness. Thus, DBDs become a rich, if not, easy set of scientific challenges that merit careful examination. Questions arising from these challenges include: What is the relationship between illness and variations in healthy behavior? Are there meaningful, qualitative distinctions that can move us beyond the seemingly arbitrary criteria for that lead to the assignment of a diagnosis? How well reliably related to differences in pattern, progress, and fullness of brain development, including related regulatory systems, are the observed clinical and behavioral differences between typical development and disorder? Pursuing these questions, and others, may not only aid in our understanding of these serious personal and societal problems, but also can break trails for understanding brain functioning in relation to development, to allow tracking of the development of illnesses in relation to brain function.

With our burgeoning knowledge about brain organization, structure, and function, our perspectives are rapidly changing with respect to how the brain relates to the emergence of cognitive, emotional, and behavior disorders. Even in what is clearly an early stage of correctly aligning and relating the many new findings about how the brain affects behavior and developmental pathways, real scientific progress seems likely to be a process of elaborating promising findings so that they can be incorporated into considerations of sensitivities to conditions, variations on major themes that apply to many children and populations, and need for greater specificity and sophistication in developing causal formulations. While some of the basic processes, tools, and areas of emphasis have been identified, the field is just beginning to glimpse how these will form a coherent description of how disruptive behaviors are formed and what biological factors, other developmental influences, and the larger context are critical to the evolution of such conditions.

The findings reviewed in this volume and the theoretical formulations offered by the contributors both reflect the most current knowledge and serve to suggest

promising areas for further study. Equally importantly, this volume is intended to identify specific areas of intellectual and technical overlap, thus opening the door to much needed, cross-speciality study, and for the ongoing conversations that will be necessary to reconcile inconsistencies, misdirections, and anomalies. These promising and vital threads of scientific knowledge and theoretical integration appear to have not substantially influenced practice, particularly in the areas of diagnosis and treatment, at least not enough to have had much impact. At present, it appears that the past decade has seen substantial progress in the level of certainty about which child referred for disruptive behavior problems actually has an illness and which does not. Such diagnostic decisions still rely on clinical judgment which seems too susceptible to biases and undue influence from circumstances of referral rather than a clear valid formulation consistently applied and resting on pathognomonic markers of adequate reliability and sensitivity.

Similarly, while there has been considerable progress in identifying a cadre of treatment and preventive interventions that can curb symptoms and direct management to more successful outcomes for children with DBDs, much interventions do not rely on validated models of causality or what elements of the intervention actually alter the course of disorder. Even those treatments with evidence of significant and lasting effects are not well understood within a neurodevelopmental framework. Most are quite general and applied to children who have a wide variety of symptoms, circumstances, and need. While useful, these blunt instruments are far from the level of differential application and specificity of training for effective use that is needed. In all likelihood, this situation is one explanation for why even for the "best" treatment and prevention efforts, a substantial majority of those children exposed do not show benefits (even when mean effect sizes are significant and substantial). As with the diagnostic process, treatment application has not been framed within an understanding of brain–behavior relations nor of presumed meaningful heterogeneity among those with a disruptive behavior. These shortcomings of the work to date also suggest great potential in approaching the seemingly disparate, yet important, recent, scientific findings on intervention effects in a vein that reflects neurodevelopmental processes in a particular child within the context of those key factors that influence both behavior and the course of development. Indeed, such a framework was used to organize the symposium and this resulting volume.

A Brief Description of Brain Development Related to Disruptive Behavior Development

Features of brain development. Neural development before and during early childhood comprises rapid shifts in form and function that lead to improved perceptual accuracy, better attentional direction, and more effective emotional and behavioral regulation. Initial exuberant synaptic connection is followed by myelination and then synaptic pruning with concurrent elaboration and then refinement of pathways

of dynamic connection between brain regions (Huttenlocher, 1990; Ramakers, 2005). While most myelination is complete by early childhood, some regions continue to myelinate into early adulthood (Lenroot & Giedd, 2006). With these developments, differentiation of brain region function also increases. Frontal regions that are the location of executive functioning, including the functions of attention, working memory, and organization of impulses and emotional stimulation, are maturing, but later and with a much longer maturational trajectory than those in the basic motor or sensory regions (Shaw et al., 2008). This rapid and complex development is such that approximately 95 % of brain volume is completed by age 6 (Giedd et al., 1996). This is the time when white matter volume, responsible for the extent, rapidity, and accuracy of communication within the brain, increases linearly with age similarly across regions, while cortical gray matter which is thought to be responsible for extent or content of knowledge grows but not in a simple linear fashion with substantial regional differences (Giedd et al., 1999). Cortical thickness shows similar development (Shaw et al., 2008). These patterns show initial increases over childhood with some decline in adolescence and then a stabilization in adulthood. Cortical thickness exhibits a marked dorsal to ventral progression with higher order cortical areas reaching peak thickness last (around 10 years of age) (Shaw et al., 2008). Accompanying these anatomic progressions are changes in brain activity. For example, glucose metabolism increases from birth to peak about age 9 before declining to stable levels in adulthood. Similarly, cortical functional development is characterized by shifts from diffuse to focal responses in attention management (Casey et al., 1997; Durston et al., 2006) and in resting state (spontaneous fluctuations) (Fair et al., 2009; Kelly et al., 2009; Supekar, Musen, & Menon, 2009). The overall developmental pattern changes from diffuse activation and inefficient brain interconnectivity to focused, more efficient activation and relation of input to output due to synaptic pruning, myelination, experience, and other elements. Thus, there is evidence that brain physiology and functions develop simultaneously during childhood and into adolescence. In particular, there are developments that seem traceable to increasing cognitive control, ability to direct attention, and understand and regulate emotions. While postnatal brain development appears to be most rapid and far reaching during the preschool years, there is essential, ongoing brain development well into and beyond adolescence.

Sex differences in brain development. Different susceptibility to DBDs and differences in the predictors and patterns of behavior by sex have been well documented (see Loeber et al., 2013 for a summary discussion). Similarly, there is extensive documentation of sex differences in brain development (Aleman & Swart, 2008; De Bellis et al., 2001; Giedd et al., 1999; Lenroot & Giedd, 2010; Lenroot et al., 2007; Sowell et al., 2007). While males appear to have large brain volumes this does not appear to be related to any particular advantages in functioning, with the general impression being that female brain development during childhood and through adolescence advances rapidly, yielding greater biological and social maturity for females during these development epochs (Lenroot & Giedd, 2010). Thus, while there has not been direct evidence about sex differences in brain development among children with DBDs that help explain different prevalence and manifestations of

disorder, this will likely be an important area of inquiry and a critical aspect of a brain development framework for understanding disruptive behavior.

Brain development and DBDs. Studies of brain physiology and function have demonstrated differences between clinical groups exhibiting the hallmark symptoms of DBD and nonclinical contrast groups. For example, in a study of 117 non-referred children (ages 7–17), Boes, Tranel, Anderson, and Nopoulos (2008) found a significant negative correlation between aggressive behaviors in boys, as measured by parent and teacher report, and the volume of the right anterior cingulate cortex. Similarly, amygdala volume was found to correlate positively with the duration of aggressive behaviors (Whittle et al., 2008). These findings have been interpreted as explaining a lessened ability to down-regulate arousal that might be responsible for aggression (Whittle et al., 2008). These differences have been related to a genetic difference. Those with the low expression of a common monoamine oxidase A (MAOA) polymorphism have been associated with more impulsive aggression in humans and animals, along with significant relative reduction in anterior cingulate cortex and amygdala volumes (Meyer-Lindenberg et al., 2006). These variations in volume were also related to lowered activation of dorsal anterior cingulate cortex during an inhibitory control task (Meyer-Lindenberg et al., 2006).

These and other findings as well as an emerging understanding of brain development suggest that the interactions between brain regions, particularly the frontal cortex and those related to emotion arousal, will be a very fertile area for further expanding developmental theory and research. Mapping such relationships to typical development and sex differences in such development also seems quite important. As noted by Marceau and Neiderhiser (2013) and Susman and Pollak (2013) there are many new methods and instruments for probing brain physiology and functioning that will allow testing new theories about how disruptive behaviors are formed. One step in doing so is articulating important questions for driving the scientific agenda. The present volume and the symposium from which it is drawn arose out of the recognition that it is important to frame the critical questions for the next steps forward in the study of DBDs. In order to do so, it is also necessary to create a framework that includes the current state of the field and allows for including the advancing understanding of disruptive behavior in the context of brain–behavior relationships.

Brain plasticity and DBDs. In addition to these physiological and functional shifts in the brain anatomy and function over the course of development that may be implicated in DBD, there are also emerging findings of a variety of pertinent genetic influences (see Marceau and Neiderhiser 2013 and Tolan, Rutter, and Dodge, 2013). Further, the emerging understanding of basic brain development and functioning has greatly increased recognition of brain plasticity, even into adulthood. In particular, studies have shown that brain development in areas thought to control judgment, self-control, and intensity of pleasure from risk-taking are still developing substantially throughout adolescence (Stuart & Steinberg, 2008). These findings highlight the extent to which disruptive behavior problems are reflective of the complex relationship between brain processes and environmental factors that affect development.

Brains in Children in Context

Developmental psychopathology theory has for 2 decades focused on how individual development is cumulative and transactional. Thus, the development of capabilities and problems both occur through a complex set of transactions between current status and the biological and contextual determinants of that current functioning and ensuing experiences (Cicchetti & Cohen, 1995; Sameroff & Mackenzie, 2003). As a result, by approaching DBDs from the perspective of brain development and function, multiple areas of scientific knowledge must be considered and integrated, including: genetics, epigenetics, and environmental influences such as individual, family, other important social relationships, schools, etc. As an example, given the emerging findings that are tracking brain function and pathophysiology related to traumatic exposures, neglect and other forms of inadequate care, as well as violence and fear inducement, understanding these sorts of environmental–biological interactions will provide needed knowledge about factors that may also contribute to DBDs; however, to take these steps will require sophistication in the incorporation of understandings about brain–behavior development within a the context of a full appreciation for the transactional development attuned to contextual variation. That view is what formed the basis for this first symposium of the Brain Research Foundation series on developmental psychopathology.

Focus of the Symposium and Organization of the Volume

Integrating brain development science with the understanding of DBD immediately raised an important issue: integration requires not only a focus on traditional topics in neurodevelopmental studies such as brain physiology and function and findings from genetics but also how to engage leading scientists in a conversation about how to apply a developmental psychopathology framework to this clinical problem. To do so require the integration of rapidly developing advances in several key areas of knowledge about DBDs with an equally rapidly advancing neurodevelopmental literature. Since there was already some degree of cross-influence between the neuroscientists and developmental scientists, the symposium created an opportunity to focus on key questions in the developmental psychopathology of disruptive behavior problems, with an eye toward the promise of greater connection between various levels of study from genetic to neurodevelopmental to person/clinical to population and contextual. That need became the opportunity for a 2-day symposium held in Chicago under the auspices of the Institute of Juvenile Research, directed at that time by symposium co-organizer Patrick Tolan and the Brain Research Foundation (then named the Children's Brain Research Foundation).

As the interest was in looking forward we framed the symposium around seven key questions:

1. What are the defining dimensions of DBDs that can best differentiate these from normative misbehavior and other forms of psychopathology and inform understanding of the development and course of DBDs?
2. What are implications of findings on the development (or lapse thereof) of empathy/conscience for socialization for understanding the development course of DBDs.
3. What are key issues in gene environment interactions models and how can recent studies inform understanding of the development and course of DBDs?
4. What are the most promising areas of study and findings about specific neurobiological processes and brain functions in understanding the development and course of DBDs.
5. What is the relation of DBD risk and presentation to gender and what are the implications of gender variations?
6. How should the multiple avenues of family influence on DBDs be understood and utilized?
7. What are the key peer and community influences on DBDs?
8. Each question formed the focus of one session, in which two experts on the topic presented a summary of the best current understanding, critical challenges, and his or her perspective on important next steps in research. Another leading expert responded to the presentations and presented his or her perspective on the same matters of critical study needs and important problems for next steps. The chapters in this volume are based on those discussions and are typically coauthored by the presenters and discussant. Each chapter thus provides a summary of the current knowledge and identifies key research issues and important challenges pertaining to the seven symposium questions. The authors also identify any overlap with or relation to other areas. Only a slice of the broad and complex field of studies relevant to each topic could be considered. Concordantly, authors were encouraged to offer expert perspectives on a scientific agenda for the area they summarized. The result is the start of a conversation about advancing study in Disruptive Behavioral Disorders as well as offer insights on specific areas.

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Chapter 2
Influences of Gene–Environment Interaction and Correlation on Disruptive Behavior in the Family Context

Kristine Marceau and Jenae M. Neiderhiser

Introduction

There are diverse developmental pathways to and among disruptive behavior disorders. As evidenced by this volume, our understanding of the development of disruptive behavior disorders has been greatly advanced through developmental strategies examining genetic, prenatal, neuroendocrine, neuroanatomical, and social influences. The current chapter focuses on advances in our understanding of the development of disruptive behavior problems that have been gained through using behavioral genetic methods, and proposes a strategy for integrating across multiple areas in order to gain a more complete understanding of the development of disruptive behaviors in children. We begin by placing the behavioral genetic work we will discuss in a larger developmental framework.

Three comprehensive, broad developmental approaches have been described for the development of disruptive behavior disorder in children: additive, interactionist, and transactional (e.g., Dodge & Petit, 2003; Kimonis & Frick, 2010). In additive models, different developmental influences work together in an aggregate way, each producing independent effects to influence trajectories of development. In interactionist models, different developmental influences produce a joint effect on development of the phenotype[1] through moderation, modifying or amplifying the influence of other developmental influences. In transactional models, different developmental factors influence each other and the phenotype of interest across development. Additive and interactionist models are combinatory, emphasizing the joint effect of previously measured "risk" or "protective" factors on an outcome.

[1] Phenotype is defined here as a measured variable of interest, for example, a measure of disruptive behavior.

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In transactional models, however, the joint effects of "risk" or "protective" factors are hypothesized to develop and change over time. Thus, in transactional models it is possible that the interrelations of different influences on behavior may change over time, or have different meaning over the course of development. Conceptually, gene–environment interplay (e.g., gene–environment correlation and interaction) best fits into the transactional approach.

Gene–environment interplay is defined here as genetic and environmental influences acting together on the development of behavior, encompassing gene–environment correlation and interaction over time. Gene–environment interplay occurs on multiple organizational levels. Environmental influences may moderate the functional roles of genes on behavior throughout development as well as at specific times, and gene variants may impact susceptibility to certain environmental influences. Even on a cellular level, gene expression may act to change internal environmental factors (i.e., hormone or neurotransmitter levels, e.g., Joffe & Cohen, 1998) which then can moderate the expression of other genes (e.g., through epigenetic mechanisms, see Meaney, 2010). On a broader scale, genes and environments work together through gene–environment correlation and interaction processes across development, and gene–environment correlation and interaction themselves may also moderate the effect of environmental influences on the development of later, more severe disruptive behaviors, particularly conduct disorder and substance abuse. On each of these organizational levels, genes and environments have transactional influences on each other and on phenotypic outcomes over the course of development. Thus, findings from behavioral genetic studies examining the role of gene–environment correlation and interaction in the developmental course of disruptive behavior problems can be considered within a transactional developmental framework.

Although an individual's genes may influence his/her behavior, the family helps to control how and when genetic influences operate. Thus, it is important to consider the role of genetic and environmental influences on the development of disruptive behavior problems within the family context. Because biological parents pass on genes (each parent shares exactly 50 % of their genes with each of their children) and provide the rearing environment for the developing child, genetic and/or environmental influences can explain parental influences on child behavior. Thus, we consider the role of genes and environments on the development of disruptive behavior problems as a family issue. Specifically, using different types of family-based designs we may begin to understand how genes and environments work together through mechanisms of gene–environment interplay including gene–environment correlation and interaction (described below). While there are limitations to each of the family-based study designs reviewed below, converging evidence from multiple study designs provides a much more nuanced picture of the development of disruptive (and other) behaviors. By considering the findings across various designs, we can be confident that the different forms of gene–environment interplay involved in the development of disruptive behavior are not mechanisms of development limited to one type of family composition, or an artifact of the particular statistics used in each type of family study. Considering behavioral

genetics as a family-based approach allows developmental behavioral geneticists to disentangle the multiple ways in which parents and children can influence each other and to consider the bidirectional effects of parent–child relationships and children's disruptive behavior problems over time.

In this chapter we will provide a brief review of the behavioral genetic approach including the current theory and methods used to investigate gene–environment interplay. We will review relevant findings from quantitative and molecular genetic research illustrating gene–environment correlation and interaction influences on the development of disruptive behavior, focusing on the role of the family environment. Finally, we offer suggestions for how findings from other models of the development of disruptive behavior can inform future research on gene–environment correlation and interaction influences on the development of disruptive behavior, and better integrate and test transactional conceptual models of the development of disruptive behavior disorders.

A Brief Overview of Behavioral Genetics

There are two broad avenues of research into how genetic and environmental influences operate to influence development: quantitative and molecular genetics. Quantitative genetic strategies take advantage of the natural quasi-experimental design of family members who vary in degree of genetic relatedness (e.g., twin, sibling, and adoption designs). Typically, quantitative genetic studies use latent modeling techniques from a broader, top down (i.e., theory to method) approach to estimate genetic and environmental influences on behavior based on quantitative genetic theory. Quantitative genetic models are built on theoretically derived assumptions, and genetic influences are operationalized as latent factors subsuming all genetic influences from structural differences in genotypes. Molecular genetic strategies use technological advances and a bottom up (i.e., method to theory) approach to examine how specific genes (or sets of genes) influence behavior. While some molecular genetic methods are hypothesis-driven, most are data-driven, as parts of or the entirety of the genome is scanned in attempt to find associations between individual genes and the phenotype of interest.

Quantitative genetics. Quantitative genetic research uses samples of families whose members share different proportions of their segregating genes. Quantitative genetic studies parse the variance in any given phenotype (e.g., disruptive behavior) into three variance components: genetic, shared, and nonshared environmental influences. First, genetic influences are derived based on quantitative genetic theory specifying the average proportion of segregating genes family members share. Using twins and siblings as an example, monozygotic (MZ) twins share 100 % of their segregating genes, dizygotic (DZ) twins and full siblings share on average 50 %, half siblings and cousin pairs whose parents are monozygotic twins share on average 25 %, and cousin pairs whose parents are dizygotic twins share on average 12.5 % of their segregating genes whereas adoptive or step siblings systematically

do not share any genes. By comparing the relative likeness of different types of siblings and/or family members for disruptive behaviors (correlations between sibling 1's disruptive behavior and sibling 2's disruptive behavior in each family, compared across sibling types), quantitative geneticists estimate the extent to which variation in genes contributes to disruptive behavior. Shared environmental influences are a latent construct representing all nongenetic influences contributing to likeness among family members. Shared environmental influences, then, are necessarily correlated 1 between siblings across all sibling types residing in the same household and 0 for related individuals not residing together (e.g., biological parents and the child they placed for adoption). Finally, nonshared environmental influences are a latent construct representing all nongenetic influences contributing to differences in family members, and are therefore uncorrelated between siblings. The estimate of nonshared environmental influences also includes error.

Together, these principles drawn from quantitative genetic theory about family similarity are used to infer genetic, shared, and nonshared environmental influences. For example, if monozygotic twins are 2 times more similar for their disruptive behaviors than dizygotic twins, genetic influences are operating, because we know that monozygotic twins share (on average) twice as many genes as dizygotic twins. However, the extent to which correlations between sibling 1 and sibling 2 disruptive behavior for monozygotic twins and dizygotic twins/full siblings are equal, or the extent to which genetically unrelated siblings are correlated for disruptive behavior suggests that shared environmental influences contribute to disruptive behaviors, but not genetic influences. The extent to which monozygotic twins are not perfectly correlated indicates the contribution of nonshared environmental influences on the phenotype.

There are several assumptions applicable to twin and sibling studies that can impact the estimates of genetic and environmental influences recovered in quantitative genetic analyses. First, quantitative genetic studies are built on the equal environments assumption: shared and nonshared environmental influences are equivalent for each sibling type. That is, monozygotic twins' environments are not more similar than genetically unrelated siblings' environments. Thus far, no systematic differences have been found negating the validity of the equal environments assumption (Loehlin & Nichols, 1976; Neiderhiser et al., 2004; Reiss, Neiderhiser, Hetherington, & Plomin, 2000).

Second, assortative mating can affect estimates of genetic and shared environmental influences. Assortative mating occurs when individuals choose their mates based on heritable characteristics for which they are alike. Thus, parents who have assortatively mated are more likely to pass on similar genetic influences to offspring. While assortative mating is generally modest for most psychological traits (e.g., Plomin, DeFries, & McClearn, 1990), there is evidence of moderate assortative mating for antisocial behavior (e.g., Du Fort, Boothroyd, Bland, Newman, & Kakuma, 2002), making this assumption less tenable in quantitative studies of disruptive behavior, at least for samples selected for high levels of disruptive behavior or focusing on extremes. It is possible, however, to test for such effects if related constructs are assessed in the parents.

The presence of assortative mating on traits involved in the intergenerational transmission of disruptive behavior disorders inflates shared environmental influences at the expense of genetic influences (because MZ and DZ twins will appear more similar, reducing the contrast in correlations that would suggest genetic influences on disruptive behavior). The inclusion of genetically unrelated siblings in quantitative genetic designs helps to attenuate this bias. Assortative mating on antisocial behavior also suggests that passive *r*GE is more likely contributing to the development of disruptive behavior disorders, thus highlighting the importance of studying gene–environment interplay especially for externalizing problems.

Molecular genetics. As noted above, molecular genetic studies examine genetic effects using a bottom up approach, starting with specific genes (either selected by a hypothesis-driven method, or using genome-wide association). Molecular genetic studies do not rely on specific sample types, but instead on the collection of DNA, and frequently require very large sample sizes. Molecular genetic studies originally sought to determine whether specific gene regions and allelic variations in genes were associated with disruptive behavior in order to determine which genes specifically contributed to latent heritability factors from quantitative genetic models. In general, several genes of the serotonin and dopamine systems have been implicated in disruptive behavior (see below), though no one particular gene has been found to explain a sizable proportion of variance on any disruptive behavior phenotypes. While some specific genes have been identified that account for variance in phenotypes of interest to the development of disruptive behavior disorders, specific genes rarely explain more than 2–3 % of the variance in any given behavioral phenotype.

Molecular genetic methods assume that the structure of DNA (i.e., which alleles are inherited) affects behavior. Developmental molecular genetic studies of humans thus far have not directly tested the role of epigenetics though there is evidence that changes in gene expression impact the development and intergenerational transmission of several phenotypes, and that gene expression can change throughout development (Meaney, 2010). Thus, in molecular genetic studies, it is assumed that individual differences in alleles carried incorporate the effects of gene expression, because the expression of different alleles would probabilistically contribute to, or eventually result in, different phenotypes. For example, the effect of methylation is essentially to "turn a gene off" or reduce the activity. If methylation varies systematically with allelic variants under study, then the effects of allelic variants are confounded with epigenetic effects. If all allele variants are systematically methylated, then no main effect of gene variant would be found (even if, when unmethylated, specific alleles were differentially correlated with behavior). Further, if methylation status also affects the phenotype, and is evenly distributed across people with each gene variant, the effect of gene variants could be washed out. In the future, studies may clarify findings from molecular genetic research by empirically disentangling effects of allele variants from epigenetic effects.

Genetic and Environmental Influences on Disruptive Behavior Problems

Univariate quantitative genetic studies consistently demonstrate that genetic influences are important for disruptive behavior problems in childhood and adolescence, though there is inconsistency in the proportion of variance explained by these genetic effects (Burt, 2009; Miles & Carey, 1997; Rhee & Waldman, 2002). Most studies indicate that the majority of variance can be explained by genetic influences with little contribution of shared environmental influences, although some reports indicate significant and sizable shared environmental influences. For example, Deater-Deckard and Plomin (1999) reported that in middle childhood, studies tend to show genetic influences account for 13–94 % of the variance in disruptive behavior problems, whereas shared environmental influences account for less than 62 % of the variance in disruptive behavior problems. This is a particularly wide range in variance estimates for both genetic and shared environmental influences, prompting developmental researchers to try to understand what causes the variability in estimates of genetic and environmental influences on disruptive behavior problems across childhood.

There are several explanations for observed differences in the relative influence of genetic and environmental contributions to disruptive behaviors across studies, including definition specificity, age, and error (see Burt, 2009; Marceau et al., 2012; Rhee & Waldman, 2002 for a discussion of these issues). The measurement of disruptive behavior problems offers yet another compelling possibility, as it is widely acknowledged that heritability estimates vary by informant (Burt, 2009). Finally, other sample-related differences (e.g., environmental adversity, Meyers & Dick, 2010) may drive differences in genetic and environmental influences on disruptive behavior problems, since estimates of genetic and environmental influences rely on variations in correlations across sibling types within a sample, and thus are particularly sample-specific.

Type of disruptive behaviors. Some systematic differences found in estimates of genetic and environmental influences across studies stem from differences in the specific disruptive behaviors assessed (i.e., aggression vs. delinquency; and conduct disorder vs. hyperactivity or oppositional defiant disorders) (e.g., Dick, Viken, Kaprio, Pulkkinen, & Rose, 2005; van der Valk, Verhulst, Neale, & Boomsma, 1998). When nonaggressive and aggressive antisocial behaviors were considered separately in a recent meta-analysis, genetic influences accounted for approximately half of the variance in nonaggressive antisocial behavior (48 %) with the remaining being split between shared and nonshared environmental influences (Burt, 2009). For aggression, however, additive genetic influences and nonshared environmental influences accounted for most of the variance (65 %), leaving little explained by shared environmental influences (5 %). Thus, shared environmental influences explained more of the etiology of nonaggressive disruptive behaviors than it did of aggressive disruptive behaviors (see also Rutter et al., 1990; van den Oord, Boomsma, & Verhulst, 1994).

There is evidence of an underlying genetic factor common to multiple externalizing problems including attention deficit hyperactivity, oppositional defiant, and conduct disorders in boys and girls in middle childhood and adolescence (e.g., Eaves et al., 2000; Tuvblad, Zheng, Raine, & Baker, 2009). This underlying genetic factor common to different externalizing disorders suggests a common genetic liability to different disruptive behavior disorders. This genetic liability to externalizing-type disorders is supported by adult literature showing that across psychiatric disorders, there appear to be two primary genetic liabilities across traits: one underlying internalizing-type disorders (i.e., anxiety, depression, and phobias) and a distinct genetic liability underlying externalizing-type disorders (i.e., substance use, antisocial, and conduct disorders; e.g., Kendler, Prescott, Myers, & Neale, 2003). It appears, based on the findings reviewed above, that the genetic liability common to externalizing-type disruptive behavior disorders is observable even in childhood. One study suggests that there is a common genetic liability underlying symptoms of both internalizing and externalizing disorders in children (Lahey, Van Hulle, Singh, Waldman, & Rathouz, 2011), though more studies are needed to confirm this finding and to understand the developmental progression of common and unique genetic liabilities to multiple types of problems. Further, these findings suggest that environmental influences are particularly important for differences in the presentation of disruptive behavior disorder (i.e., which specific disorder a child is diagnosed as having).

Age. A second source of systematic differences found in estimates of genetic and environmental influences across studies is age, or developmental change within the child. Overall, most studies have shown that genetic and nonshared environmental influences increase, whereas shared environmental influences decrease, from adolescence to adulthood (i.e., as individuals mature and widen social circles beyond the home; Miles & Carey, 1997). In contrast, Rhee and Waldman (2002) showed different patterns of estimates of genetic and environmental influences using age groupings. Comparing "childhood" vs. "adolescence" vs. "adulthood," findings showed that both genetic and shared environmental influences decreased with age, whereas nonshared environmental influences increased. This inconsistency may be caused by the use of very wide age ranges in each group in Rhee and Waldman (2002) (i.e., "childhood" included samples with a mean age of 2 as well as samples with a mean age of 10).

A more recent meta-analysis clarified how age affects the relative genetic and environmental influences on externalizing problems by narrowing the age ranges of comparison groups and considering aggressive and nonaggressive externalizing behaviors separately. Burt's (2009) meta-analysis grouped youth into three age groups: 1–5, 6–10, and 11–18. Results indicated that genetic and environmental influences on aggressive vs. nonaggressive behaviors did not differ in early and middle childhood, but were pronounced by adolescence (i.e., genetic influences were stronger for aggression, whereas shared environmental influences were stronger for rule-breaking). Moreover, genetic influences on aggression increased with age while shared environmental influences decreased, whereas genetic influences on rule-breaking decreased with age and shared environmental influences remained stable (Burt, 2009). Generally, findings suggest that there are age-related changes in

genetic and environmental influences on externalizing problems across childhood and adolescence. For aggressive externalizing behaviors, genetic influences can be expected to increase across adolescence, but when externalizing behaviors are primarily nonaggressive environmental influences may become more salient across adolescence.

Consistent with the cross-sectional work reviewed above, longitudinal studies of disruptive behavior have shown that genetic influences make an important contribution to the stability of disruptive behaviors but that environmental influences exert primarily age-specific influences on disruptive behaviors (e.g., Petitclerc, Boivin, Dionne, Perusse, & Tremblay, 2011). For example, from 20 to 54 months of age, genetic influences accounted for an underlying liability in disregard for rules across early childhood, while environmental influences on disregard for rules were largely age-specific. Further, genetic influences accounted for intercept levels of disregard for rules, and there were small trends for increases in nonshared environmental influences on disregard for rules over time (Petitclerc et al., 2011). However, across childhood (age 3–12 years) genetic and shared environmental influences both contributed to stability in aggressive behaviors (Van Beijsterveldt, Bartels, Hudziak, & Boomsma, 2003). Genetic and shared environmental influences both contributed to the stability in conduct disorder and oppositional defiant disorder symptoms (examined together) from age 11 to 14 (Burt, McGue, Krueger, & Iacono, 2005), whereas solely genetic influences accounted for stability in antisocial behavior from middle to late adolescence (Neiderhiser, Reiss, Hetherington, & Plomin, 1999). Together, these findings suggest that genetic influences play an important role in the stability of several different types of disruptive behaviors from early childhood through adolescence. However, during middle childhood and early adolescence, shared environmental influences also exert an influence on the stability of disruptive behaviors.

Middle childhood appears to be a particularly important age to examine genetic and environmental influences on disruptive behavior problems, because early-onset disorders are generally defined as having severe problems before the age of 10 (e.g., Zahn-Waxler, Shirtcliff, & Marceau, 2008). Further, the transition from middle childhood to adolescence appears to coincide with meaningful shifts in genetic and environmental influences. For example, shared environmental influences on observed externalizing behavior were greater in two samples of children in middle childhood, whereas genetic influences on observed externalizing behavior were greater in a sample of adolescents (Marceau et al., 2012). Further, Burt and Neiderhiser (2009) showed that age moderates the genetic and environmental influences on delinquent behaviors specifically, in that genetic influences increase dramatically from age 10 to 18, whereas shared environmental influences decreased.

In summary, there are genetic and environmental influences on disruptive behavior problems in childhood and adolescence. While the relative influences of genes and environments differ for distinct disruptive behavior disorders, there is evidence of an underlying genetic vulnerability common to multiple disruptive behavior problems and to multiple types of disruptive behavior problems over time. There is also evidence that the relative influences of genetic and environmental influences on

externalizing problems change across childhood and adolescence. Taken together, these findings suggest that it is essential to take a developmental perspective when studying disruptive behavior problems. Further, it is important to understand the mechanisms by which changing genetic and environmental influences impact the development of disruptive behavior problems.

Genes cannot act to change behavior without working through biological functions within the individual and the function of genes is often moderated by environmental influences. Thus, molecular genetic studies searching for main effects of specific gene variants fall short of testing for mechanisms of development. In quantitative genetic studies, while parsing variance into discrete categories made great impact on how researchers think about development (McGue, 2010) simply quantifying latent genetic and environmental influences hasn't satisfied developmental researchers. Considering theories of development that emphasize transactional influences among genetic, biological, and environmental influences, simply parsing the variance in phenotypes and the covariance across phenotypes, falls short of testing developmental mechanisms. Thus, studies using behavioral genetic approaches have moved beyond measures of genetic and environmental influences in the development of disruptive behavior (see Moffitt, 2005 for review). The remainder of this chapter focuses on this next generation of behavioral genetic studies seeking to understand how genetic and environmental influences work together in the development of disruptive behavior problems, rather than those that examine the relative influence of genes and environments.

Gene–Environment Interplay

An emerging body of research demonstrates how genes and family environmental factors work together to influence child and adolescent development (Horwitz, Marceau, & Neiderhiser, 2011). Broadly, the goals of research investigating gene–environment interplay are to understand how genetic influences of both parents and children operate through environmental mechanisms, and to understand how genetic factors may moderate the effects of frequently studied "environmental" influences. Harnessing the power of genetically informed, family-based designs, researchers have made great progress on understanding how genes and environments work together in the development of disruptive behavior.

Conceptualizing Gene–Environment Correlation and Interaction

The two most often examined forms of gene–environment interplay are genotype–environment correlation and genotype × environment interaction. Genotype–environment correlation (*r*GE) refers to correlations between genes

and environments. Typically, three types of *r*GE are described: passive, active, and evocative (Plomin, DeFries, & Loehlin, 1977; Scarr & McCartney, 1983). Passive *r*GE occurs when parents pass on genes to their offspring and also provide an environment correlated with the heritable characteristics of the offspring. This type of *r*GE would be expected to occur more commonly during infancy and early childhood (i.e., before children can actively choose or influence their environment) although there is little support for this. For example, there was some evidence of passive *r*GE contributing to the association between maternal criticism and adolescent externalizing problems (Narusyte et al., 2011). Active *r*GE occurs when individuals seek out environments correlated with their heritable characteristics, whereas evocative *r*GE occurs when individuals evoke responses from the environment because of their heritable characteristics. These different forms of *r*GE are not mutually exclusive, and may simultaneously affect expression of a phenotype (e.g., Narusyte et al., 2011; Neiderhiser, Reiss, Lichtenstein, Spotts, & Ganiban, 2007; Neiderhiser et al., 2004).

The other commonly studied form of gene–environment interplay is genotype × environment interaction (G × E). G × E tests whether genetic factors moderate the influence of environmental factors, or whether environmental factors moderate the influence of genes on behavior. G × E and *r*GE are conceptually independent, but likely occur simultaneously in development. A number of different behavioral genetic designs have been used to examine the role of *r*GE and G × E. *r*GE and G × E have been investigated using latent genetic and environmental constructs (e.g., Narusyte et al., 2008, 2011; Neiderhiser et al., 2004, 2007; Tuvblad, Grann, & Lichtenstein, 2006), or using specific, measured genetic and environmental influences (see review below).

It is important to note that the "E" in *r*GE and G × E is not a truly environmental factor—in fact it is highly unlikely that there are any truly environmental factors. Commonly studied "environmental" influences on behavior include childhood stressors like abuse, availability and access to drugs and alcohol, negative peer groups, religiosity, parental monitoring, and harsh parenting (Meyers & Dick, 2010). The confounding of genes in these environmental influences is especially problematic for family environmental influences like parenting. Especially considering behavioral genetics as a family-based approach, family environmental influences are of primary interest as a predictor of child behavior problems. An ongoing goal of behavioral geneticists should be (and for many, is) better and more nuanced measurement of environmental factors, like parenting. Poor measurement is a limitation common to behavioral genetic studies generally, because the number of participants needed for adequate power for behavioral genetic analyses is so high that the cost of research limits the feasibility of some measures. This limitation has likely contributed to the mixed findings found across behavioral genetic studies. Behavioral genetic studies can be used not only to identify *r*GE and G × E operating during development but also to clarify the phenotypes of interest (Moffitt, 2005).

Evidence of Gene–Environment Correlation and Interaction

Quantitative genetic studies. A wide body of quantitative genetic research using twin/sibling studies has shown that genetic influences account for a large proportion of the correlation between negative parenting and child or adolescent adjustment (Burt et al., 2005; Horwitz et al., 2011; Pike, McGuire, Hetherington, & Reiss, 1996; Reiss et al., 2000). Broadly, this suggests the influence of *r*GE between the environmental factor "negative parenting" and influences of the children's genes on externalizing problems.

One study attempted to examine genetic influences on negative parenting and externalizing behavior longitudinally by also considering a child temperamental mediator of this association. Specifically, genetic influences on the association between parental criticism and adolescent antisocial behavior were partially explained by adolescent aggressive temperament 3 years earlier (Narusyte, Andershed, Neiderhiser, & Lichtenstein, 2007), suggesting that adolescents' aggressive temperament evokes negative parenting, which, in turn, shapes adolescents' development of antisocial behavior. Further, genetic influences accounted for the correlation between environmental risk (e.g., negative life events) and externalizing behavior, suggesting gene–environment correlation (Button, Lau, Maughan, & Eley, 2008).

Other longitudinal studies have found evidence for genetic influences on the associations between parenting and adolescent antisocial behavior over time. Specifically, findings from a cross-lagged longitudinal design suggested roughly equal genetic, shared environmental, and nonshared environmental influences explaining the association between parent–child conflict in early adolescence and youth externalizing behavior in mid-adolescence (Burt et al., 2005). An earlier study also found that parent–child negativity in middle adolescence contributed to change in antisocial behavior from middle to later adolescence and this association was explained by primarily genetic influences (Neiderhiser et al., 1999). These findings, taken together, suggest *r*GE, showing that the environmental risk (parent–child conflict) is associated with the phenotype (externalizing behavior) because of the adolescents' genetic influences. Most likely, this *r*GE represents evocative *r*GE, as the power to detect evocative *r*GE lies in the influence of children's genes. However, in twin and sibling only studies, *r*GE is inferred, not tested, and it is not possible to determine which type of *r*GE is driving the associations, or if passive and evocative *r*GE are occurring simultaneously.

Studies of twins who are parents can help to clarify the direction of effects and presence of *r*GE. For example, intergenerational transmission of conduct problems was attributable to direct environmental influence for boys, but parents' genetic influences and environmental risk were confounded (suggesting passive *r*GE) in girls (D'Onofrio et al., 2007). However, the transmission of alcohol use disorder in twin parents to externalizing problems (risk for alcohol use disorder) in child and adolescent offspring was entirely mediated by genetic influences (Waldron, Martin, Heath, & Phil, 2009). Several studies of parents who are twins suggest that

parenting is influenced by parents' genotype and environmental influences (e.g., Kendler, 1996; Losoya, Callor, Rowe, & Goldsmith, 1997; Narusyte et al., 2008; Neiderhiser et al., 2004, 2007). This evidence supports earlier findings that parenting may impact offspring development via passive *r*GE while both evocative *r*GE and causal, environmental mechanisms may also be operating. Samples with parents who are twins are, however, limited in power to test for evocative *r*GE because of limited variability in genetic relatedness of offspring (genes are correlated on average 0.25 for children of MZ twins, 0.125 for children of DZ twins). Nonetheless, this strategy has permitted a careful examination of the role of passive *r*GE in influencing child and adolescent outcomes.

In response to the limitations of each type of twin study, and to enable passive and evocative *r*GE to be disentangled and the direct effects of environment to be estimated, the extended children-of-twins (ECOT) design (Narusyte et al., 2008, 2011) was developed. The ECOT design uses samples of twins who are children (classic twin design) and twins who are parents (children of twins design) to examine how genetic and environmental influences of the children and of the parents influence phenotypes of interest. These two designs are analyzed together in the same model. The ECOT design can distinguish between passive and evocative *r*GE operating within families as well as estimate direct environmental effects on child behavior. The power to detect evocative *r*GE lies in the child-based design, which takes advantage of sibling types of differing genetic relatedness, thus estimating the influence of children's own genes on their behavior and on their parents' behavior. Similarly, the power to detect passive *r*GE lies in the parent-based design, which tests the influence of parents' genes and environmental influences on their behavior and their children's behavior. By analyzing both sample types together, the ECOT model distinguishes between direct environmental influences, passive, and evocative *r*GE (see Fig. 2.1).

The ECOT design has shown that the association between adolescent externalizing problems and maternal criticism arises because of evocative *r*GE, and to a lesser extent passive *r*GE, but that paternal criticism has a direct environmental effect exacerbating adolescents' externalizing problems (Narusyte et al., 2011). Thus, while parental criticism is associated with adolescent's externalizing problems, these findings suggest that the nature of the associations differs for relationships with mothers vs. fathers. Paternal criticism appears to be an environmental risk factor for adolescent externalizing problems, so the direction of effects is such that fathering impacts child behavior. However, mothers respond to their children's externalizing problems with criticism, so the direction of effects is such that child behavior impacts mothering. In addition to child effects on mothering, there is a second mechanism for the association between maternal criticism and externalizing problems: mothers also pass on a critical parenting environment consistent with her genes—both of which increase the probability that her adolescent will engage in externalizing behaviors. However, another study suggested that the association between parental negativity and adolescent externalizing problems was explained entirely by evocative rGE for mothers and fathers (Marceau, Horwitz et al., in press). Therefore, findings from ECOT designs suggest that different types of parenting behaviors may be associated with adolescent externalizing problems through different mechanisms.

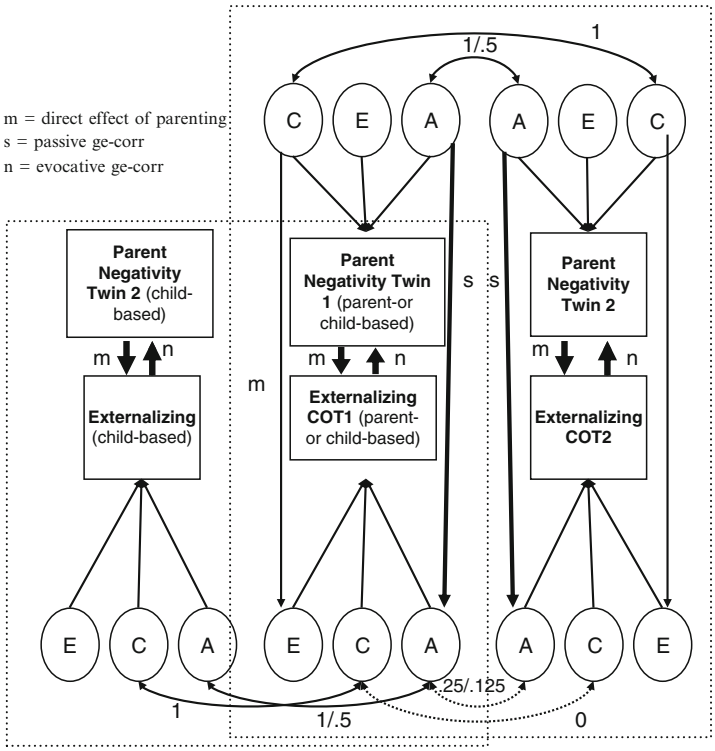

Fig. 2.1 Extended children of twins (ECOT) model. This is a representation of the ECOT model testing the association between parent negativity and child externalizing problems. The lower box on the *left* represents the classic twin sample, while the larger box on the *right* represents the children of twins sample. *M* is a measure of the direct effect of parenting, while *s* indicates passive *r*GE and *n* indicates evocative *r*GE. *A* are additive genetic effects (correlated 1 for MZ twins, 0.5 for DZ twins, etc., see text), *C* are shared environmental influences, and *E* are nonshared environmental influences

Converging evidence from family-based studies of twin children, twin parents, and a combination of both twin children and parents suggest that both evocative and passive *r*GE underlie the association between negative parenting behaviors and adolescent disruptive behaviors. In light of the studies using the ECOT design, and other studies suggesting differences in *r*GE mechanisms that underlie parenting behaviors (e.g., Neiderhiser et al., 2004, 2007), future quantitative genetic studies should also examine sex differences of both parents and children in the mechanisms underlying the association between parenting and externalizing behavior.

Because estimates of genetic and environmental influences are latent in twin and sibling studies, twin/sibling studies cannot test whether genetic predispositions moderate the influence of environmental factors (gene–environment interaction). However, these studies can test whether genetically and environmentally influenced constructs moderate the influence of genes on behavior. Both parental negativity

and warmth have been found to moderate genetic influences on aggressive and nonaggressive forms of adolescent antisocial behavior (Feinberg, Button, Neiderhiser, Reiss, & Hetherington, 2007) such that genetic influences were greater for adolescent antisocial behavior when parenting behaviors were more negative or less warm. Further, the magnitude of the influence of genetic risk on externalizing behavior was found to be contextually dependent, even after controlling for gene–environment correlation, suggesting G×E (Button et al., 2008). Generally, findings from twin studies testing moderation of genetic influences on externalizing behavior by putatively environmental influences suggest that genetic influences on externalizing spectrum disorders are greater in the presence of environmental adversity (see Meyers & Dick, 2010; Tuvblad et al., 2006).

Adoption designs also use genetic relatedness of family members to disentangle genetic from environmental influences on child and/or adolescent behavior. Adoption designs use the genetic (un)relatedness of family members by taking advantage of the natural break of the confound between genetic and environmental influences provided by parents to offspring. However, they differ from the quantitative genetic studies described above in the way that genetic risk is inferred—genetic influences are operationalized as birth parent characteristics that influence children's behavior when they are not reared by the birth parent. Adoption designs have also been extended to study differences between adoptive children and biological children raised together. Any similarity between adoptive and biological children must be due to shared environmental influences because they do not share genes, but do share the same rearing environment. The extent to which parents and their biological children are more similar on the phenotype than parents and their adoptive children are indicates genetic influences on the phenotype. Finally, the extent to which characteristics of the biological parent and adoptive parent are correlated indicates evocative *r*GE, because the genetic risk passed from biological parents to children is evoking negative responses from the parent.

Generally, findings from adoption studies suggest that evocative *r*GE explains associations between negative family environmental influences (e.g., parenting/parent–child conflict) and youths' disruptive behavior symptoms (e.g., Deater-Decker & O'Connor, 2000; Ge et al., 1996; Narusyte et al., 2007; O'Connor, Deater-Deckard, Fulker, Rutter, & Plomin, 1998). For example, birth mothers' antisocial behavior (genetic risk) was associated with adoptive parents' negative control (environment) in middle childhood and early adolescence. Children at genetic risk for antisocial behavior consistently received more negative, controlling parenting behaviors, suggesting evocative *r*GE. Birth parents' antisocial behavior did not completely explain the association between adoptive parents' negative control and children's externalizing problems, however, suggesting that negative, controlling parenting may also be an independent environmental influence on children's externalizing problems (O'Connor et al., 1998).

Findings comparing adopted and biological children of the same parents also suggest that passive *r*GE operates in infancy, early and middle childhood, and adolescence (Braungart-Rieker, Rende, Plomin, DeFries, & Fulker, 1995; McGue, Sharma, & Benson, 1996). However, adoption studies comparing adolescent

antisocial behavior of biological and adopted children in the same families suggest that shared environmental influences contribute to stability of antisocial behavior (Burt, McGue, & Iacono, 2010), and that maternal depression represents a shared environmental risk for antisocial behavior (maternal depression had the same effect on biological and genetically unrelated offspring; Tully, Iacono, & McGue, 2008). Also in adolescence, parent–child conflict predicted conduct problems, but conduct problems did not predict later parent–child conflict in a sample of adoptive families, suggesting the role of shared environmental influences in the development of conduct disorder, rather than evocative *r*GE (Klahr, McGue, Iacono, & Burt, 2011).

Thus, like findings from twin and sibling studies, adoption studies confirm that there are multiple mechanisms explaining the association between negative parenting and disruptive behavior in childhood. In many family types (families where children are twins, or the children of twins, or adopted, or where adopted and biological offspring are reared in the same home by the same parents), evocative *r*GE plays a particularly prominent role in the development of disruptive behavior, and in the associations between parenting and disruptive behavior, though there has been some evidence that passive *r*GE also operates in the development of disruptive behavior problems. Further, in each of these family-based study designs, parenting—particularly fathering—has been shown to exert a purely environmental influence on disruptive behavior. More studies are required to understand whether there are gender differences in these associations, and whether there are differences in the associations between mothering vs. fathering in adoption studies.

G×E can also be tested using adoption designs. The classic example is that youth with biological parents who have psychopathology, and adoptive parents with psychopathology are much more likely to develop psychopathology than youth who have either biological or adoptive parents with psychopathology (Cadoret, Cain, & Crowe, 1983; see Reiss & Leve, 2007). Generally, adoption studies have shown that the confluence of both genetic (biological parent psychopathology) and environmental risk factors (harsh or negative parenting or family environments, adoptive parent or sibling psychopathology) confers an additional, interactive risk for developing disruptive behavior disorders in childhood and adolescence (Reiss & Leve, 2007).

There is evidence of G×E in the development of externalizing behavior even in infancy and toddlerhood. For example, birth parents' substance dependence and antisocial behavior predicted higher levels of novelty seeking during a frustration task (an early predictor of externalizing behavior) in 9-month-olds only when adoptive parents also had higher levels of depressive and anxiety symptoms (Leve et al., 2010). Among 18-month-olds, marital instability between adoptive parents earlier in infancy predicted elevated levels of toddlers' anger and frustration only among toddlers whose birth mothers reported high levels of anger and frustration (Rhoades et al., 2011). In summary, studies using quantitative genetic designs including twin and adoption designs suggest that positive and negative aspects of parenting and the development of disruptive behaviors are linked through passive and evocative *r*GE as well as through G×E.

Molecular genetic studies. Molecular genetic analyses can be applied to any type of study if DNA is collected. In molecular genetic studies, genetic influences are measured, not estimated. Molecular genetic studies have examined genes associated with behavior, as well as genes that interact with environmental influences on behavior. There are three overarching approaches to finding specific genes to use in measured *r*GE and G×E studies: candidate gene approach, linkage, and association studies (see Meyers & Dick, 2010; Plomin & Rutter, 1998 for review). The candidate gene approach is hypothesis-driven: specific genes are chosen for study because of hypothesized or known biologic functions influencing the development of the phenotype.

Linkage and association studies are data-driven. Using advances in technology, large portions of the genome are scanned and processed in order to identify regions of the genome that are associated with the phenotype of interest. Linkage studies use this technology by comparing frequencies of alleles across similarly affected family members. Linkage studies work well for identifying single regions or genes that impact a phenotype, but are not optimal for finding multiple genes implicated in a single phenotype. Because disruptive behavior problems, like most complex phenotypes, are hypothesized to be influenced by multiple genes, genome-wide association studies (GWAS) are thought to be better suited to identify specific genes influencing disruptive behavior. GWAS have the added benefit over linkage studies of scanning the entire genome, instead of distinct regions, and have more power to detect subtle effects of specific genes on the phenotype of interest. An association with a specific gene variant and the phenotype of interest indicates *r*GE, while an interaction between a specific gene variant and some measured environmental factor predicting disruptive behavior problems indicates G×E. Generally, molecular genetic studies of disruptive behavior have focused on G×E effects.

Specific genes from several systems (e.g., serotonergic, dopaminergic, GABA) have been implicated in disruptive behavior. Here, we focus primarily on candidate genes of the serotonergic system. We note, however, that this is not the only systems implicated in G×E studies of the development of DBD. For example, studies have suggested that genes of the dopaminergic system (e.g., DRD2, DRD4, DAT1, COMT) interact with environmental influences (e.g., parenting, negative life events; Brennan et al., 2011; Creemers et al., 2011; Kahn, Khoury, Nichols, & Lanphear, 2003; Zai et al., 2011) in the development of disruptive behavior problems. GABRA2 (a receptor gene for GABA implicated originally in adult alcoholism) has also been associated with externalizing and alcohol use problems in adolescents (see Meyers & Dick, 2010). Further, parental monitoring may buffer the association between specific GABRA2 alleles and externalizing problems (Dick et al., 2007).

The serotonergic system, most often examined by considering a common polymorphism in the serotonin transporter receptor gene (5HTTLPR), is hypothesized to affect behavior through modulation of the stress response system (see Caspi, Hariri, Holmes, Uher, & Moffitt, 2010). Thus, specific alleles of 5HTTLPR may create a sensitivity to environmental influences in youth (though evidence is mixed as to which alleles may confer "risk"). If the child also experiences negative environmental influences, they are more likely to develop behavior problems. Though

5HTTLPR has received more attention in the development of depression, the same mechanism is thought to apply to the development of disruptive behavior problems. For example, youth had increased externalizing behaviors if they had the 5HTTLPR long allele variant (LL) and antisocial biological parents, or if they had one or more short alleles (SS/SL) and biological parents with alcoholism (Cadoret et al., 2003). 5HTTLPR alleles coding for high and low activity vs. intermediate serotonin transporter activity interacted with self-blame of interparental conflict to predict attention deficit hyperactivity disorder symptoms (Nikolas, Friderici, Waldman, Jernigan, & Nigg, 2010).

MAOA is a gene involved in the degradation of several neurotransmitters, including serotonin. Thus low-activity alleles of MAOA contribute to extra serotonin, and are thought to operate through a mechanism similar to variants of 5HTTLPR increasing the activity of serotonin receptors (both result in more serotonin, active for longer periods of time in the system). The interaction of specific alleles of MAOA and maternal disengagement has been shown to predict serious and violent delinquency (Beaver, DeLisi, Wright, & Vaughn, 2009). Further, low-activity alleles of MAOA interacted with sexual abuse and harsh parenting to predict externalizing disorders in young adulthood (Derringer, Krueger, Irons, & Iacono, 2010), and child maltreatment to predict childhood antisocial behavior (Caspi et al., 2002) especially in boys (Kim-Cohen et al., 2006). Thus, specific alleles of genes implicated in regulating the activity of several neurotransmitters have been implicated in the development of disruptive behavior problems.

Though less frequently reported, molecular genetic studies can also detect *r*GE. Applicable to the development of disruptive behavior in children, 5HTTLPR and OXTR (an oxytocin receptor gene) alleles in parents were associated with their observed parenting (Bakermans-Kranenburg & van IJzendoorn, 2008), evidencing passive *r*GE. However, most studies investigating reporting specific gene–environment correlations have assessed adults (see Jaffee & Price, 2007). While *r*GE and G×E are modeled separately, they are not necessarily independent. Including *r*GE in molecular genetic studies of G×E is particularly important because failing to do so biases results of G×E (Jaffee & Price, 2007). In summary, studies using a molecular genetic approach have identified several specific gene–environment interactions important in the development of disruptive behavior disorder. While the role of passive *r*GE has been demonstrated, more research is needed to understand how *r*GE operates at the level of specific genes in the development of disruptive behavior disorders.

Bridging Quantitative and Molecular Genetic Findings

A major criticism of behavioral genetics is the vast difference between top down (quantitative) and bottom up (molecular) approaches to understanding genetic influences on behavior. Many quantitative genetic studies have posited that finding increased genetic influences on a phenotype identifies that phenotype as appropriate

for molecular genetic studies. However, molecular genetics can also help inform/interpret quantitative genetic findings. An obvious limitation of twin studies is the "black box" argument: detecting genetic influence on a phenotype does not tell us which genes are influencing behavior, or how. Findings from molecular genetic studies can be used to inform what types of genes are likely subsumed in a latent genetic factor. The mismatch between the percent of variance explained by specific genes and attributable to a latent genetic factor also indicates that taking a specific, single gene approach does not come close to explaining how genetic influences impact behavior. We are still developing a framework for integrating these different conceptualizations and approaches to studying how genetic and environmental influences are interrelated and collectively affect behavioral development.

Very recently, advances in analytic methods for examining GWAS/molecular genetic data show promise for reconciling differences in genetic and environmental influences obtained through molecular and quantitative genetic methods. Using GWAS to compound the influence of all measured genes in a large sample, the total genetic influence on intelligence was estimated at 40–50 %, which is quite similar to the 60 % typically reported by quantitative genetic studies (Davies et al., 2011). This study suggests that examining poly-gene correlations and interactions is an important future direction for molecular genetic studies of individual's characteristics, and should be applied to the development of disruptive behaviors in the future.

Even on a smaller scale, for investigators without access to GWAS data but who can collect DNA, investigating poly-gene correlations and interactions (e.g., Schmidt, Fox, & Hamer, 2007) and *r*GE and G×E together is a logical next step to bridging quantitative and molecular genetic findings. The idea of experience–expectant plasticity (see Lenroot & Giedd, 2011) and the concept of plasticity genes (Belsky et al., 2009) have gained traction recently. That is, certain clusters of genes may together exert influence on the individual's sensitivity to environmental influences and context (i.e., creating a biological sensitivity to context; e.g., Ellis, Jackson, & Boyce, 2006, or differential susceptibility to the environment; Belsky, Bakermans-Kranenburg, & Van IJzendoorn, 2007). Generally, findings reviewed here do support these hypotheses. For example, both 5-HTT and MAOA have been implicated in resilience in addition to risk for disruptive behavior (see Kim-Cohen & Gold, 2009 for review). Thus, examining gene variants with similar effects that contribute to both risk and resiliency in the same model testing gene–gene and gene–environment interactions and correlations will advance our understanding of the role of genes in behavioral development. However, studies testing different forms of genetic influences on plasticity must also consider the timing of gene expression, resulting in sensitive periods of development for different types of internal and external environmental influences (e.g., Lenroot & Giedd, 2011).

What do behavioral genetic studies tell us about development? While linkage studies and GWAS are useful for detecting genes associated with outcomes, these studies do not help us to understand the process by which behavior develops. Likewise, non-longitudinal quantitative genetic studies also cannot speak to the relative influences of genetic and environmental influences across development.

In order to be of interest to developmentalists, genes must have a functional effect on a biological process that eventually reaches the brain to affect the behaviors and development of the individual. Indeed, a principle of developmental biology is that the only way genes may influence a phenotype is through interaction with the environment (e.g., Meaney, 2010).

Behavioral genetic approaches have more recently been applied by developmentalists to inform research on the mechanisms of behavioral development. Behavioral genetic designs are particularly useful for detecting environmental mechanisms (Moffitt, 2005). For example, using longitudinal quantitative genetic studies, researchers have discovered that genetic influences on a phenotype can change with age. While this may reflect measurement differences at different ages, it is unlikely that drastic changes in proportions of genetic and environmental influences on a phenotype over time are due only to measurement error (and this potential explanation can be tested within longitudinal quantitative genetic models). Instead, it is more likely that longitudinal quantitative genetic studies pick up on developmental changes in the importance of genes, operating through *r*GE and G×E. Quantitative genetic studies are often assumed to be a purely structural measure of genetic influences. That is, genetic influences are estimated by comparing phenotypes in different groups with different average percentages of shared differentiating genes. If quantitative genetic studies were purely structural, then the unstandardized variance for the genetic influence would remain constant across development. Because unstandardized variance estimates do change across time within the same sample, findings from quantitative genetic studies must be driven by functional differences in the genes shared by twins and siblings. However, twin studies cannot tell us which genes twins and siblings do or do not share act to impact behavior.

From a molecular genetic perspective, the candidate gene approach has potential to inform developmental research because genes are selected using a hypothesized biologic mechanism. However, the candidate gene approach is limited by our understanding of how biological processes impact behavior. Thus far, with little collaboration between biologists and behavioral geneticists in psychology-related fields, the potential of the candidate gene approach is likely far from realized.

Susman and Pollak (Chap. 3) review neuroanatomical and neuroendocrine influences on the development of disruptive behavior disorders. These studies have helped to identify potential candidate genes for use in G×E studies (e.g., 5-HTT). Single gene and single gene by specific environmental interaction influences have predicted only small amounts of the variance in disruptive behaviors (e.g., Plomin & Rutter, 1998). However, applying the candidate gene approach based on findings from other biological research (i.e., neuroanatomical and neuroendocrine factors) will help us to identify other specific genes likely to be involved, albeit not necessarily in a direct "main effect" way. Building on the substantial advances in our understanding of the development of disruptive behavior in somewhat disparate fields exemplified in this volume, it is now time to work towards integrative approaches to examining the development of disruptive behavior problems.

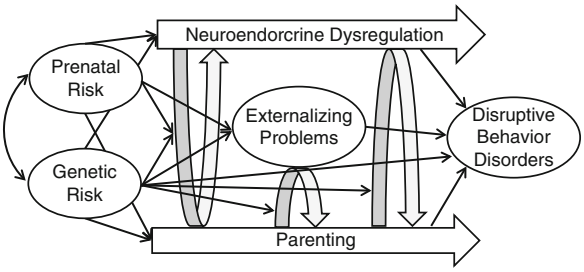

Fig. 2.2 An integrative transactional developmental model of the development of disruptive behavior disorders. This is a proposed transactional model in which genetic, prenatal, and neuroendocrine risks are considered in conjunction with parenting over time in the development of disruptive behavior disorders. *Thick arrows* represent the development of neuroendocrine dysregulation and parenting over time (across childhood and adolescence), and the transactional nature of this co-development. Genetic influences are not only present for each construct but also predict the nature of how transactions among other constructs occur and develop. This is an example of how multiple biological and environmental risk factors can be considered together in a transactional model, and tested using genetically informed study designs

As an example, in Fig. 2.2 we present a conceptual model of a comprehensive, transactional approach for the development of disruptive behavior problems. Genetic risk is an important component in the model, influencing prenatal risk, the development of neuroendocrine dysregulation and parenting over time, transactions between parenting and neuroendocrine dysregulation over time, early externalizing problems, transactions between parenting and earlier externalizing problems over time, and ultimately disruptive behavior disorders.

Existing evidence supports parts of this model, though to date important components remain untested. For example, there are genetic influences on externalizing behavior in childhood and adolescence (e.g., Burt, 2009; Rhee & Waldman, 2002), and both parents' and children's genes influence parenting (e.g., Neiderhiser et al., 2004, 2007). The studies reviewed above provide evidence for how genes and environments influence associations between parenting and externalizing behavior across adolescence (e.g., Burt et al., 2005; Narusyte et al., 2011; Neiderhiser et al., 1999). Poor emotional health in mothers prenatally is associated with disruptive behavior disorder and substance use during pregnancy is associated with offspring risk for substance use disorders (e.g., Allen, Lewinsohn, & Seeley, 1998), indicating the role of prenatal risk in the development of disruptive behavior problems. There is also some evidence that genetic influences are associated with prenatal risk for early externalizing problems (e.g., Marceau, Hajal et al., in press; Pemberton et al., 2010). A few studies have shown genetic influences on neuroendocrine dysregulation (e.g., Bartels, Van den Berg, Sluyter, Boomsma, & de Geus, 2003; Wüst, Federenko, Hellhammer, & Kirschbaum, 2000). Prenatal risk factors (maternal stress and mental health problems) predict offspring neuroendocrine dysregulation (e.g., Wadhwa et al., 2001). The role of

neuroendocrine regulation in the development of disruptive behavior problems is explicated in Chap. 3 of Susman and Pollak.

Evidence from studies investigating biological sensitivity to context suggests that both genetic influences and cortisol dysregulation may serve as biological risk factors for the development of disruptive behavior problems (e.g., Belsky et al., 2007; Ellis et al., 2006). However, these studies have not yet taken the next step to apply quantitative genetic methods to understanding how neuroanatomical and neuroendocrine processes are related to behavior. Thus, there is a call for more comprehensive models of the development of disruptive behavior that incorporates multiple biological factors and social/environmental factors to characterize and predict the development of disruptive behavior disorders.

By including measures of hormones in different genetically informed designs, this model could be tested using measured genetic risk, either using combinations of candidate genes coding for stress and sex hormones, parenting, and externalizing problems (i.e., molecular genetic studies), or inferred through birth parent characteristics of externalizing psychopathology and/or neuroendocrine dysregulation (i.e., adoption design) or both. This model could also be applied in longitudinal, multivariate quantitative genetic studies. Including two distinct, but related biologic mechanisms (in this example, genes and hormones) gives us information using measured genetic influences on multiple risk factors (e.g., genetic risk for neuroendocrine dysregulation and externalizing problems), or on how gene–environment correlation operates for associations found in the literature (e.g., the associations between harsh parenting and externalizing, or neuroendocrine dysregulation and externalizing). Further, candidate genes for each phenotype in the model can be tested separately and together to investigate gene–gene interaction, and polygene × environment correlation and interaction.

In this example, behavior genetic and hormone studies are paired for a more comprehensive model of the development of disruptive behavior problems. Quantitative genetic studies have also been brought to bear to some extent on neuroanatomical correlates of disruptive behavior disorders (see Lenroot & Giedd, 2011 for review). A similar model could be developed incorporating neuroanatomical development. Thus, combining quantitative and molecular genetic approaches to understanding the joint influence of genetic and environmental influences on the development of disruptive behavior problems with other biological approaches (e.g., neuroanatomical, neuroendocrine) will help us to clarify studies of G × E and *r*GE, and why and how different risk factors impact disruptive behaviors across development and at specific developmental sensitive periods.

Future Directions

There are several directions researchers using a developmental behavioral genetic approach should take to continue to advance our understanding how genetic and environmental influences operate together in the development of disruptive

behavior disorders. First, from a molecular genetic perspective, it has become increasingly important to understand how multiple genes together impact development, and how groups of genes act in concert with environmental influences through gene–environment correlation and interaction processes. Investigating the role of plasticity genes, and the ways in which composites of genes believed to code for openness to the environment will help us to understand other proposed mechanisms of development including biological sensitivity to context theory, from a genetic perspective as described above.

Future studies should attempt to understand how reconcile genetic and environmental influences estimated from different genetically informed designs. The recent GWAS explaining a sizable proportion of the heritability estimates recovered in quantitative genetic studies by compounding measured allelic differences (Davies et al., 2011) provides an excellent example of one method for reconciling differences across approaches. As relevant data accumulate, this approach can be applied to the study of disruptive behavior disorders and also to samples of different ages to incorporate possible developmental differences. Additionally, twin/sibling studies can also use composites sets of genes believed to be related either conceptually or biologically within quantitative genetic frameworks to test the proportion of the additive genetic component estimated in quantitative genetic designs are accounted for by composites of multiple genes.

Further, considering behavioral genetics as a family issue highlights the importance of including both parents' and children's genes in molecular genetic studies. By including both parents' and children's genes in studies of the transmission of disruptive behaviors, it is then possible to begin to understand how the transmission of specific genes and parenting behaviors contributes to the association between parenting and child disruptive behaviors. In other words, obtaining a complete picture both from a genetic and environmental perspective is crucial for understanding how genes and environments work together to influence development.

Finally, developmental researchers must work with other scientists in other disciplines to integrate across fields. Studies investigating the confluence of genetic and endocrine or neuroanatomical influences, like our theoretically derived conceptual model for the development of disruptive behaviors, will advance our understanding of how genes, physiological, and family environmental influences exert transactional influences on the development of disruptive behavior problems over time. Genetic risk is an important component in the model, influencing prenatal risk, the development of neuroendocrine dysregulation and negative parenting over time, and both early childhood and adolescent externalizing problems, as per quantitative genetic theory. It is worth noting here that this cross-disciplinary approach is becoming much more commonplace in a variety of fields, including developmental behavioral genetics. Because behavioral genetic research involves the recruitment and assessment of difficult-to-obtain samples, there is often an effort to maximize data collection by collaborating with a wide variety of researchers. We are suggesting that this type of cross-disciplinary work be continued and

expanded to include endocrine and neuroanatomical processes as well as genetic and family influences to help provide a more complete picture of mechanisms of the development of disruptive behavior disorders.

Genetic influences are also hypothesized to moderate transactions between parenting and neuroendocrine dysregulation over time and transactions between parenting and early externalizing problems over time. These hypothesized transactions are in accordance with the theoretical frameworks, including gene–environment interplay and the biological sensitivity to context theory, described in this chapter and throughout the volume. The developmental transactions between genetic, hormonal, and family environmental influences over time play the most prominent role in the development of adolescent externalizing problems. Therefore, in the future, the strengths of developmental behavioral genetic approaches should be harnessed in combination with more advanced longitudinal data analytic approaches and hypothesized physiological mechanisms in order to match with current theoretical frameworks hypothesizing transactional influences of genetic, hormonal, and environmental influences on the development on disruptive behavior problems.

Conclusions. Developmental behavioral genetic approaches have facilitated important advances in understanding the associations between parents' and children's behaviors, and the role of the family in disruptive behavior disorders. We now understand not only that parents pass genes and environments consistent with those genes to their children which may result in a higher probability that the child develops disruptive behavior disorders but also that the genetically and environmentally influenced disruptive behaviors of the child in turn impact parenting behaviors. We also are now beginning to understand the multiple mechanisms by which genetic and environmental influences exert transactional influences on each other and on family members' behaviors, which may serve to exacerbate or attenuate disruptive behavior disorders.

While research in developmental behavioral genetics has provided substantial insight into family issues surrounding genetic and environmental influences on disruptive behavior disorders in children, there are many exciting possibilities for future research. As the strategies for data collection continue to advance, especially in regard to the techniques available (i.e., using cell phones to collect data on more occasions), developmental behavioral geneticists will be able to fine-tune models of gene–environment interplay and track the joint influences of genes and environments on other aspects of parenting and child behaviors—including how gene–environment interplay may shape daily interactions and variations in children's disruptive behavior problems on shorter-term time scales. Through creative extensions of data collection and analytic techniques, developmental behavioral genetics and collaboration across multiple disciplines developmental behavioral genetic approaches will continue to advance our understanding of the role of the family in the development of disruptive behaviors.

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Chapter 3
Neurobiology of Disruptive Behavior: A Developmental Perspective and Relevant Findings

Elizabeth J. Susman and Seth Pollak

Throughout ancient and modern history, scientists, philosophers, and the public have chronicled the emotional and disruptive problems of children and youth. In reality, over the course of development, for most children, emotional and behavioral systems and the unfolding of neurobiological development interact seamlessly with the contingency structure of the social environment. However, underlying these complex behaviors are myriad skills that are necessary for successful adaptation to novel experiences and to the dynamic social contexts in which children develop. These skills include encoding and conveying emotional and behavioral signals between caregivers, peers, and persons in the wider social contexts. Adaptation to these varied contexts reflects rapid and complex learning. These emotional and behavioral learning processes become increasingly intricate as relevant neuroanatomical and neurobiological systems mature suggesting that more sophisticated emotional and behavioral skills rely solely upon the growth of relevant neural substrates. A developmental perspective entails a vastly different approach to emotional and behavioral learning and to disruptive behavior and suggests a dialectical approach: the merging of nature and nurture and persons and contexts (Sameroff, 2010). Understanding the dialectical processes underlying neurobiological functioning in children with regulation problems might not only indicate which children are most likely to develop severe disruptive behavior but also stimulate the development of new prevention and intervention efforts.

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In this chapter we address the question: *how might neurobiological systems be changed by social experiences and what are some of the mechanisms involved in these changes*? We first present a brief theoretical perspective and history of the rapid evolution of research on the neurobiology of development and how these advances have illuminated understanding of emotional (ER) and behavioral regulation (BR). Here we use the terms ER, BR, and disruptive behavior interchangeably because both ER and BR problems can lead to disruptive behavior. We then provide examples of how integration across systems is played out in the behavioral neuroscience of ER and BR from two independent laboratories to demonstrate the ways in which a variety of developmental human, social neuroscience-based approaches can address critical conceptual and methodological issues in the emergence of disruptive behavior. To do so, we present perspectives on how developmental neurobiology constrains and enriches theories of ER and BR with application to disruptive behavior. Technological advances that have assisted advances in the neurobiology of behavioral development are then reviewed. Two issues organize this wider question, integration across species and the importance of timing of developmental events (sensitive periods). Finally, a summary is presented and suggestions are raised to guide future research.

A Perspective on ER and BR

The perspective advanced here is that disruptive behavior reflects individual–context interaction in development. Some children may be born with endogenous, genetic, and neurodevelopmental-based predispositions to misinterpret emotional and behavior environmental cues that influence constraints on regulatory functions. An impulsive temperamental tendency is illustrative of deficits in the ability to restrain behavior in reward situations. Neurobiologically based predispositions, like temperament, may predispose children to disruptive behavior. These tendencies are assumed to result from genetic influences and the nature of the prenatal, postnatal, and early childhood experiences and may have long-term repercussions for emotional, behavioral, and neuroendocrine development. But these tendencies are malleable such that emotional and social experiences that children encounter organize affective and behavioral neural circuitry so as to override inherited qualities. Stated otherwise neurodevelopmental tendencies are presumed to be moderated by the social environment so as to further or diminish tendencies toward DBD (Dodge, Pettit, Bates, & Valente, 1995). This perspective is consistent with a recent review by Tremblay (2010) who suggests that children learn behaviors from their environment and that the onset of disruptive disorder is triggered by accumulated exposure to disruptive models in the environment (Van Goozen & Fairchild, 2008, 2009). The current and Tremblay perspective do not rule out genetic and prenatal influences. The mechanisms underlying the effects of prenatal and early caregiving remain poorly understood, but data from nonhuman animal model studies have provided insight into potential molecular, cellular, and brain and environmental mechanisms

of neural circuitry that may predispose toward DBD. In animal model studies, prenatal exposure to teratogens effects are seen at the cellular differential level, genetic and social environmental levels (see Gagnidze, Pfaff, & Mong, 2010), and effects of prenatal cocaine exposure. We focus primary on the neuroendocrine and neuroanatomical systems as a modulator of the neurobehavioral development and ER and BR.

A major conceptual advance in understanding disruptive behavior is the integration of the person and the environment based on the notion that the unfolding of development depends on the context in which it occurs. Biological processes can potentiate or attenuate a behavior but specific effects on emotions and behavior depend on the micro- and macro environments for actualization. It follows that disruptive behavior is not rooted in a reflex or instinct but is exquisitely sensitive to context. The current emphasis on epigenetic studies points to the importance of the micro environment and epigenomic transformations (Miller, 2010). The emerging field of epigenetics focuses on how a gene's function or expression can be changed without affecting the basic DNA structure of a gene. Epigenetic processes occur naturally during cell division in normal development. Toxins, nutrients, prenatal or postnatal environmental exposures can activate or suppress a gene without altering its genetic code. The epigenetic findings lead to the recognition that the activation of genes is inextricably bound to environmental events that can ultimately dictate gene expression concurrently or at any time in the lifespan. The caveat is that much of the epigenetic work is based on animal model studies and the timing of epigenetic changes is not known. These genetic and other advances on the integration of person and context naturally led to the recognition that there is not a one-to-one correspondence between an emotion or behavior and a biological function/structure. In spite of these and other major conceptual and technological advances, two major tasks confront the human neuroscientist: *Can we coherently translate from nonhuman animal to human studies*? *What is the effect of time and timing of emotional and social experiences that the child encounters on organizing affective and neural circuitry relevant to behavior regulation*?

DBD and Technological Advances

Advances in theory, methodology, and technology have led to the identification of risk factors, correlates, and outcomes of ER and BR relevant to DBD. There is also convincing evidence that certain individual and family characteristics predispose to DBD. Harsh and inconsistent parenting has been repeatedly shown to be associated with conduct disorder and oppositional defiant disorder. Perinatal complications, genetic predisposition, neurocognitive deficits, low IQ, and parental antisocial behavior are also implicated (Thompson et al., 2010). Major questions remain about the exact nature of risk factors and the way in which the person, family, and environment interact to produce or prevent ER and BR regulation problems. Few studies have established a causative sequence of antecedents and outcomes. A consistent plea in previous conferences and in the published literature is the need for

sophisticated integration of biological, behavioral, and environmental processes (see Stoff & Susman, 2005). This critical merging of neurobiological paradigms is now evident and publications, like the current volume, reflect this important integration. Innovative new technologies play a major role in facilitating the application of theory-based analyses at the brain, biological and behavior levels.

Technologies to advance the neurobiology of ER and BR are designed to identify both structural and functional characteristics of individuals. For instance, within the past 3 decades the diverse area of behavioral neuroscience has emerged based on advances in understanding the neurochemistry, structure, and function of the brain and psychophysiology (Cacioppo, Tassinary, & Berntson, 2007). The evolution of modern neurobiology of human behavior is long, even though techniques of endocrinology, psychophysiology, and brain mapping vary in historical time. In the instance of neuroendocrine effects on human behavior, as far back as the 1849, a German physiologist showed the covariation of testosterone and rooster aggressive behavior (Freeman, Bloom, & McGuire, 2001; Hoberman & Yesalis, 1995). This early work became the model for testosterone and aggression research in mammals with the putative mechanism being the effect of peripherally circulating and sexually energizing function of testosterone (Archer, 2006). A century later the molecular structure of testosterone, its receptor and cellular action, and the location of testosterone receptors in the brain of subhuman primates identified the brain vs. peripherally circulating testosterone as the likely critical mechanism in the testosterone and aggressive behavior link. Technological innovations in endocrinology consisting of radioimmunoassay (RIA) and enzyme-linked immunosorbent assay (ELISA) techniques provided a noninvasive modality for assaying serum and saliva concentrations of testosterone and cortisol and other behavior-activating hormones.

The background for brain imaging techniques like magnetic resonance imaging (MRI) and other imaging techniques is comparably long. Nikola Tesla developed the Rotating Magnetic Field in 1882 in Budapest, Hungary (http://www.teslasociety.com/mri.htm). Tesla units now are the standard metric for assessing magnetic fields emitted from atoms. Functional MRI (fMRI) has a similar long history given that Roy and Sherrington's 1890 paper described the regulation of blood supply of the brain and suggested that neural activity was accompanied by a regional increase in cerebral blood flow (http://people.ee.duke.edu/~jshorey/MRIHomepage/fmri.html). But not until 1990 was there a way to noninvasively measure the flow of blood in cortical areas. Ogawa and colleagues working on rodents discovered that the oxygenation level of blood acts as a contrast agent in MR images (Ogawa, Lee, Kay, & Tank, 1990). They demonstrated that signals received from vessels were changed by pharmacologically induced changes in blood flow from the brain. It was suggested that these signals are a consequence of changing the content of deoxyhemoglobin in the blood. These fundamental discoveries in physics and the biomedical sciences have revolutionized understanding of the brain's structure and function and provide the platform for contemporary human neurodevelopment including the brain basis for psychopathology.

One of the many advantages of MRI and fMRI is that neuroimaging can be done concomitantly as one examines behavior in real time. Specifically, fMRI can examine blood flow in real time, while a specific action is taking place, allowing for attribution of increased activity in a specific brain region to increased need for oxygen

consumption. In addition to measures of blood flow, and presumed metabolic activity, one can look at connectivity and see what regions of the brain are working together when a specific task or behavior occurs. Additionally, spectroscopy has the potential to examine what neurotransmitters may be involved in a particular action. These are powerful techniques that are revolutionizing the examination of the brain, but there remains a powerful limit to this technology and direct observation of behavior remains quite important (see Aue, Lavelle, & Cacioppo, 2009).

Structural MRI and maltreatment. Both structural and functional MRI, event-related potential (ERP) and other technologies are being utilized to identify areas and functions of the brain linked to experiences, psychopathologies, and developmental changes. The experience of maltreatment provides an example of how experience including isolation affects brain functioning (Boccia & Pedersen, 2001). (See Cicchetti & Manly, 2001; Cicchetti & Toth, 1995; Cullerton-Sen et al., 2008; O'Connor & Rutter, 2000; O'Connor et al., 2003 for definition of maltreatment and psychological consequences.) Evidence from ERP studies suggests that physically abused and typically developing children have differences in their neural responses to anger. To investigate whether maltreatment was related to structural brain changes, structural MRI studies have been used to provide detailed anatomical images. The orbitofrontal cortex (oFC) is deeply involved in learning social cues, and therefore has important implications for socio-emotional development and behavioral regulation (Bachevalier & Loveland, 2006; Schore, 1996). Further, longitudinal neuroimaging research in child, adolescent, and adult populations, implicates the oFC as one of the last regions in the brain to fully develop (Gogtay et al., 2004), with changes in the oFC seen well into the third decade of life. The protracted development of the oFC suggests that it may be particularly vulnerable to postnatal experience. Recent data revealed that maltreated children had smaller oFC volumes compared to non-maltreated children, and that the size of the child's oFC region predicted the amount of stress that children reported experiencing.

Based on the social and behavioral deficits demonstrated by post-institutionalized children, Pollak and colleagues (Bauer, Hanson, Pierson, Davidson, & Pollak, 2009) examined the question of whether there were structural brain differences based on early care experience. In this study, they focused specifically on the cerebellum. The cerebellum is a brain region that is highly influenced by experience rather than genetic endowment (Giedd, Schmitt, & Neale, 2007). Further, subregions of this structure have been implicated in cognitive and social behavior (Riva & Giorgi, 2000; Schmahmann, Weilburg, & Sherman, 2007; Tavano et al., 2007). They found that the posterior–superior lobe of the cerebellum was smaller in the post-institutionalized children as compared to typically developing children; this region was also associated with children's performance on a task of executive function. Children with a smaller superior–posterior lobe volume showed poor executive control (Bauer et al., 2009). The results of this and other studies (Pollak, 2005, 2008) suggest a mechanism by which the early experience of deprivation could exert lasting consequences on social regulation.

Electrophysiological measures. Insights into the processes involved in ER and learning have been facilitated by the study of electrophysiology in at-risk children.

For example, physically abused children are reared in an environment in which anger is an extremely salient cue. It is highly predictive of danger and as such, it is adaptive for the child to be sensitized to this signal. Indeed, we have demonstrated that children who experience physical abuse are quicker than typically developing children to identify anger (Pollak, Cicchetti, Hornung, & Reed, 2000). However, the behavioral studies alone do not provide any evidence indicating how this enhanced perceptual ability serves as a risk factor for the child's own problems in ER or BR. Pollak and his collaborators hypothesized that these behavioral features reflected that physically abused children were devoting disproportionate cognitive resources to signals of anger. Such privileged processing of anger might deflect resources from other important cognitive and emotional processes necessary for healthy social functioning. To test this hypothesis, they used an electrophysiological approach called the ERP. An ERP is averaged electroencephalogram (EEG) time-locked to specific stimuli. ERPs have exquisite temporal resolution (on the order of milliseconds), but relatively poor spatial resolution. As such, ERPs can be used as an index of various cognitive processes including attention (Luck, 2005). A specific aspect of the ERP, the P3b component, is thought to reflect selective attention toward task-relevant information (Israel, Chesney, Wickens, & Donchin, 1980). As expected, physically abused children showed an enhanced P3b in response to angry faces compared to other emotions (Pollak, Cicchetti, Klorman, & Brumaghim, 1997; Pollak, Klorman, Thatcher, & Cicchetti, 2001). Further, using the P3b as an index of attention, they observed that physically abused children showed enhanced attentional allocation toward vocal expressions of anger and P3b amplitude in response to anger was associated with severity of physical maltreatment (Shackman, Shackman, & Pollak, 2007). Maltreated children also have difficulty disengaging attention from angry faces (Pollak & Tolley-Schell, 2003), and show impaired regulation of goal-directed attention (Shackman et al., 2007). Research using the N2 ERP component, an index of conflict processing (Nieuwenhuis, Yeung, Van den Wildenberg, & Ridderinkhof, 2003) revealed that physically abused children attend to facial signals of anger even when instructed to ignore them (Shackman et al., 2007), and that the degree of cognitive conflict experienced in response to task-irrelevant angry faces predicts poorer task performance (i.e., slower reaction times; Shackman, Shackman, & Pollak, 2007). In this series of investigations, ERPs provided insight into an aspect of maltreated children's social development—attention to anger—something that could not have been observed by behavioral methods alone. The severity of the maltreatment predicted attention to anger, and the more attention devoted to anger, the worse the children performed on the task.

Coherence of Nonhuman and Human Model Studies

The historical and technological advances discussed above led to major advances in understanding biological influences on brain–behavior regulation. For instance, insight into the biological influences of parenting has come from animal model

studies showing that rodent maternal behavior can effect long-term changes in responses of the offspring to stress; these changes reflect altered gene expression, the so-called environmental programming (Meaney & Szyf, 2005). A very consistent body of evidence for these "early environment by gene" interactions involves a neurotransmitter transporter called 5-HTT that fine-tunes transmission of serotonin by reuptaking it from the synaptic cleft. The gene comes in two common allelic variants: the long (l) allele and the short (s) allele, which confer higher and lower serotonin reuptake efficiency to the 5HTT, respectively. Animal studies have shown that in stressful conditions, those with two long alleles cope better. Mice with one or two copies of the short allele show more fearful reactions to stressors such as loud sounds. In addition, monkeys with the short allele that are raised in stressful conditions have impaired serotonin transmission. These animal model findings have been replicated in humans to some extent by showing that the short allele is related to some forms of psychopathology. Social interactions between young organisms and their caregivers also appear to have downstream effects on systems such as the hypothalamic–pituitary–adrenal (HPA) axis, functions associated with the orbital-ventral regions of the prefrontal cortex (PFC) (Schrijver, Pallier, Brown, & Wurber, 2004), and neuropeptide systems that regulate social behavior (Carter, 2005). A critical question concerns how to examine the ontogenesis of these mechanisms in humans. As noted in the previous section, recent studies with nonhuman mammals are leading to new insights about the biological basis of emotions and creating models to motivate biologically informed human studies. However, translation between species is not always straightforward, and we must exercise caution in applying basic findings with nonhuman animals to human children, especially in the domain of emotion (for discussion, see Sanchez & Pollak, 2009).

Brain Development and Emotional Regulation

A neuroscience approach to ER and BR requires highly standardized assessment procedures that allow concurrent recording of neural and other physiological measures. Neuroscience approaches tend to focus on quite specific dimensions of ER. One key dimension along which ER strategies can be organized is in terms of those processes that are voluntary or effortful vs. those that are automatic. The existence of automatic ER is predicated on the existence of neural circuits that modulate and attenuate certain forms of negative affect once they are elicited. Corresponding mechanisms that sustain positive affect may also exist. These mechanisms can be invoked automatically and then can be co-activated along with the generation of the emotion. The most basic forms of automatic ER are simple forms of emotion learning such as extinction. In extinction learning, a conditioned stimulus (CS) is presented without the accompanying unconditioned stimulus (US) and the responses previously associated with the CS (i.e., the conditioned response; for example, electrodermal activity in the case of human autonomic conditioning) diminish in magnitude with repeated presentations. In rodents, such extinction learning is dependent upon

the medial PFC. Because simple cue-based emotional associative learning is thought to be amygdala-dependent (Phelps & LeDoux, 2005), the extinction process is understood to depend upon inhibitory pathways from PFC to amygdala that attenuate amygdala responsivity. Output pathways from the central nucleus of the amygdala directly control the autonomic outflow that indexes conditioned responding. Rodent studies have strongly confirmed the role of the medial PFC in modulating amygdala activity as the basic architecture for extinction learning (Quirk, Garcia, & Gonzalez-Lima, 2006). Human imaging studies with simple fear conditioning and extinction are consistent with this rodent evidence (Phelps, Delgado, Nearing, & LeDoux, 2004). These imaging studies have not been carried out with abused youth who may have vastly different amygdala activity given vastly different extinction experiences in the presence of anger specifically.

Later work by Raine and colleagues indicates that cognitive and affective-emotional processing deficits are associated with psychopathology and perhaps DBD and less severe ER (Glenn & Raine, 2009). Abnormal brain structure and function, particularly the amygdala and oFC appear to be implicated in DBD. Yu et al. summarize by suggesting that brain imaging studies have suggested that: the orbitofrontal, ventromedial prefrontal, and the cingulate cortex are crucial in decision-making, behavioral control, and emotional regulation, and that deficits in these regions may contribute to features such as impulsivity and impaired moral judgment in psychopathic individuals; and, the medial temporal regions, particularly the amygdala and hippocampus, are critical for emotional processing, and thus, when impaired, predispose to a shallow affect and lack of empathy in psychopathic people (p. 814). Using fMRI, employing a similar perspective, Finger et al. (2008) found abnormal ventromedial PFC function in children and adolescents with CU traits and disruptive behavior disorders during a reversal learning task. The extent to which these findings generalize to a normal population of children is unknown.

Neuroendocrinology of stress and ER and BR. Endocrine systems play an important role in regulation of social behavior, and animal studies have provided a great deal of evidence that the endocrine system can be altered through early experience (Sanchez et al., 2005; see also discussion above and below). Endocrine systems include the hypothalamic pituitary gonadal (HPG) axis, HPA axis, and posterior hypothalamic hormones. Recent work investigates the effects of early adverse experience on the neuropeptide oxytocin (OT) and the stress-related hormone cortisol. Oxytocin (OT) is a polypeptide hormone and neuroregulator produced in the hypothalamus and released centrally and peripherally into the blood stream via axon terminals in the posterior pituitary (Kendrick, Keverne, Baldwin, & Sharman, 1986), and appears to be part of the neural system of reward circuitry that includes the nucleus accumbens (Lovic & Fleming, 2004). For example, in nonhuman animals and humans alike, higher levels of oxytocin are associated with decreases in stress hormones, such as cortisol, and other behaviors such as increases in positive social interactions and attachment behaviors (Grippo, Trahanas, Zimmerman, Porges, & Carter, 2009; Kosfeld, Heinrichs, Zak, Fischbacher, & Fehr, 2005; Witt, Carter, & Walton, 1990; for a review, see Carter, 1998).

Oxytocin is generally not examined in relation to behavior regulation in humans especially children with DBD given that obtaining sufficient blood volume for assays cannot be unobtrusively obtained. However, it has theoretical relevance given that oxytocin is considered an affiliative or "love" hormone. In contrast to the emotions associated with OT, DBD is characterized by hostile emotions and behaviors sometimes resulting in social isolation by peers and family. Oxytocin may attenuate the effects of isolation or similar negative-DBD-related emotions as has been demonstrated in small mammals. Carter (1988) has assessed the effects of oxytocin in the prairie vole, a socially monogamous mammal that forms social bonds and exhibits high parasympathetic activity making it a good model for considering the neurobiological systems that mediate sociality (Carter, 1998). To test the effects of isolation on psychophysiological parameters, adult female prairie voles were exposed to social isolation or continued pairing with female sibling (Grippo, Trahanas, Zimmerman, Porges, & Carter, 2009). Isolation significantly increased basal heart rate (HR) and reduced HR variability and vagal regulation of the heart in isolated animals. But these changes were prevented with oxytocin administration supporting the hypothesis proposed by Grippo and colleagues that oxytocinergic mechanisms can protect against behavioral and cardiac dysfunction in response to chronic social stressors. The positive effect of oxytocin on empathy is supported by other findings showing genetic variations in the oxytocin receptor are related to empathic tendencies and lower reactivity to stress (Rodrigues, Saslow, Garcia, John, & Keltner, 2009). Overall, assessment of oxytocin has much to offer in relation to shedding light on positive or approach behaviors given the putatively affiliative nature of this peptide hormone. Higher levels of oxytocin may promote affiliative behavior and low DBD but this relation has not been demonstrated in humans.

Oxytocin and cortisol are both affected by adverse rearing conditions. Cortisol, the end product of the HPA axis, modulates a wide range of biological responses such as energy release, cardiovascular function, immune activity, growth, emotion, and cognition (Diorio, Viau, & Meaney, 1993; Sapolsky, Romero, & Munck, 2000; Takahashi et al., 2004). (See below for an extended discussion of cortisol.) Secretion of cortisol allows the organism to regulate metabolic processes and to adapt and cope effectively with current stressors. However, chronic elevation of cortisol impairs behavioral adaptation and has been associated with ER and BR difficulties and psychopathology (Goodyer, Park, Netherton, & Herbert, 2001; Gunnar & Vazquez, 2001; Heim, Owens, Plotsky, & Nemeroff, 1997; Sapolsky, 2000). Other than affiliation, oxytocin is linked to a limited number of theoretical concepts that are proposed mechanisms linking stress and ER and BR in human model studies.

The behavioral problems of post-institutionalized children are consistent with dysregulation in the oxytocin system and the HPA axis. To examine these questions, Wismer and colleagues investigated the response of oxytocin and cortisol to a social game with their mother and a stranger. This investigation demonstrated that, unlike typically developing children, post-institutionalized children have an abnormally muted oxytocin response after interacting with their mother (Wismer-Fries, Ziegler, Kurian, Jacoris, & Pollak, 2005). Further, post-institutionalized children showed

prolonged elevations in cortisol levels following the interaction with their mother, but not the stranger. More severe neglect was associated with the highest basal cortisol levels similar to the higher levels observed in sexually abused girls during the childhood years. The severe neglect children also had the most impaired cortisol regulation following the mother interaction (Wismer-Fries, Shirtcliff, & Pollak, 2008). These results suggest that early social deprivation may disrupt the function of the oxytocin system and HPA axis. The use of hormone measures provides insight into post-institutionalized children's experience of a social interaction with their mother. The disrupted response of the oxytocin system and HPA axis suggests that for post-institutionalized children, interactions with their mother may be stressful, rather than calming and comforting. If this is the case, it is easy to infer how such experiences could interfere with the development of adaptive social relationships.

Cortisol and Developmental Transitions

The heaviest concentration of work on integrating neurobiological development and ER and BR is based on the HPA stress system (Dickerson & Kemeny, 2004). Consequently, theoretical concepts and perspectives are emerging rapidly to guide the design and interpretation of findings on the stress system and ER and BR. The assumption is that functional abnormalities of HPA are involved in ER, BR, and other psychopathologies such as major depression (see Chrousos & Gold, 1992 for a comprehensive description of the anatomy and physiology of the HPA stress system). Briefly, the HPA axis component of the stress system consists of the hypothalamus, pituitary, and adrenals. Following an endogenous or exogenous stressor, through connections from the amygdala and cortex, the hypothalamus is stimulated to secrete corticotrophin releasing hormone (CRH), a 41-amino acid peptide derived from a 191-amino acid preprohormone. CRH is secreted by the paraventricular nucleus (PVN) of the hypothalamus and stimulates the secretion of adrenocorticotropic hormone (ACTH), a polypeptide tropic hormone produced and secreted by the anterior pituitary gland. ACTH is synthesized from pre-pro-opiomelanocortin (pre-POMC) and its main function is to stimulate the adrenal glands to secrete the principle glucocorticoid, cortisol.

Mechanisms whereby the HPA axis is involved in neurodevelopment are reviewed elsewhere (e.g., Gunnar & Vazquez, 2001; Susman, 2006). The notion is that failures in social regulation of the HPA axis in early development such as in the case of inadequate parenting plays a role in shaping the cortical and limbic circuits involved in modulating later novel and threatening experiences (Gunnar & Quevedo, 2008). Early toxic prenatal experiences, abusive and insensitive caretaking and the consistency of the caregiving environment are experiences presumed to alter the development of threat, fear, and stress-relevant systems.

The HPA axis glucocorticoid, cortisol, has become exceedingly fashionable as a neurobiological probe on the HPA axis given its abundance and accessibility in saliva. In the past, the mixed sets of findings regarding the direction of relations

between cortisol and emotions and behaviors were a constant frustration to scientists searching for coherence based on what is known about the physiology of stress. An underlying assumption in most studies is that cortisol rises when confronted with novel, challenging and fearful situations. In one of the first papers on individual differences in cortisol and aggression, cortisol was shown to decrease in some children, increase in others or remain stable in yet other children when confronted with a novel physical stressor (Susman et al., 2007). The apparent lack of coherence between sets of findings now can be accounted for by the many emotions and behaviors assessed and contextual and methodological paradigms used in various studies (Dickerson & Kemeny, 2004). The recognition that the *contexts* in which children are reared influence basal morning cortisol (e.g., child care settings, Roisman et al., 2009) provided an important insight into explaining inconsistencies across studies. Parental insensitivity and child care experiences in the preschool period predicted lower morning cortisol more than a decade later in 15 year olds. These and other findings have led to an interest in the meaning of *lower basal* and *reactive* cortisol and associations with disruptive behavior.

The Attenuation Hypothesis

Increasingly clear is that there are interindividual differences in morning and diurnal, basal cortisol concentrations and reactive cortisol concentrations in response to stressors. A seemingly paradoxical and puzzling set of findings in the neurobiology of stress is the negative association between basal cortisol levels and disruptive behavior. The evidence suggests that basal cortisol is lower in individuals with varying forms of disruptive behavior (e.g., McBurnett et al., 1991; Susman, Dorn, Inoff-Germain, Nottelmann, & Chrousos, 1997; Vanyukov et al., 1993). Whereas higher basal cortisol putatively predisposes to fear, lower cortisol seems to predispose toward externalizing and aggressive behavior (Schulkin, 2003). The *attenuation hypothesis* emerged to begin to account for apparent divergent and paradoxical findings. The *attenuation* hypothesis refers to the tendency for disruptive, externalizing, and criminal individuals to exhibit lower concentrations of cortisol and other products of the stress system (e.g., CRH) (Susman, 2006). The hypothesis proposes that these lower cortisol levels are a result of earlier experiences such as a chaotic, inconsistent, or neglectful parenting or a traumatic experience such as child abuse. The result is a down regulation of HPA axis activity. Attenuation of the stress system is grounded in the principle that individuals adapt over time when confronted with potentially stressful challenges that involve activation of neural, neuroendocrine, and neuroendocrine-immune mechanisms. It follows that individuals experiencing chronic stressors will adapt by downregulating the stress response to as to preserve vital resources. This process is referred to as allostasis or stability through change (McEwen, 1998). Allostatic load includes frequent activation of allostatic systems, such as chronically high cortisol levels, or the failure to shut off allostatic or adaptive activity after stress. Down regulation of HPA activity is proposed to be

an adaptive response given that allostatic load or chronic activation of the HPA axis can lead to disease as high levels of cortisol impose strain on multiple systems (e.g., the pancreas leading to insulin resistance).

Recent studies confirm that even seemingly minor traumatic experiences attenuate basal cortisol similar to what has been true of more extreme experiences like child maltreatment or early institutionalization. As discussed above, more time in child care centers and insensitive parenting during the preschool period, experiences that are seemingly minor and normative stressors, predicted lower awakening cortisol at age 15 (Roisman et al., 2009). Similarly, longitudinal a multi-method assessment of interparental conflict was associated with lower levels of child cortisol reactivity to simulated conflict between parents (Davies, Sturge-Apple, Cicchetti, & Cummings, 2007). Diminished cortisol reactivity, in turn, predicted increases in parental reports of child externalizing symptoms over a 2-year period. These and other sets of finding have led to the suggestion that attenuated basal cortisol level may be a consequence of early experiences, as in nonhuman models, and may be a vulnerability for later or persistent disruptive behavior.

Attenuated cortisol is speculated to be associated with disruptive behavior via low empathy, callousness, or sensation seeking. With regard to sensation seeking, Quay (1965) developed the hypothesis that low arousal can be pathological and may lead to excessive sensation seeking behavior that increases the probability of disruptive tendencies, including criminal behavior. Even in nonclinical populations sensation seeking influences the individual to seek or to avoid experiences that are perceived necessary to maintain an optimal level of arousal (Kohn, 1987). Raine and colleagues showed the longitudinal associations between low autonomic nervous system (ANS) activity and later criminal behavior (Raine, Venables, & Williams, 1990). A later report showed that sensation seeking as early as age 3 was predictive of aggression at age 11, suggesting the stable affiliation of sensation seeking and deviant behavior (Raine, Reynolds, Venables, Mednick, & Farrington, 1998). Low arousal as reflected in low cortisol may parallel to low ANS arousal associated with criminal and disruptive behavior (Raine et al., 1990). Attenuated basal cortisol and ANS indices may reflect common genetic and child rearing environmental roots.

The HPA axis and ANS represent the interacting dual arms of the stress system and are likely affected by similar genes, rearing conditions, and common parental and family interaction processes thereby explaining attenuation in both HPA and ANS activity. With regard to family processes, a recent report showed the importance of considering both individual variations in emotional experiences, specifically, depression, and family processes. Internalizing symptoms moderated the association between adolescents' reported distress and blunted cortisol reactivity in response to family conflict (Spies, Margolin, Susman, & Gordis, 2011). The longitudinal findings also showed that adolescents with current and past internalizing symptoms had a blunted cortisol response but this was not the case in adolescents who never qualified as depressed. Without the benefit of HPA reactivity, which typically prepares individuals to handle the external environment, Spies et al. suggest that some adolescents may be more prone to show the poor coping that accompanies DBD.

On the other hand, reduced cortisol reactivity to distress may be a functional pattern for adolescents with internalizing symptoms in families where conflict or violence is a frequent occurrence. To date the bulk of the evidence linking attenuated basal cortisol and ANS activity and disruptive behavior is correlational or retrospective (see Spies et al., 2011 for the exception) and awaits further longitudinal study.

The attenuation hypothesis and puberty. Neuroendocrine and rapid physical growth changes, like puberty, tend to be sensitive periods of the lifespan for the development of behavior problems, ER problems, and overt psychopathology. The interactions between these changes and the multiple changes in emotions, social roles, and behavior and relationships with parents and same- and opposite-sex peers associated with puberty have received much attention. A major advance in the last few decades in understanding changes in gonadal hormones and behavior was the identification of receptors for steroid hormones and brain differentiation (Joëls, 1997; McCarthy, 1994) and genetic expression in sex steroid cells (Gagnidze et al., 2010) in the brain that may influence ER and BR. But how gonadal steroid (testosterone and estrogens) modulate puberty-related brain changes and the neuroendocrinology of stress and ER and BR remain speculative. The literature strongly supports that the *timing* of neuroendocrine events like puberty and its associated gonadal steroid hormones changes are related to individual differences in ER and BR (Susman & Dorn, 2009). The ability to ferret out the effects of neuroendocrine-hormone changes and social-role changes and problems in ER and BR is exceedingly difficult with current methodologies. Nonetheless, the timing of the activation of the HPG axis, physical growth and social role changes, and ER and BR at puberty is described extensively in the literature. [See Ge and Natsuaki (2009) and Negriff, Susman, & Trickett (2010) for theories on timing of puberty and implications for ER and BR].

Early adolescence. Traditionally is considered an especially sensitive and vulnerable period for the expression of both internalizing and externalizing behavior problems because of the rapid, neuroendocrine, puberty-related changes that are differentially timed for males and females. We recently demonstrated that the timing of the secondary sexual physical changes at puberty (Tanner stage) moderates both attenuated and accentuated profiles of cortisol and salivary alpha amylase (sAA), a surrogate marker of ANS activity (Susman et al., 2010). For boys, timing of puberty moderated the association between cortisol and sAA reactivity and antisocial behavior. *Higher* cortisol reactivity in *later* timing boys was related to a composite measure of antisocial behavior and rule-breaking behavior problems. In contrast, lower or attenuated sAA reactivity and *earlier* timing of puberty in boys was related to rule breaking and conduct disorder symptoms. The interaction between timing of puberty and HPA or sympathetic–adrenal–medullary (SAM) regulation and timing of puberty in boys suggests that growth-related reproductive, neuroendocrine mechanisms are sensitive to the extensively documented adverse ER and BR consequences of off-time pubertal development. Of note is that the effects were for boys and not girls suggesting that future work could entertain questions regarding sex differences in sex steroid-sensitive neurobiological functioning.

Attenuation and sexual maltreatment. Perhaps one of the most compelling findings supporting the attenuation hypothesis and exposure to traumatic experiences and neuroendocrine development showed the acute vs. chronic trajectories of cortisol in sexually abused girls. Inconsistencies have pervaded the literature regarding the attenuation vs. the accentuation of basal cortisol activity following maltreatment. To address these inconsistencies, the developmental course of basal cortisol was assessed at six time points from childhood through adolescence and into young adulthood in young women. The aim was to determine whether childhood abuse results in disrupted cortisol activity (Trickett, Noll, Susman, Shenk, & Putnam, 2010). Morning basal cortisol was measured in females with confirmed familial sexual abuse and a nonabused comparison group. First, using a cohort sequential design the hypothesis was tested that the normative developmental course for basal cortisol levels is, on average, a steady increase from middle childhood into early adulthood after which time there is a leveling off of cortisol. The linear trend in cortisol for sexually abused females was significantly less steep compared to nonabused girls from age 6 to 30 indicating attenuation in cortisol activity starting during the pubertal years with significantly lower levels of cortisol by early adulthood. As a more direct test of the attenuation hypothesis, time since the disclosure of abuse was considered as an influence on cortisol levels. Cortisol activity was initially significantly higher (close to the time of disclosure) but slopes were significantly less steep (slope of change) for abused females across the longitudinal study. These longitudinal findings convincingly demonstrate how the experience of childhood sexual abuse has the potential for disrupting the neurobiology of stress, thereby providing further support that victims of sexual abuse experience HPA neuroendocrine alterations characterized by low activity.

Prenatal Influences on Neurobiological Development

Pre- and perinatal risks for DBD can be traced to early causes as well as strategies for prevention of DBD. Much of what is known about pre- and perinatal effects on behavior are based on animal model studies. Specific areas of the brain affected by teratogens in the prenatal brain are extensively demonstrated. The harmful neurobiological effects of illicit substances as teratogens are well documented. In animal model studies prenatal cocaine exposure affects the dopamine receptors in the striatum in mice (Tropea et al., 2008). A close analog to the animal model studies of early influences on ER and BR are the human model studies of the effects of illicit substance use on emotions and cognition. Exposure to cocaine shows both structural and functional change in the brains of children. Specifically, diffusion tensor imaging (DTI) showed frontal lobe microstructural changes suggesting a less mature brain after prenatal cocaine exposure (Warner et al., 2006). The effect of prenatal exposure of illicit substances on humans is evident in emotions, obesity, neurocognitive development, memory, and executive function (see reviews in Singer & Richardson, 2011) and attention and inhibition (Carmody, Bennett, & Lewis, 2011).

Nicotine exposure also has been shown to lead to disturbances in neuronal path finding, abnormalities in cell proliferation and differentiation, and disruptions in the development of the cholinergic and catecholaminergic systems all have been reported in molecular animal studies of in utero exposure to nicotine (Ernst, Moolchan, & Robinson, 2001). Human prenatal exposure to tobacco smoke is a major risk factor for the human newborn, increasing morbidity and even mortality in the neonatal period. In utero exposure to tobacco is associated with motor, sensory, and cognitive deficits in infants and toddlers, suggesting a toxic effect of tobacco on early neurodevelopment. Specifically, findings indicate that in utero exposure to tobacco is associated with motor, sensory, and cognitive deficits in infants and toddlers, suggesting a toxic effect of tobacco on early neurodevelopment (Wickstrom, 2007). Finally, the effects of alcohol exposure on fetal development are legend (see review, Ismail, Buckley, Budacki, Jabbar, & Gallicano, 2010) yet prevention efforts have not been entirely successful in eliminating exposure to prenatal illicit substances in the USA.

The extent to which prenatal illicit drugs exposure influences the development of psychopathology, specifically, DBD, remains speculative as these drug-related brain changes cannot be disentangled from the known environmental and genetic risks. For instance, there are correlated environmental and perinatal medical complications with prenatal cocaine exposure that cannot be disentangled from prenatal exposure risks. Children with DBD can come from similar environmental risks; maternal depression, early hostile parenting practices, and mother's young gynecological age at the birth of her child, and mother's antisocial behavior during both adolescence and pregnancy. Tremblay (2010) reviews multiple other early family characteristics that affect the development of DBD: mother's low level of education, smoking during pregnancy family low income, family dysfunction, lack of stimulation, presence of siblings and mother's hostile or coercive parenting. The question remains as to the scientific appropriateness of attempting to separate prenatal and genetic and environmental risks given the inherent comorbidity between all three factors in the etiology of regulatory disorders.

A human, prenatal neuroendocrine approach is highly relevant to understanding prenatal influences on ER and BR. These studies assess the effects of psychological stress on fetal, child and adult development, and children's development (Entringer, Kumsta, Hellhammer, Wadhwa, & Wüst, 2009). In a longitudinal study of stress and teen pregnancy, the assumption was made that psychosocial stressors in the lives of pregnant adolescents affect the fetal and maternal HPA and HPG axis milieu thereby influencing fetal brain development and subsequent ER and BR. The first set of findings demonstrated that stress-related hormones were related to ER and BR in pregnant adolescents. We examined CRH as a potential mechanism involved in ER and BR during pregnancy and the early postpartum period. In addition to being produced in the hypothalamus, CRH is synthesized in peripheral tissues and is expressed in large quantities in the placenta. CRH is a marker in the placenta that determines the length of gestation and the timing of parturition and delivery and is involved in fetal lung maturation. Its links to behavior during pregnancy were unknown but given that hypothalamic CRH is secreted in response to stress, that

CRH is high during bouts of depression, and that animal model studies show maternal stress affects fetal development, CRH was measured in early pregnancy and the postpartum period as well as maternal depression and conduct disorder symptoms. Lower CRH concentrations in early pregnancy (<16 weeks gestation) were related to depression symptoms in early pregnancy and predicted symptoms in the last trimester of pregnancy (34–40 weeks) (Susman et al., 1999). Lower concentrations of CRH also were related to a greater number of conduct disorder symptoms in early pregnancy and in the postpartum period (<4 weeks postpartum). The findings support our hypothesis that CRH is associated with both ER and BR during pregnancy and beyond. A recent study supported our findings and speculated that CRH levels might be inversely associated with risk of postpartum depressive symptoms (Rich-Edwards et al., 2008). A methodological problem in relating CRH during pregnancy ER and BH is that it is not easy to determine the extent to which CRH in the peripherally circulating plasma of pregnant women reflects placental or hypothalamic stress-sensitive activity with the latter being the most potent influence on neurobiological development. The placental component of CRH is only to a small degree dependent upon the maternal HPA axis that is closely associated with the stress response. However, placental CRH is likely biologically active because the CRH binding protein (CRH-BP), which shows a parallel rise to CRH during pregnancy (Suda et al., 1989), does not bind at the same place on placental CRH as the CRH receptor. Regardless of its hypothalamic or placental origin, the total circulating CRH pool may influence, or be influenced by, depression and disruptive behavior in the mothers. Of note is that lower or blunted levels of CRH did not predict indices of neonatal behavior. But the extent to which depression and disruptive behavior are related to CRH and that CRH is critical to the healthy development of the fetus, especially to fetal lung maturation, low CRH secondary to ER or BR may indirectly affect fetal brain development and optimal neurobiological development. Alternatively, common and yet unidentified third factors may influence the pathway between CRH and depression and conduct disorder symptoms. It is most likely the case that a vicious cycle exists with maternal abnormalities of the central neuroendocrine/ANS during pregnancy predisposing infants to ER and BR problems and vice versa.

The steroid hormone fetal milieu and aggression and temperament. In a related report, support was found for the role of the prenatal maternal endocrine milieu and neurobiological development in children's aggressive behavior and temperament (Susman, Schmeelk, Ponirakis, & Gariepy, 2001). Contemporary theories suggest that temperament has biological roots and that prenatal and environmental mechanisms influence aspects of the neurobiology of temperament (DiPietro, Hodgson, Costigan, & Johnson, 1996). Although partially biologically rooted, temperament is not static but is adaptive to environmental demands (Rothbart & Ahadi, 1994). In a longitudinal study of pregnant adolescents, temperament that was hypothesized to be a dynamic process is affected by stress-related fluctuations in the maternal endocrine milieu and emotions during gestation and the early postnatal years. The hypothesis focused on relating clustering of maternal, adrenal, and gonadal

hormones and emotions and the child's aggressive behavior and temperament at age 3 years. Illustrative findings included the following: Verbal aggression and nonverbal aggression were significantly higher in children of mothers in the low *prenatal* hormone cluster than children of mothers in the high prenatal hormone cluster. Children of mothers in the *postpartum* low testosterone (T), estradiol (E_2), androstenedione (Δ4-A), and medium cortisol (Cort) cluster (mainly low hormone cluster) exhibited significantly more physical aggression than children of mothers in the medium T and Δ4-A, high E2 and low Cort cluster. Maternal patterns of steroid hormones, emotions, and parenting attitudes and practices were related to multiple aspects of temperament (activity level, reactivity and soothability, attentional focus, high pleasure, and fear) when the children were age 3 years. The findings support, but cannot confirm, the potential disruptive influence of the prenatal adrenal and gonadal milieu in the development of children's aggressive behavior and temperament. Overall the findings support the importance of the prenatal hormone environment as the time when neurobiological development is vulnerable to insults from maternal stress-related emotions and behavior. (See also Schmeelk, Granger, Susman, & Chrousos, 1999 for a related study of CRH and immune functioning and infant postnatal complications).

We conclude by suggesting that the neuroendocrine, HPG axis is implicated in emotional and behavioral regulation likely beginning during prenatal development and extending throughout the lifespan. In addition, DBD is linked to both the HPA and ANS-SNS aspects of the stress system with hypoarousal of the HPA axis being fairly well documented, although hyperarousal of the HPA axis is evident in some studies of ER and BR. As mentioned above the effects of prenatal exposure to toxins cannot be disentangled from genetic and environmental influences that impinge on the developing fetus.

Sex Differences

Sex differences are evident in the overall prevalence of DBD and it is not known how early brain organization influences these sex differences. It is known that exposure to prenatal cocaine affects inattention and inhibition with males being higher on both dimensions than females after exposure (Carmody et al., 2011). Sex differences increase with age so it is difficult to identify prenatal factors that lead to the higher incidence of DBD in males compared to females. To the extent that testosterone is putatively responsible for sexually dimorphic differences in fetal brain development, then testosterone may be implicated in DBD as well. The topic of sex differences is too massive to review herein. Nonetheless, Paus (2010) and colleagues show remarkable differences in brain development in males and females. Sexual dimorphism in brain structure assessed in vivo with MRI, is most prominent in brain size. Sex differences are present at birth and increase through childhood and adolescence into adulthood. The extent to which these structural differences contribute to regulation of behavior remains unknown.

Summary and Conclusion

The findings presented above show that neuroendocrine, stress-related adrenal and gonadal hormones are possibly related in significant ways to fetal brain development, ER, BR, and DBD. Answers to many fundamental questions regarding structural and functional neurobiological development and disruptive behavior can only be obtained via animal probes at this time. Problems remain using animal model research to explain human brain development. As an example, it is not apparent how stressful events (such as handling or isolation) in nonhuman animals approximate the kinds of caregiving and traumatic experiences that human children experience. Variations in the timing, quantity, and quality of parental care and stress exposure are operationally different across species. Another critical issue with regard to prenatal, childhood, and adolescent stress-related experiences concerns the effects of developmental timing, as described above. For instance, maltreatment may interact with the puberty transition to influence cortisol secretion (Trickett et al., 2010). The slope of change in basal cortisol was higher in sexually abused girls until the pubertal years but then the slope became lower than the slope for maltreated girls during the pubertal years. There has been little information on how experiences like maltreatment and other early influences of stress affect the neurobiology of brain development in humans. The strongest effects of early experience on stress neurobiology in the rodent are observed during the first 2 weeks of the pup's life, but the timing and even the existence of a comparable period in human infant development is to be determined. For these reasons, the phenomena of institutionalization and child abuse and neglect have begun to take center stage, both in questions about nature-nurture effects on human development and as a test case for translation between human and nonhuman models of neurobehavioral development (Pollak, 2005). Indeed, behavioral genetic analyses suggest that many of the emotional problems observed in abused children are attributable to environmental effects, with vulnerability to experience modulated by genetic factors (Jaffee & Price, 2007; Jaffee et al., 2004; Kaufman et al., 2004) as described above.

In this chapter, we illustrate ways in which neurobiological systems appear to be changed by social experiences. Children are confronted with abundant opportunities to attach emotional significance to cues in their environments. For this reason, the central nervous system draws attention to important features in the environment and allows regulation of responses to change (Rueda, Posner, & Rothbart, 2005). We have shown that from the prenatal period onward, social experiences may heighten the salience of emotional cues and, conversely, the absence of some developmentally appropriate experiences may hinder emotional and behavioral development because of insufficient learning opportunities. We also showed that the timing of social experiences appears to modulate prenatal, childhood, and adolescent ER and BR. Maternal endogenous (prenatal hormones) and exogenously induced moods appear to have long-term effects on the child's aggressive behavior and temperament.

We propose that a future need is for scientists to make transformational changes in the piecemeal studies of the past by answering a series of questions. What are the

important questions to be addressed in future research regarding the neurobiological etiology, trajectory, and outcomes of DBD? A vast empirical basis exists regarding the onset and outcome of DBD but how will these findings inform future research? What theoretical and methodological innovations are required to advance the prevention of DBD? Why do boys have a higher incidence of DBD than girls? The answers to these questions will necessarily build on past empirical findings. Nonetheless, it will be desirable for studies to be hypothesis-driven and based on merging or new theories of brain–behavior interactions. Past work has been primarily atheoretical with exceptions such as theoretical concepts like ER and BR during childhood and adolescence. These concepts are assumed to be predictably associated with adult psychopathology that likely stem from dysfunctions in multiple social and neural systems. Overall, advances in understanding the interactions between brain and behavior are likely to be derived from interdisciplinary, longitudinal, and technologically advanced investigations characterized by the inclusion of indices of genetic, neural, and social underpinnings of the neurobiology of DBD. Innovative approaches to brain and behavior currently are exemplified in contemporary behavioral neuroscience that grew out of the biomedical, psychology, and brain sciences. Scientists in these areas increasingly have adopted an integrated approach to understanding brain development. Effects of isolation on brain development in the early years are rooted in psychological studies of the effects of isolation in subhuman. Current brain imaging work has the potential for illustrating the myriad ways in which the human brain develops based upon the *timing and quality of input received from the social environment*. *Imaging studies hold much promise for understanding the neurobiology of disruptive behavior*.

The search for genetic markers will most assuredly enlighten work on the person by environment integration and is consistent with the perspective upon which this chapter is based, that brain development inherently represents gene and environment interaction. Genes of the serotonergic and dopaminergic systems receive substantial attention in understanding DBD (Lahey et al., 2011). A trend likely to be promising is the assessment of genes that affect early brain development that in turn influence later DBD. Gao and colleagues suggest that future molecular genetic studies identifying genes' coding for early brain abnormalities are needed to substantiate the neurodevelopmental hypothesis regarding the onset of antisocial behavior (Gao, Glenn, Schug, Yang, & Raine, 2009). Future investigation that includes genetic risks for neurobiological functioning might focus on which children are most likely to persist in engaging in severe antisocial behavior so as to guide the development of new interventions. At the same time, it is unlikely that single genes interacting with learning experiences will explain the emergence of complex DBD in humans. There is a building consensus that complex traits are not the product of a single gene. Thus, mechanistic understanding of the ways in which humans are influenced by the interaction of genes and their environments is a necessary next step in research.

fMRI and MRI technologies will likely be used extensively in the future (Logothetis, 2008) as these techniques have the potential for establishing developmental trajectories for neural development, an essential endeavor if brain

dysfunctions are to be explained in relation to normative brain changes. This new tool already is telling us the developmental sequence whereby the brain controls cognitive, emotional, and behavior control. For instance, asynchrony in brain development is used to explain risk taking in adolescents. Groups of interdisciplinary scientists now show that changes in prefrontal development from adolescence to adulthood are associated with suboptimal and risky choices (Galvan et al., 2006). The PFC is one of the last areas of the brain to develop compared to limbic and amygdala areas (e.g., see Casey, Jones, & Somerville, 2011). If cognitive control and an immature PFC were the primary basis for suboptimal choice behavior, then children should exhibit behavior similarly or even worse than adolescents, given the PFC and cognitive abilities are less well developed in children. Immature prefrontal function alone does not appear to account for adolescent choices in their behavior. Casey et al. (2011) further suggest that the context in which decisions are made is an important consideration as children typically have less unsupervised social and sexual activities than adolescents. Developmental studies also are telling us that more needs to be known about early neurocognitive development. Thompson and colleagues suggest that birth cohort studies have yielded limited information on how pre- and perinatal factors and early neurodevelopment relate to child psychopathology (Thompson et al., 2010). They suggest the need for epidemiological research with a specific focus on early neurodevelopment, measures of early childhood psychopathology, and long-term follow-up.

In a similar vein, Tremblay (2010) suggests that prevention of deficits that lead to antisocial behavior requires early, intensive, and long-term support to parents and child. These studies are suggested to be longitudinal, collaborative across sites, and involve analysis at multiple levels of analysis. The importance of understanding early development and later disorders has been considered important for decades. Recent advances in epigenetics support an emphasis on prenatal insults and behavioral development. Regulatory regions of the genome can be modified through epigenetic processes during prenatal life to make an individual more likely to experience chronic diseases later in life (Thornburg, Shannon, Thuillier, & Turker, 2010). Maternal prenatal stress is one mechanism perhaps contributing to epigenesis. The good news is that affected or marked regions of DNA during the prenatal period can become "unmarked" under the influence of dietary nutrients. These exciting new findings indicate that interventions to "unmark" affected DNA via individual and family interventions hold promise for the future. The successful interventions carried out by Olds et al. (2004) may have affected DNA but retrospective studies cannot answer these questions. In brief, the epigenetic story provides a promising basic mechanism that provides an environmentally based explanation of intergenerational transmission for physical and mental disorders involving genes but the disorder is not directly genetically transmitted. With regard to the translational utility of neurobiology, recent work on the endocrinology of stress, for instance, and regulation of behavior is gaining in salience in the prevention and treatment of childhood disorders. Fisher and colleagues showed that family interventions improved the pattern of diurnal cortisol in foster children (Fisher, Stoolmiller, Gunnar, & Burraston, 2007). A characteristic pattern arising from disrupted caregiving is a low early-morning

cortisol level that changes little from morning to evening. More normative cortisol levels are expected to parallel better behavior. A primary finding was that early morning cortisol increased in foster children in the intervention group over the course of the study. These transitional studies are sorely needed along with long-term longitudinal studies in the prevention of disruptive behavior given the high cost of disruptive behavior to the individual and to society.

Pharmacological interventions are one possibility for changing neurobiological functioning; however, far less invasive interventions may be equally effective in some instances. The types of intervention that will be effective in recalibrating amygdala functioning via neurofeedback, for instance, and perhaps promoting sensitivity in disruptive youth may be appropriate strategies for intervention but current knowledge on these and other strategies are not yet known to be efficacious intervention strategies. In addition, increasing empathy via talk therapies may be effective in reducing callousness and subsequent behavior. Together the promising directions discussed above will tell us how the neurobiology of brain development and social learning experiences predispose toward disruptive behavior disorder.

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Chapter 4
Callous-Unemotional Traits and Developmental Pathways to the Disruptive Behavior Disorders

Paul J. Frick, R. James Blair, and F. Xavier Castellanos

Introduction

Research on the development of antisocial and aggressive behavior in children and adolescents has consistently shown that such behaviors are heterogeneous and may result from a number of different causal mechanisms (Dodge & Pettit, 2003; Frick & Viding, 2009; Moffitt, 2006). This research has important implications for both research and practice related to the disruptive behavior disorders. First, the various subgroups of youth within conduct problems often show distinct social, biological, cognitive, and emotional correlates to their problem behavior that need to be integrated into causal models (Blair, 2005; Frick & White, 2008). Second, these subgroups of youths may also differ in the severity of their behavior and their long-term outcomes (Frick & Dickens, 2006; Moffitt, 2006). Third, these subgroups may require different approaches to treatment in order to address their disruptive behaviors (Frick, 2006, 2009). Based on this research, there have been a large number of attempts to define more homogenous subgroups of youths with disruptive behavior disorders who differ on their behavioral manifestations, developmental course and

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outcome, etiology, and response to treatment. In this chapter, we first provide a summary of some recent attempts to define distinct developmental pathways through which children may develop severe patterns of antisocial and aggressive behavior. After this, we focus on one approach that we feel has particular promise for both integrating past approaches and for guiding future research in this area. This approach focuses on the presence or absence of callous-unemotional (CU) traits (i.e., a lack of guilt and empathy; deficits in emotional responding) in children and adolescents with conduct problems. We summarize some key issues in the research using these traits for understanding distinct developmental pathways to disruptive behavior disorders and we highlight several critical steps that would advance this area of work for both theory and practice.

Past Attempts to Subtype Children and Adolescents with Conduct Problems

Childhood-onset and adolescent-onset conduct problems. Perhaps one of the most commonly used methods for subtyping antisocial children and adolescents with severe conduct problems or delinquency is based on the age at which their severe antisocial behavior first emerges. This distinction has been used to differentiate those who start showing delinquent acts (Patterson & Yoerger, 1997; Tibbetts & Piquero, 1999) or serious conduct problems (American Psychiatric Association, 2000) prior to the onset of adolescence (i.e., early-onset or childhood-onset) and those who start showing serious conduct problems coinciding with the onset of adolescence (i.e., late-onset or adolescent-onset). There have been a number of reviews of an extensive literature to support this distinction (e.g., Moffitt, 2006; Patterson, 1996). To summarize this work, the childhood-onset group is more likely to show aggressive behaviors in childhood and adolescence (Moffitt, Caspi, Dickson, Silva, & Stanton, 1996) and is more likely to continue to show antisocial and criminal behavior into adulthood (Moffitt, Caspi, Harrington, & Milne, 2002). Further, the childhood-onset group is more likely to show neuropsychological (e.g., deficits in executive functioning) and cognitive (e.g., low intelligence) deficits (Raine, Yaralian, Reynolds, Venables, & Mednick, 2002). Children in this group are also more likely to show temperamental and personality risk factors, such as impulsivity (McCabe, Hough, Wood, & Yeh, 2001), attention deficits (Fergusson, Lynsky, & Horwood, 1996), and problems in emotional regulation (Moffitt et al., 1996). Research also suggests that this group comes from homes with greater levels of family instability, more family conflict, and with parents who use less effective parenting strategies (Aguilar, Sroufe, Egeland, & Carlson, 2000; McCabe et al., 2001; Patterson & Yoerger, 1997; Woodward, Fergusson, & Horwood, 2002).

Thus, children in the childhood-onset group appear to have a more severe and chronic pattern of antisocial behavior that is related to both dispositional risk factors and problems in their socializing environments (Moffitt, 2006). In contrast, children in the adolescent-onset group tend to show problems that are more likely to be limited to adolescence (Moffitt et al., 2002). Also, when children within the

adolescent-onset group differ from control children without conduct problems, it is often in showing higher levels of rebelliousness and being more rejecting of conventional values (Dandreaux & Frick, 2009; Moffitt et al., 1996). Thus, this group has been conceptualized as showing an exaggeration of the normative process of adolescent rebellion (Moffitt, 2006). Given that their behavior is viewed as an exaggeration of a process specific to adolescence, and not due to enduring vulnerabilities, their antisocial behavior is less likely to persist beyond adolescence. However, they may still have impairments that persist into adulthood due to the consequences of their adolescent antisocial behavior (e.g., a criminal record, dropping out of school, substance abuse; Moffitt & Caspi, 2001).

Subtypes based on comorbidity. Another consistent research finding is that children with disruptive behavior disorders often have other types of emotional and behavioral problems as well. Some attempts to subtype children with conduct problems have used the presence of co-occurring conditions to separate unique subgroups. One attempt of particular interest has focused on the combination of the inattentive, impulsive, and hyperactive behaviors associated with a diagnosis of Attention-Deficit/Hyperactivity Disorder (ADHD) with significant conduct problems and antisocial behavior (Lynam, 1996). Substantial research supports this approach, in that children with both types of problems show a more severe and aggressive pattern of antisocial behavior than children with conduct problems alone (Lilienfeld & Waldman, 1990; Waschbusch, 2002). In addition, children with ADHD and conduct problems have poorer outcomes, such as showing higher rates of delinquency in adolescence and higher rates of arrests in adulthood (Babinski, Hartsough, & Lambert, 1999; Loeber, Brinthaupt, & Green, 1990). Importantly, however, the vast majority of children with childhood-onset Conduct Disorder, especially those in clinic-referred samples, show this comorbidity with ADHD (Abikoff & Klein, 1992). As a result, this method of subtyping often does not designate a group that is very distinct from the group defined by an early age of onset.

Subtypes based on types of aggression. Another approach to subtyping children with conduct problems is to distinguish between children with aggressive and nonaggressive forms of conduct problems (American Psychiatric Association, 1980; Frick et al., 1993). More recent extensions of this approach have focused on the types of aggressive behavior exhibited by the child or adolescent with conduct problems. Specifically, research has indicated that two distinct types of aggression can be identified in samples of children or adolescents with conduct problems (Poulin & Boivin, 2000). Reactive aggression is characterized by impulsive defensive responses to a perceived provocation or threat and is usually accompanied by a display of intense physiological reactivity. In contrast, proactive or instrumental aggression is not associated with provocation but is defined as aggression in pursuit of an instrumental goal and is usually premeditated and planned (Dodge & Pettit, 2003). Two recent meta-analyses suggest that these two types of aggression tend to be highly correlated in children and adolescents ($r = 0.68$; Card & Little, 2006; $r = 0.64$; Polman, Orobio de Castro, Koops, van Boxtel, & Merk, 2007). Despite this high correlation, factor analyses have consistently supported that these two types of

aggression can be separated (Poulin & Boivin, 2000; Salmivalli & Nieminen, 2002). Further, there have been a number of studies supporting different correlates to the two types of aggression in samples of youths. Specifically, proactive aggression has been more highly correlated with delinquency and alcohol abuse in adolescence, as well as criminality in adulthood (Pulkkinen, 1996; Vitaro, Brendgen, & Tremblay, 2002). In contrast, reactive aggression has been more highly correlated with school adjustment problems and peer rejection (Poulin & Boivin, 2000; Waschbusch, Willoughby, & Pelham, 1998).

The two types of aggression have also been associated with different social, cognitive, and emotional characteristics. Specifically, reactive aggression has been associated with a tendency to attribute hostile intent to ambiguous provocations by peers and difficulty developing nonaggressive solutions to problems in social encounters (Crick & Dodge, 1996; Hubbard, Dodge, Cillessen, Coie, & Schwartz, 2001), whereas proactive aggression has been associated with a tendency to overestimate the possible positive consequences of aggressive behavior and underestimate the probability of getting punished because of their behavior (Price & Dodge, 1989; Schwartz et al., 1998). Further, reactive aggression, but not proactive aggression, has been associated with heightened physiological reactivity to perceived provocation (Hubbard et al., 2002; Munoz, Frick, Kimonis, & Aucoin, 2008; Pitts, 1997).

Despite the growing evidence for these differential correlates to the two types of aggression, the utility of this distinction has been questioned (Bushman & Anderson, 2001; Walters, 2005). One primary concern expressed in these critiques is that the dichotomous distinction between reactive and proactive aggression does not address the high correlation between the two types of aggression. Further, studies have consistently shown a distinct pattern of overlap between the two types of aggression. That is, there appears to be two groups of aggressive children; the first is highly aggressive and shows both types of aggressive behavior and the second group is less aggressive overall and shows only reactive types of aggression (Frick, Cornell, Barry, Bodin, & Dane, 2003; Munoz et al., 2008; Pitts, 1997). Thus, it is possible that differences between the two types of aggression are largely due to the proactive group being more severely aggressive overall.

Subtypes based on the construct of psychopathy. Another attempt to define meaningful subgroups of children and adolescents with disruptive behavior disorders is based on a long history of clinical research with adults showing that psychopathic traits designate an important subgroup of antisocial individuals (Cleckley, 1976; Hare, 1993; Lykken, 1995). Psychopathic traits have historically not focused solely on the antisocial behavior of the individual but have placed a greater emphasis on the affective (e.g., lack of empathy; lack of guilt; shallow emotions) and interpersonal (e.g., egocentricity; callous use of others for own gain) style of the person. Importantly, antisocial adults who also show the affective and interpersonal facets of psychopathy show a much more severe, violent, and chronic pattern of antisocial behavior (Hare & Neumann, 2008) and they show very different affective, cognitive, and neurological characteristics compared to antisocial individuals without these traits (Blair, Mitchell, & Blair, 2005; Newman & Lorenz, 2003; Patrick, 2007).

Across the past several decades, there have been several similar attempts to use the affective and interpersonal traits of psychopathy to designate a distinct group of children and adolescents with disruptive behavior disorders (Forth, Hart, & Hare, 1990; Frick, 2009; McCord & McCord, 1964; Quay, 1964). To illustrate one such approach, the third edition of the Diagnostic and Statistical Manual of Mental Disorders (DSM-III; American Psychiatric Association, 1980) made distinctions among children with Conduct Disorder based on whether or not they were "socialized" or "undersocialized." The following quote from the DSM-III describes the characteristics of the undersocialized type and illustrates its link to the construct of psychopathy:

> The *Undersocialized* types {of CD} are characterized by a failure to establish a normal degree of affection, empathy, or bond with others. Peer relationships are generally lacking, although the youngster may have superficial relationships with other youngsters. Characteristically, the child does not extend himself or herself for others unless there is an obvious immediate advantage. Egocentrism is shown by readiness to manipulate others for favors without any effort to reciprocate. There is generally a lack of concern for the feelings, wishes, and well-being of others, as shown by callous behavior. Appropriate feelings of remorse are generally absent. Such a child may readily inform on his or her companions and try to place blame on them (p. 45; American Psychiatric Association, 1980).

Research on this subtype of Conduct Disorder supported its validity in that adolescents who were classified as both undersocialized and aggressive tended to have poorer adjustment in juvenile institutions and were more likely to continue to show antisocial behavior into adulthood, when compared to other antisocial adolescents (Frick & Loney, 1999; Quay, 1987). Also, the undersocialized-aggressive group was more likely to show several neuropsychological correlates to their antisocial behavior, such as low serotonin levels and autonomic irregularities (Lahey, Hart, Pliszka, Applegate, & McBurnett, 1993; Quay, 1993; Raine, 1993).

Despite the promising findings for this method of subtyping children with disruptive behavior disorders, there was considerable confusion over the core features that should define the undersocialized subgroup and differentiate it from other groups of antisocial youths. This confusion was due to two main issues. First, in an attempt to avoid using the pejorative term "psychopathy," the term "undersocialized" was used. Unfortunately, this term did not clearly describe the affective or interpersonal features of psychopathy and led to other connotations (e.g., the child is not well socialized by parents; the child is unable to form peer groups). Second, the operational definition provided in the DSM-III for the undersocialized subgroup listed several indicators of which no more than one could be present. This list included only one symptom specific to the affective and interpersonal dimensions of psychopathy (i.e., "apparently feels guilt or remorse when such a reaction is appropriate not just when caught or in difficulty"). The other four symptoms focused on indicators of social attachment (e.g., "has one or more peer group friendships that have lasted over 6 months"; "avoids blaming or informing on companions") that have not proven to be reliable indicators of the affective and interpersonal features of psychopathy.

As a result of these problems in the definition of undersocialized Conduct Disorder, this method for classifying subgroups of children with this disruptive behavior disorder was not continued in later editions of the manual. However, in

recent years, a significant body of research has emerged refining how the key features associated with psychopathy may be expressed in children and adolescents and demonstrating the clinical and etiological importance of using these features to designate a subgroup of antisocial youths. Specifically, there appears to be a subgroup of antisocial children and adolescents who show a callous (e.g., lack of empathy; absence of guilt) and unemotional (e.g., shallow or deficient emotional responses) interpersonal style. Notably, these traits have documented important subgroups of antisocial youths in community (Frick, Cornell, Barry, et al., 2003), clinic-referred (Christian, Frick, Hill, Tyler, & Frazer, 1997), and forensic samples (Lawing, Frick, & Cruise, 2010). They have been assessed and validated in preschool (Kimonis, Frick, Boris, et al., 2006), school-age (Frick, Bodin, & Barry, 2000), and adolescent (Gretton, Hare, & Catchpole, 2004) samples, as well as in samples in North America (Frick et al., 2000 Gretton et al., 2004), England (Blair, 1997; Viding, Simmonds, Petrides, & Federickson, 2009), Belgium (Roose, Bijttbier, Decoene, Claes, & Frick, 2010), Sweden (Enebrink, Anderson, & Langstrom, 2005), Germany (Essau, Sasagawa, & Frick, 2006), Greek Cypress (Fanti, Frick, & Georgiou, 2009), Australia (Dadds, Fraser, Frost, & Hawes, 2005), and Israel (Somech & Elizur, 2009). They also have proven to be important for designating important subgroups of antisocial youths in samples of both boys (Kruh, Frick, & Clements, 2005) and girls (Marsee & Frick, 2007) and in large ($n = 7{,}977$) representative samples (Rowe et al., 2009).

From the available research, it is difficult to estimate the percentage of antisocial youths, or youths with Conduct Disorder who would be high on CU traits. This difficulty is largely because research to date has used various assessment instruments, cut scores, and informants to designate children and adolescents high on CU traits. For example, within adolescents in the juvenile justice system, the percentages of persons with high CU traits have ranged from 13 to 36 % (Caputo, Frick, & Brodsky, 1999; Corrado, Vincent, Hart, & Cohen, 2004; Gretton et al., 2004). In clinic-referred children (ages 6–13) with disruptive behavior disorder diagnoses, approximately 35 % were also high on CU traits (Christian et al., 1997). Finally, in a nationally representative sample of 5–16 year olds, about 46 % of children and adolescents with Conduct Disorder had high rates of CU traits (Rowe et al., 2009). Thus, the available research suggests that from 13 to 46 % of antisocial youths or youths with Conduct Disorder show high rates of CU traits.

The rest of the current chapter focuses on research showing the importance of this subgroup of antisocial youths with CU traits for understanding, assessing, preventing, and treating children and adolescents with severe conduct problems. Given the size of this literature and the availability of several recent reviews (Blair, Peschart, Budhani, Mitchell, & Pine, 2006; Frick, 2009; Frick & White, 2008), an exhaustive review of this research is beyond the scope of this chapter. However, in the following sections, we provide a selective review of some of the key findings which illustrates the great potential of this approach to subtyping antisocial youths.

Key Issues in Research on Callous-Unemotional Traits

Stability of CU Traits in Children and Adolescents

There is now considerable data to suggest that the CU traits are relatively stable from late childhood to early adolescence (Frick, Kimonis, Dandreaux, & Farrell, 2003; Munoz & Frick, 2007; Obradović, Pardini, Long, & Loeber, 2007). For example, Frick, Kimonis, Dandreaux, & Farrell, (2003) reported a stability estimate of 0.71 across 4 years using an intraclass correlation coefficient (ICC) for parent ratings of CU traits in a sample of children with an average age of 10.6 years at the initial assessment. This level of stability is much higher than is typically reported for parent ratings of other aspects of children's adjustment (Verhulst, Koot, & Berden, 1990). With respect to younger children, Dadds et al. (2005) found moderate 1-year stability estimates for parent-reported CU traits ($r = 0.55$) in a community sample of Australian children who were 4–9 years of age. Several studies have compared the stability of these traits across different methods of assessment. For example, Obradović et al. (2007) reported relatively high rates of stability for parent report of CU traits ($r = 0.50$) over a 9-year period but lower (but still significant) levels of stability for teacher ($r = 0.27$) ratings, in a sample of boys who were 8 years of age at the initial assessment. Munoz and Frick (2007) compared the 3-year stability of parent and youth self-report ratings of CU traits in a non-referred sample of young adolescents (average age of 13.4 at initial assessments) and found very high stability for parent ratings ($r = 0.71$) and moderate but still significant stability for self-report ratings ($r = 0.48$).

These traits have also proven to be relatively stable from adolescence to adulthood (Blonigen, Hicks, Kruger, Patrick, & Iacono, 2006; Forsman, Lichtenstein, Andershed, & Larsson, 2008; Loney, Taylor, Butler, & Iacono, 2007). For example, Forsman et al. (2008) reported that CU traits were relatively stable for both boys ($r = 0.43$) and girls ($r = 0.54$) from age 16 to 19. Blonigen et al. (2006) reported that self-reported CU traits were relatively stable ($r = 0.60$) from late adolescence (age 17) into early adulthood (age 24). Further, Loney et al. (2007) reported that self-report of CU traits in adolescence (ages 16–18) was moderately stable (ICC = 0.40) over a 6-year follow-up period.

Finally, two studies have addressed the long-term stability of CU traits from childhood to adulthood. Both studies reported that CU traits in childhood were significantly associated with measures of psychopathic traits in adulthood, even when controlling for childhood conduct problems and other risk factors for antisocial behavior (Burke, Loeber, & Lahey, 2007; Lynam, Caspi, Moffitt, Loeber, & Stouthamer-Loeber, 2007). Importantly, Lynam et al. (2007) showed that the correlation over 11 years (from age 13 to 24 years) between CU traits in childhood and an adult measure of psychopathy was $r = 0.31$. These studies suggest that the stability of CU traits is similar to what is typically found for other personality traits in children and adolescents (Roberts & DelVecchio, 2000). However, these findings also clearly suggest that CU traits are not unchangeable. To illustrate this, Lynam

et al. (2007) reported that children at age 13 who were in the upper 10 % of CU traits at age 13 were 3.22 times more likely to show elevations on a measure of psychopathy 11 years later. However, only 21 % of the boys who scored in the upper 10 % on the measure of CU traits at age 13 were elevated on measures of psychopathy at age 24. Thus, CU traits in childhood were clearly a risk factor for showing high levels of psychopathic traits in adulthood, but a large number of boys seemed to show reductions in their rate of CU traits over time (see also Frick, Kimonis, et al., 2003 for a similar pattern of change).

CU Traits and the Severity, Stability, and Treatment Amenability of Antisocial Behavior

Several recent qualitative (Frick & Dickens, 2006; Frick & White, 2008) and quantitative (Edens, Campbell, & Weir, 2007; Leistico, Salekin, DeCoster, & Rogers, 2008) reviews have been published showing that CU traits are predictive of a more severe, stable, and aggressive pattern of behavior in antisocial youth. For example, Edens et al. (2007) conducted a quantitative meta-analysis of 21 nonoverlapping samples showing that measures that include CU traits were associated with general or violent recidivism with effect sizes of $r = 0.24$ and $r = 0.25$, respectively. Similarly, Frick and Dickens (2006) reported on a qualitative review of 24 published studies using 22 independent samples. Ten of these studies showed a concurrent association between CU traits and measures of aggressive, antisocial, or delinquent behavior, and 14 studies showed a predictive relationship with follow-up intervals ranging from 6 months to 10 years. Frick and White (2008) reviewed eight additional concurrent studies and three additional longitudinal studies showing an association between CU traits and the severity of antisocial behavior. Across these two qualitative reviews, the studies included community ($n = 6$), clinic-referred ($n = 4$), and forensic ($n = 13$) samples and had samples ranging in age from 4 to 20. Importantly, this research also suggests that children and adolescents with CU traits show a more severe and pervasive pattern of aggressive behavior and they also tend to show aggression that is more premeditated and instrumental (i.e., for gain) in nature (Flight & Forth, 2007; Frick, Cornell, Barry, et al., 2003; Kruh et al., 2005).

Frick and Dickens (2006) also reviewed five studies showing that CU traits were associated with poorer treatment outcomes in samples of antisocial youths. However, several more recent studies suggest that children with CU traits may be difficult to treat, but that certain types of treatment may still be effective. For example, Hawes and Dadds (2005) reported that clinic-referred boys (ages 4–9) with conduct problems and CU traits were less responsive to a parenting intervention than boys with conduct problems who were low on CU traits. However, this differential effectiveness was not consistently found across all phases of the treatment. That is, children with and without CU traits seemed to respond equally well to the first part of the intervention that focused on teaching parents methods of using positive reinforcement to encourage prosocial behavior. In contrast, only the group without CU traits

showed added improvement with the second part of the intervention that focused on teaching parents more effective discipline strategies. Waschbusch, Carrey, Willoughby, King, and Andrade (2007) reported that children (ages 7–12) with conduct problems and CU traits responded less well to behavior therapy alone than children with conduct problems without CU traits. However, children showed marked improvement when stimulant medication was added to the behavior therapy, although the children with CU traits were still less likely to score in the normative range than those without these traits. Finally, Caldwell, Skeem, Salekin, and Van Rybroek (2006) demonstrated that adolescent offenders with CU traits improved when treated using an intensive treatment program that utilized reward-oriented approaches, targeted the self-interests of the adolescent, and taught empathy skills. Specifically, they reported that adolescent offenders high on these traits who received the intensive treatment were less likely to recidivate in a 2-year follow-up period than offenders with these traits who underwent a standard treatment program in the same correctional facility.

CU Traits and Past Subtyping Attempts

Thus, research suggests that the subgroup of antisocial youths with CU traits appears to be clinically important. Further, this research also suggests that this method of subtyping antisocial youths could help to integrate and advance many of the subtyping methods reviewed previously. First, CU traits are more likely to be present in children with a childhood-onset of antisocial behavior (Dandreaux & Frick, 2009; Moffitt et al., 1996; Silverthorn, Frick, & Reynolds, 2001), consistent with the contention that the early-onset group shows a more chronic and characterological disturbance (Moffitt, 2006). However, within children with a childhood-onset to their conduct problems, these traits seem to designate a more severe group (Christian et al., 1997; Dadds et al., 2005). Also, these traits seem to have predictive utility, even controlling for the age of onset of serious antisocial behavior. For example, in a sample of high-risk boys followed into adulthood, CU traits predicted a higher likelihood of being a violent offender, even controlling for an onset of delinquency by age 10 (Loeber et al., 2005). Finally, there is evidence that many of the social, genetic, emotional, and cognitive correlates to CU traits that are reviewed in the next section are not found in children with a childhood-onset to their conduct problems who do not show these traits (Frick & White, 2008).

Second, similar findings have been reported when CU traits have been related to the impulsive and overactive behaviors associated with ADHD. That is, children with CU traits and conduct problems do show high levels of impulsivity and diagnoses of ADHD. However, within youths with both CD and ADHD, it seems to be the CU traits that are associated with the most severe behavior problems (Christian et al., 1997) and the most stable patterns of antisocial behavior (Frick, Stickle, Dandreaux, Farrell, & Kimonis, 2005). Further, only those youths who are impulsive, antisocial, *and* who show CU traits show the distinct genetic, emotional, and

cognitive characteristics that are similar to adults with psychopathy (Barry et al., 2000; Loney, Frick, Clements, Ellis, & Kerlin, 2003; Viding, Jones, Frick, Moffitt, & Plomin, 2008). For example, Barry et al. (2000) studied a clinic-referred sample of children ages 6–13. They reported that only children with ADHD, conduct problems, and CU traits showed low levels of fear and a reward-dominant response style, similar to adults with psychopathy, whereas those with ADHD and conduct problems alone did not show these characteristics. Finally, as noted above, children and adolescents with CU traits are more likely to show the combination of reactive and proactive aggression that has also been used to designate an important subgroup of antisocial youths. Unfortunately, it is not clear if the poor outcome for children with this severe pattern of aggressive behavior is due to the aggressive behavior itself or to the presence of CU traits. However, there is evidence that some of the social-cognitive deficits (e.g., a tendency to emphasize the rewarding aspects of aggressive behavior and ignore the punishments) (Pardini, Lochman, & Frick, 2003) and some of the emotional characteristics (e.g., lack of emotional responsiveness to provocation) (Munoz, Frick, Kimonis, & Aucoin, 2008) that have been associated with proactive aggression may be more specifically associated with the CU traits.

In summary, children and adolescents who show conduct problems and CU traits show characteristics similar to groups identified using other subtyping approaches. That is, they are more likely to show a childhood-onset to their conduct problems, they show a high rate of ADHD, and they are more likely to show a severe pattern of aggression involving both reactive and proactive aggression. Thus, CU traits may help to integrate these past subtyping approaches. More importantly, CU traits seem to designate a more specific group than past subtyping approaches. Specifically, they seem to designate a unique group within those youths with a childhood-onset to their conduct problems and within those who show co-occurring ADHD. Further, these traits may provide a method for differentiating within aggressive youths those who show distinct emotional and cognitive characteristics better than past approaches which have relied on highly correlated dimensions of aggressive behavior (i.e., reactive and proactive aggression).

CU Traits, Antisocial Behavior, and Parenting

To this point, we have reviewed evidence that CU traits seem to define a clinically important group of antisocial youth, based largely on the severe, stable, and aggressive nature of their behavior. However, research also suggests that children and adolescents with severe conduct problems who also show high levels of CU traits show a number of distinct characteristics that could reflect differential causal processes. For example, failure in parental socialization is a central component of many theories developed to explain the etiology of conduct problems (e.g., Patterson, 1996). Further, ineffective parenting strategies have been repeatedly linked to the development of antisocial behavior in numerous studies (Frick, 2006). However, there is evidence to suggest that the association between conduct problems and dysfunctional parenting practices may be different for youth with and without CU

traits. Wootton, Frick, Shelton, and Silverthorn (1997) studied a sample of both non-referred and clinic-referred youth ages 6–13. They reported that a composite measure of several dysfunctional parenting practices (i.e., low parental involvement, failure to use positive reinforcement, poor monitoring and supervision, inconsistent discipline, and use of corporal punishment) were strongly related to conduct problems in children without CU traits but unrelated to conduct problems in children high on these traits. These findings have been replicated in several samples including non-referred school children in grades 3 and 4 (Oxford, Cavell, & Hughes, 2003), high-risk girls (ages 7 and 8; Hipwell et al., 2007), and in adolescent juvenile offenders (Edens, Skopp, & Cahill, 2008).

Thus, there is now relatively consistent evidence to suggest that conduct problems are more strongly related to many types of ineffective parenting practices in the absence of CU traits. It is important to note, however, that these findings should not be interpreted to suggest that other parenting dimensions or other factors within the family context may not be related to conduct problems in youth with high CU traits. It is possible that the dimensions of parenting that have been studied in this body of research (i.e., methods of parental socialization) are less related to conduct problems in youth with CU traits, but that other aspects of parenting (e.g., the parent–child relationship) could still play an important role in the development and maintenance of conduct problems in these youths (Fowles & Kochanska, 2000; Lynam, Loeber, & Stouthamer-Loeber, 2008; Robison, Frick, & Morris, 2005). Further, these findings do not necessarily suggest that parental socializations practices may not influence the stability of the CU traits themselves. For example, Frick, Kimonis, et al. (2003) showed that more effective parental socialization practices were related to a decrease in the level of CU traits in children over a 4-year study period.

CU Traits, Antisocial Behavior, and Personality

Children with CU traits and conduct problems also show distinct personality characteristics compared to those without such traits. For example, children with CU traits show higher scores on measures of fearless or thrill-seeking behaviors (Essau et al., 2006; Frick, Lilienfeld, Ellis, Loney, & Silverthorn, 1999; Pardini, 2006). Also, CU traits have been negatively correlated with measures of trait anxiety or neuroticism, whereas level of conduct problems has been positively correlated with measures of trait anxiety (Andershed, Gustafson, Kerr, & Stattin, 2002; Frick et al., 1999; Lynam et al., 2005; Pardini, Lochman, & Powell, 2007). Importantly, the negative correlation between measures of CU traits and trait anxiety/neuroticism is generally only found when controlling for the level of conduct problems (Frick et al., 1999; Lynam et al., 2005). That is, children with CU traits tend to show less trait anxiety *given the same level of conduct problems*. This pattern of results suggests that children with CU traits are less distressed by their behavior problems, perhaps with less concern about impact for themselves and others, compared to youth with comparable levels of conduct problems (Frick et al., 1999; Pardini et al., 2003).

CU Traits and Genetics

Several studies have examined the heritability of CU traits (e.g., Larsson, Andershed, & Lichtenstein, 2006; Taylor, Loney, Bobadilla, Iacono, & McGue, 2003; Viding, Blair, Moffitt, & Plomin, 2005). Larsson et al. (2006) and Taylor et al. (2003) provided similar estimates of the amount of variation in CU traits accounted for by genetic effects (i.e., 43 % and 42 %, respectively), whereas Viding et al. (2005) reported heritability of 68 % in those probands showing elevated CU traits. Importantly, a substantial proportion of this genetic variance for explaining CU traits has been shown to be independent of aggression (Taylor et al., 2003) and hyperactivity (Viding et al., 2008). Moreover, genetic factors appear to contribute substantially to the stability of CU traits across time (Forsman et al., 2008).

Interestingly, Viding et al. (2005) demonstrated that the heritability of the antisocial behavior at age 7 for those youth with the most severe conduct problems was strikingly affected by the level of the youth's CU traits. The heritability of antisocial behavior for those high on CU traits was considerably greater (0.81) than for those low on CU traits (0.30). This result was replicated in the same sample 2 years later at age 9 (Viding et al., 2008). Moreover, similar work by an independent lab revealed that, while a common genetic factor loaded substantially on both CU traits and antisocial behavior, a common shared environmental factor loaded exclusively on antisocial behavior (Larsson et al., 2007). Finally, recent provocative work reported that left posterior cingulate and right dorsal anterior cingulate (dACC) gray matter concentrations showed significant heritability (0.46 and 0.37, respectively) and that common genes explained the phenotypic relationship between these regions and psychopathic traits, which include CU traits (Rijsdijk et al., 2010). These last data suggest that the genetic contribution to CU traits might manifest through an impact on anterior and posterior cingulate cortex (PCC) development. Both regions have been implicated in adult psychopathy (Kiehl, 2006). However, as yet, there are no clear indications that computational processes mediated by these neural systems are disrupted in this population.

CU Traits and Neuro-Cognitive Impairment

A series of studies have examined the neuro-cognitive impairments shown by youths with elevated CU traits in response to the emotional displays of others. Early work indicated that youths with elevated CU traits showed reduced autonomic responses to the distress of others (Blair, 1999). Children with elevated CU traits also showed reduced recognition of fearful and, to a lesser extent, sad facial expressions (Blair, Colledge, Murray, & Mitchell, 2001; Stevens, Charman, & Blair, 2001), and fearful vocal tones (Blair, Budhani, Colledge, & Scott, 2005). More recently, studies have demonstrated reduced attentional orienting to distress cues in youth with elevated CU traits and antisocial behavior (Kimonis, Frick, Fazekas, & Loney, 2006; Kimonis et al., 2008). Interestingly, work has shown that the selective deficit in fear

recognition can be ameliorated if the child's attention is focused on the eye region (Dadds et al., 2006). Indeed, youths with elevated CU traits show a reduction in both the number and duration of fixations on the eye region when processing fearful expressions (Dadds, El Masry, Wimalaweera, & Guastella, 2008). Moreover, increasing the child's focus on the eye region significantly reduces the impairment in fear recognition seen in youth with elevated CU traits (Dadds et al., 2008).

A second series of studies have examined specific forms of emotional learning involving the learning of the valence of objects and actions following experience with reinforcement and punishment. In particular, studies have demonstrated that youth with elevated CU traits show impairments in extinction. These studies involve learning to stop a previously rewarded response following a reinforcement contingency change such that it now comes to be progressively more associated with punishment (Fisher & Blair, 1998; O'Brien & Frick, 1996). They also showed impairments in reversal learning, involving learning to reverse the response associated with a stimulus following a change in reinforcement contingency (Blair, Colledge, & Mitchell, 2001; Budhani & Blair, 2005). Critically, a fine grained analysis of the behavioral performance demonstrated, in contrast to past explanations for psychopathy (Lykken, 1995), that youth with CU traits are not simply unresponsive to punishment. Specifically, on the trial immediately following a punishment, the youth with CU traits is as likely as a comparison youth to make the alternative response to the stimulus (i.e., they are as likely as a comparison youth to adapt their behavior in response to punishment). This alteration of responding immediately following a punishment is thought to reflect the recruitment of dorsal anterior cingulate/dorsomedial frontal cortex in response to the response conflict induced by the punishment information. These behavioral data indicate that this form of response to punishment is intact in youth with CU traits, an impression reinforced by fMRI work indicating appropriate recruitment of dorsal anterior cingulate/dorsomedial frontal cortex in response to punishment during a reversal learning task (Finger et al., 2008).

The problem that youth with CU traits seem to have on reversal learning tasks is a significantly increased tendency to revert to the older, now unreinforced response, in the reversal phase (Budhani & Blair, 2005). In fact, they are significantly more likely to revert to the older now unreinforced response following a reward for the newly correct response (Budhani & Blair, 2005). The ability to maintain responding to the newly correct response is thought to reflect the role of orbital frontal cortex (OFC) in representing the value of the newly correct response. This value representation should successfully guide the individual's decision-making. These behavioral data indicate appropriate recruitment of OFC in the representation of reinforcement information is disrupted in youth with CU traits. This impression is reinforced by fMRI work showing disrupted representation of reinforcement information in youth with CU traits (Finger et al., 2008).

It has been argued that deficits in responding to social cues critical for moral socialization (the distress of others) and specific forms of emotional learning (stimulus-reinforcement learning in particular) interfere with the ability of the individual with elevated CU traits to be efficiently socialized (Blair, 2007). This is

thought to underlie the deficits reported in the moral judgments made by children and adolescents with these traits (Blair, 1997). Moreover, it likely contributes to their increased propensity to show the positive outcome expectancies regarding aggressive situations with peers which were discussed previously. As a result, the individual is less likely to represent the negative consequences of the victim's distress.

Key Theoretical and Methodological Issues for Advancing Knowledge on This Topic

Developmental Models of CU Traits

Taken together, this selective review suggests that there is a growing body of research indicating a number of social, personality, emotional, cognitive, and neurological factors that differentiate antisocial youth with and without CU traits. Thus, it is important that causal models of antisocial and aggressive behavior consider the developmental processes involved in the etiology of these traits and/or the antisocial and aggressive behavior displayed by youth with them. Further, such research needs to incorporate research on the normal development of empathy, guilt, and other aspects of conscience with research on characteristics of antisocial youths showing CU traits. For example, many of the characteristics of children with CU traits closely resemble a temperament that has been described as behaviorally uninhibited or fearless (Frick & Morris, 2004; Pardini, 2006). Specifically, uninhibited children tend to seek out novel and dangerous activities and show less physiological arousal to threats of punishments (Kagan & Snidman, 1991; Rothbart, 1981). Importantly, there is also evidence that children with this uninhibited or fearless temperament score lower on measures of conscience development (Kochanska, Gross, Lin, & Nichols, 2002; Rothbart, Ahadi, & Hershey, 1994).

Based on these findings, there have been a number of theories developed to explain this link between an uninhibited temperament and impairments in conscience development. For example, Kochanska (1993) proposed that the anxiety and discomforting arousal that follow wrong-doing and punishment are integral in the development of an internal system that functions to inhibit misbehavior, even in the absence of the punishing agent. She proposed that behaviorally uninhibited children may not experience this "deviation anxiety" which could impede conscience development. Dadds and Salmon (2003) proposed a similar model that also focused on the child's responsiveness to parental socialization attempts and, in particular, their sensitivity to punishment. In support of these theoretical models, Pardini (2006) reported that scores on a measure of fearlessness were correlated with a measure of CU traits, but this association was mediated by a measure of punishment insensitivity.

Blair and colleagues (Blair, 1995; Blair, Colledge, Murray, et al., 2001; Blair, Jones, Clark, & Smith, 1997) have also proposed a theoretical model focusing more

specifically on the development of empathetic concern in response to the distress in others. They suggest that humans are biologically prepared to respond to distress cues in others with increased autonomic activity in what they have labeled the violence inhibition mechanism (VIM). This negative emotional response develops before the infant or toddler is cognitively able to take the perspective of others, such as when a young child becomes upset in response to the cries of another child (Blair, 1995). According to this model, these early negative emotional responses to the distress of others become conditioned to behaviors in the child that led to distress in others. Through a process of conditioning, the child learns to inhibit such behaviors as a way of avoiding this negative arousal. Children with the behaviorally uninhibited temperament may not experience this negative arousal and, as a result, do not experience this conditioning.

Importantly, these models focusing on conscience development are important because they set the stage for early preventive interventions that can target children who may be at risk for problems in development due to their temperamental characteristics but who may not yet manifest serious behavioral problems. However, to guide these interventions, it is important to consider what might moderate the link between the temperamental risk and problems with conscience development. For example, Kochanska (Kochanska, 1997; Kochanska & Murray, 2000) proposed that the parent–child relationship, especially the responsiveness towards each other, may be a critical socialization component for uninhibited children. This aspect of parenting does not rely on punishment-related arousal for internalization. Instead, it focuses on the positive qualities of the parent–child relationship (Kochanska & Murray, 2000). In support of this proposal, attachment security was shown to be predictive of conscience development in temperamentally fearless children (Kochanska, 1995, 1997). Also, Cornell and Frick (2007) specifically tested several interactions between behavioral inhibition and parenting in predicting scores on measures of guilt and empathy in young (age 3–5 years) children. They reported an interaction with parental consistency in discipline, such that children who were behaviorally inhibited showed higher levels of guilt, irrespective of the consistency of parenting. However, uninhibited (i.e., fearless) children showed higher levels of guilt only when parental consistency was high. Cornell and Frick (2007) also reported an interaction between authoritarian parenting (i.e., use of strong rule-oriented and obedience-oriented parenting) and behavioral inhibition, such that authoritarian parenting was unrelated to parent ratings of guilt in behaviorally inhibited children but positively related to levels of guilt in uninhibited children. The authors interpreted these findings to suggest that behaviorally inhibited children were predisposed to develop appropriate levels of guilt and often did so, even with less than optimal parenting. However, behaviorally uninhibited children required stronger and more consistent parenting to develop appropriate levels of guilt.

To summarize, this model specifies that problems in conscience development are the key developmental mechanisms leading to the antisocial behavior in children with CU traits. Risk for these problems in conscience development stems from a fearless and uninhibited temperament that can make a child more difficult to socialize and that can negatively influence the early experience of empathy. However,

certain types of parenting (i.e., strong and consistent parenting; responsive parent–child relationship) can help a child with such a temperament overcome this risk and develop healthier levels of guilt and empathy.

Developmental Models for Other Children with Childhood-Onset Conduct Problems

As noted previously, children with CU traits represent only one subgroup of children and adolescents who show disruptive behavior disorders. Thus, the developmental model outlined above may not be useful for explaining the processes involved in the etiology of other children with a childhood-onset to their conduct problems. However, research that has separated those with CU traits from other early-onset antisocial youths has documented several characteristics of those without CU traits that also could help in developing causal models to explain their antisocial and aggressive behavior.

Specifically, antisocial youths without CU traits often show high rates of anxiety (Andershed et al., 2002; Frick et al., 1999; Pardini et al., 2007), they do not typically show problems in empathy and guilt and they appear to be distressed by the effects of their behavior on others (Loney et al., 2003). Thus, the antisocial behavior in this group does not seem to be easily explained by the deficits in conscience development proposed as being critical for understanding the conduct problems in children with CU traits. However, youth with severe conduct problems without CU traits show high levels of impulsivity (Christian et al., 1997; Frick, Cornell, Bodin, et al., 2003), are more likely to show deficits in verbal intelligence (Loney et al., 1998) and are more likely to show a hostile attribution bias in social situations (Frick, Cornell, Bodin, et al., 2003). As noted above, they are also more likely to come from families with high rates of dysfunctional parenting practices (Edens et al., 2008; Hipwell et al., 2007; Oxford et al., 2003; Wootton et al., 1997). Further, this group without CU traits is less likely to be aggressive but, when they are aggressive, it is often confined to reactive forms of aggression (Frick, Cornell, Barry, et al., 2003; Kruh et al., 2005). Also, this group seems to be highly reactive to emotional stimuli (Kimonis, Frick, Fazekas, et al., 2006; Loney et al., 2003; Munoz et al., 2008) and to the distress of others (Pardini et al., 2003).

Given these characteristics, it seems that children without CU traits could have deficits in either the cognitive or emotional regulation of their behavior. Specifically, the deficits in verbal abilities combined with inadequate socializing experiences could result in problems in the executive control of behavior, such as an inability to anticipate the negative consequence to behavior or an inability to delay gratification. Further, the cognitive (e.g., hostile attributional biases) and emotional (e.g., strong reactivity to negative stimuli) characteristics, again combined with inadequate socializing experiences, could lead to problems regulating emotion (Frick, 2006; Frick & Morris, 2004). These problems in emotional regulation could result in the child committing impulsive and unplanned aggressive and antisocial acts for which

he or she may be remorseful afterwards but may still have difficulty controlling in the future.

A Cognitive Neuroscience Approach to CU Traits

One limitation in the developmental model provided for understanding the causes of CU traits is that it does not specify what could lead to the behaviorally uninhibited temperament which places the child at risk for problems in conscience development. As reviewed previously, there is evidence that heredity plays some role, but the available behavioral genetic studies do not indicate what neurological endophenotype or endophenotypes may be inherited and lead to this temperamental style. A cognitive neuroscience perspective could be very beneficial in advancing this aspect of the developmental model. Further, a cognitive neuroscience perspective could help in further understanding the different causal mechanisms involved in the development of severe conduct problems for those youths with and without elevated CU traits.

Cognitive neuroscience, by definition, is concerned with the functional neural architecture (i.e., how components of brain regions interact to achieve particular tasks). A cognitive neuroscience model of a psychiatric condition is not only concerned with what computational processes are impaired in a patient with the disorder (for an example of a cognitive model of CU traits, see Blair, 1995) or what neural systems are dysfunctional in patients with the disorder (for an example of a neuroscience model of CU traits, see Kiehl, 2006). Instead, a cognitive neuroscience model should provide an account of how the computational processes disrupted within specific neural systems can give rise to the development of the disorder (Blair, 2005).

Three core neural systems show indications of dysfunction in youth with CU traits: the amygdala, OFC and, albeit with considerably less data, the caudate. Patients with amygdala lesions show selective impairment for the recognition of fearful expressions (Adolphs, 2002), which as noted previously, are also shown by youth with CU traits (Blair, Colledge, Murray, et al., 2001; Stevens et al., 2001). Moreover, this impairment for the recognition of fearful expressions is reduced in patients with amygdala lesions if the experiment focuses the subject's attention on the eye region of the stimulus (Adolphs et al., 2005), something that is again also seen in youth with CU traits (Dadds et al., 2006). More directly, fMRI studies have shown reduced amygdala responses to fearful expressions in youth with CU traits (Marsh et al., 2008), a result that has been recently replicated (Jones, Laurens, Herba, Barker, & Viding, 2009).

Specific regions of OFC are critical for extinction, reversal learning, and affect-based decision-making more generally (Bechara, Damasio, & Damasio, 2000; Rolls, 1997). Patients with OFC lesions show impairment in extinction (Hornak et al., 2004), reversal learning (Swainson et al., 2000), and decision-making (Bechara et al., 2000). Again, these impairments are similar to those found for youth with CU traits. Specifically, youth with CU traits show impairment in extinction

(Fisher & Blair, 1998; O'Brien & Frick, 1996), reversal learning (Budhani & Blair, 2005), and decision-making (Blair, Colledge, & Mitchell, 2001). More directly, fMRI studies have shown atypical OFC responses during reversal learning (Finger et al., 2008) and simple decision-making (performance on the passive avoidance learning task; Finger et al., 2011) in youth with CU traits.

From a cognitive neuroscience perspective, it is the amygdala's role in stimulus-reinforcement learning and the OFC's role in the representation of reinforcement information and prediction error signaling that are particularly compromised in youth with CU traits (Blair, 2005, 2007). There are considerable data demonstrating that the amygdala allows the formation of stimulus-reinforcement associations (Everitt, Cardinal, Parkinson, & Robbins, 2003; LeDoux, 2007). It is argued that the fearful expressions of others serve as aversive reinforcement, punishers; representations of actions/objects associated with these expressions will be associated with this aversive reinforcement, making the individual less likely to engage in or approach these actions/objects (Blair, 2003). In the context of stimulus-reinforcement-based decision-making (e.g., during passive avoidance learning), the amygdala is thought to feed forward expectancies of reinforcement to OFC to allow successful decision-making to occur. Because of the impairment in stimulus-reinforcement learning and because of dysfunction in the ability of OFC to represent reinforcement information, decision-making is profoundly compromised in children and adolescents with CU traits.

In addition to the OFC's role in the representation of reinforcement information, the OFC, and also the caudate, are critical for the detection of prediction errors (Haruno & Kawato, 2006; O'Doherty, Buchanan, Seymour, & Dolan, 2006; O'Doherty, Dayan, Friston, Critchley, & Dolan, 2003). Prediction errors occur when the individual expects a certain level of reinforcement which is not received (i.e., they receive unexpected levels of reward or punishment). Unexpected rewards are associated with positive prediction errors and increased OFC and caudate activity while unexpected punishments are associated with negative prediction errors and decreased OFC and caudate activity (Haruno & Kawato, 2006; O'Doherty et al., 2003, 2006). Youth with CU traits show indications of dysfunctional OFC and caudate signaling of both positive (Finger et al., 2011) and negative (Finger et al., 2008) prediction error signaling. Importantly, prediction error signaling is critical for rapid learning about the value associated with an action or object (Rescorla & Wagner, 1972). Dysfunctional prediction error signaling will thus exacerbate more basic deficits in stimulus-reinforcement learning and other forms of emotional learning in other systems (e.g., the amygdala).

Two other regions that should be considered, given recent data that common genes explained the phenotypic relationship between them and psychopathic traits (Rijsdijk et al., 2010), are dACC and PCC. Both regions have been considered dysfunctional in adults who show psychopathic traits (Kiehl, 2006). However, as yet, a detailed cognitive neuroscience model of how these regions might be dysfunctional and how this dysfunction might be associated with CU traits has not been provided. Partly, this reflects an absence of detailed models of these two relatively large regions of cortex. One function reliably ascribed to dACC is the resolution of

response conflict (Botvinick, Cohen, & Carter, 2004). However, this function of the dACC appears intact in youth with CU traits. Individuals with CU traits show appropriate recruitment of this region in response to the response conflict punishment error signals during reversal learning (Finger et al., 2008). It is perhaps here where a cognitive neuroscience model becomes most critical. It is unlikely, though not impossible, that all functions of the dACC and PCC are dysfunctional in CU traits. Indeed, it is unlikely that all the functions of the amygdala and OFC are dysfunctional in CU traits. CU traits are not a neurological condition where a particular brain system, or set of systems, is destroyed but rather a psychiatric condition where specific functional roles of specific neural systems are likely compromised while others remain intact. As yet, there have been no demonstrations of impairment in any specific functional process attributed to the dACC or PCC.

Critical Next Steps for Major Advances

Taken together, the research reviewed in this chapter suggests that the presence or absence of CU traits seems to be critical for designating important pathways in the development of disruptive behavior disorders which may involve different social, emotional, cognitive, and biological risk factors. These theoretical models point the way to several potentially important directions for future research. For example, a key component to the developmental models outlined in this manuscript relates to the different temperaments (e.g., fearlessness and low behavioral inhibition; high levels of emotional reactivity) and related neurological systems (e.g., reduced amygdala responses; abnormal responses of the OFC) that may place a child at risk for manifesting severe antisocial and aggressive behavior. However, the vast majority of research has focused on children and adolescents who already show disruptive behaviors. As a result, it will be critical for future research to study children with the hypothesized temperamental or biological risk factors early in life to determine how well they predict later CU traits and severe antisocial behavior. Such prospective research is not only important for providing strong tests of the predictive utility of the developmental model, but this research could also help to uncover other protective factors that may reduce the likelihood that a child with a temperamental risk factor will show severe disruptive behavior problems.

As for treatment implications, although much of the existing research on treating youths with CU traits has focused on the difficulty in successfully altering their chronic antisocial and aggressive behavior (Frick & Dickens, 2006), we reviewed several studies which have demonstrated some success in treating children and adolescents with CU traits (Caldwell et al., 2006; Hawes & Dadds, 2005; Waschbusch et al., 2007). Importantly, these studies have consistently tailored their approaches to treating children with CU traits based on the findings of the unique behavioral, emotional, and cognitive characteristics of these youth. Thus, it is critical that basic research on children with CU traits continues to be used to advance an evidence-based approach to treatment. Further, more treatment studies are critically needed

that attempt to tailor their intervention to the specific needs of children with CU traits. For example, two treatment methods which were designed to provide comprehensive and individualized treatments for antisocial children and adolescents are Multisystemic Therapy (Henggeler & Lee, 2003) and Functional Family Therapy (Alexander & Parsons, 1982). Both treatments have proven to be successful in treating adolescents with even very severe antisocial behavior (Gordon, Graves, & Arbuthnot, 1995; Henggeler, Pickrel, & Brondino, 1999). However, it has not been tested whether they work equally well for youths with and without CU traits. Further, if they are successful for children and adolescents from the different developmental pathways, it would be important to document what components led to success for those in each group.

For treatments to be tailored to the unique needs of children and adolescents with CU traits, however, it is also critical that methods for assessing these traits be advanced. CU traits have been assessed using several different formats, including parent and teacher ratings scales (Frick et al., 2000; Lynam, 1997), self-report scales (Andershed et al., 2002; Munoz & Frick, 2007), parent and youth structured interviews (Lahey et al., 2008), and clinician ratings (Forth, Kosson, & Hare, 2003). Unfortunately, most of these measures have included only a limited number of items specifically assessing this dimension, often with as few as four (Forth et al., 2003) or six (Frick & Hare, 2001) items specifically assessing CU traits. Further, and possibly owing to this limited item pool, measures of CU traits often have had some significant psychometric limitations, such as displaying poor internal consistency in some response formats (Poythress et al., 2006).

A more extended assessment of CU traits using 24 items has been developed and its factor structure has been tested in non-referred samples of adolescents in Germany ($n = 1{,}443$; Essau et al., 2006), Belgium ($n = 455$; Roose et al., 2010), and Greek Cyprus ($n = 347$; Fanti et al., 2009) and in a sample of juvenile offenders in the United States ($n = 248$; Kimonis et al., 2008). Across all four samples using four different languages, a very similar bi-factor structure seemed to fit the data best, with a general CU factor accounting for covariance among all items and three independent subfactors (i.e., uncaring, callous, and unemotional) reflecting unique patterns of covariance among particular groups of items. Importantly, the total scores from this measure proved to be internally consistent in all samples ($\alpha = 0.73$–0.89) and was consistently associated with several measures of antisocial and aggressive behavior, suggesting that this extended measure of CU traits may overcome some of the limitations of past measures with more limited item content.

As with treatment, assessing youth with CU traits could also be aided by experimental research. For example, Kimonis, Frick, Munoz, and Aucoin (2007) reported that in a sample of 88 detained adolescent boys, a self-report measure of CU traits was associated with measures of aggression and delinquency severity. However, when scores on a laboratory measure of youths' responsiveness to distress cues were included in the prediction of the various outcomes, the combination of high self-reported CU traits and reduced responsiveness to distress cues showed the best prediction of self-reported proactive aggression, self-reported violent delinquency, and official records of violent arrests. Thus, the combination of the self-report with

a laboratory measure of emotional processing showed stronger associations with these important outcomes than either of these methods alone. Future studies are needed to determine what combination of assessment techniques and formats provides the best method for assessing children and adolescents with these traits. To promote further advancements in assessment practices, as well as to encourage additional basic research on this subgroup of antisocial youths, it is critical that the importance of CU traits for designating a distinct group of antisocial youth be recognized in diagnostic criteria. This is best illustrated by a study of 7,977 children ages 5–16 from the United Kingdom (Rowe et al., 2009). In this large nationally representative sample, 2 % of the sample were diagnosed with Conduct Disorder and 46 % of these youth also showed elevated CU traits. Importantly, the group high on CU traits showed a more severe behavioral disturbance (e.g., more conduct problems and less prosocial behavior) and was at substantially higher risk for being rediagnosed with Conduct Disorder 3 years later.

Thus, this research suggests that the diagnostic criteria for Conduct Disorder would be enhanced by including some method for designating youth with this disorder who also display significant levels of CU traits. Unfortunately, much of the research to date on CU traits has used dimensional scales that make it hard to translate findings into specific diagnostic criteria. Also, it is critical that such an approach avoids some of the problems associated with previous attempts to integrate these traits into diagnostic classification systems, such as ensuring that the name clearly reflects the core behavioral characteristics of these youths and that only items that are most reflective of this construct based on recent research be used to define this subgroup of youths with disruptive behavior disorders.

Conclusions About State of Knowledge and Implications for Cognitive Neuroscience Research

As reviewed above, the evidence for distinguishing between youth with Conduct Disorder with and without CU traits is now compelling. Such a differentiation is supported by predictive validity (prediction of mid- and long-term stability of conduct problems, aggression, psychopathic traits, and antisocial behaviors); differential treatment response (lack of response when parents were taught more effective discipline strategies (Hawes & Dadds, 2005); differential improvement from adjunctive stimulants (Waschbusch et al., 2007); improvement when intensive reward-oriented approaches applied (Caldwell et al., 2006)); differential relationships with trait anxiety, impulsivity, and autonomic reactivity, differential patterns of heritability (e.g., Viding et al., 2008); neuro-cognitive impairments (reduced orienting to distress cues (Kimonis, Frick, Fazekas, et al., 2006; Kimonis et al., 2008)); abnormalities in reversal learning (e.g., Blair, Colledge, & Mitchell, 2001; Budhani & Blair, 2005); and most recently by heritable variations in gray matter concentration (Rijsdijk et al., 2010).

While each individual result may be debated, the breadth and depth of the evidence supporting the clinical, developmental, psychological, and neurobiological importance of distinguishing youth with conduct problems by the presence or absence of CU traits can no longer be ignored. The very mass of evidence points to the one factor that has long prevented the broader acceptance of distinguishing on the basis of CU traits—the understandable concern that such a designation would become an indelible mark of deterministic condemnation and an invitation to "lock them up and throw away the key." This partly reflects the conviction that entrenched antisocial behaviors, and particularly those often characterized as "psychopathic," are immune to treatment, and that the only rational response is to protect the larger society from such predatory individuals.

Fortunately, the very data that provide the basis for insisting on the importance of quantifying CU traits also suggests that the picture is not so bleak, at least when the individuals in question are still children or adolescents. The long-term stability of CU traits is modest and is not equivalent to immutable destiny. The extant data suggest that the majority of youth with elevated CU traits do not proceed to manifest the most malignant outcomes. Such results highlight the importance of further improving predictive ability so as to best target those at the greatest risk of the worst outcomes.

Such critically needed advances are now feasible and, as argued above, could be aid greatly by a concerted application of developmental cognitive neuroscience approaches. While our ignorance is still vast, identification of some of the core neural structures/systems implicated in Conduct Disorder with CU traits represents a hard won achievement. The leading candidate regions are the amygdala, OFC, caudate nucleus, and the anterior as well as the posterior cingulate cortices. An urgent priority for the field is the formulation of testable mechanistic hypotheses that can inform our understanding of the information processing that is subserved by these regions, which are all involved in the emotional and/or cognitive regulation of affect and behavior. As if that were straightforward, the field also needs to be able to do so in the context of early development, ideally starting in preschool, and while taking into account the ecological contributions of family and community. Posing such an imposing challenge would have been an invitation to resignation until recently. But if it may be said that an army marches on its stomach, then psychology and cognitive neuroscience depend equally crucially on the psychometric properties of the phenotypes of interest. One important reason for optimism, then, is the broad collaborative validation of the Inventory of Callous-Unemotional Traits (e.g., Kimonis et al., 2008). The availability of an accepted validated instrument that is amenable to international use provides an essential basis for large-scale collaborations. These conditions then permit the formulation of a high-risk, high-reward collaborative research endeavor to harness recent developments in developmental psychopathology, cognitive neuroscience, and a particular type of functional brain imaging.

Although brain imaging represents some of the best technology available to developmental scientists, it still resembles nineteenth century daguerreotypes in the requirement that participants remain extraordinarily still for 6–10 min at a time. In the foreseeable future, techniques such as real-time motion correction will likely

make this requirement obsolete, but such methods are not yet available for widespread use. Beyond the problem posed by participant motion, constructing tasks that can be performed during scanning by a wide age range is also a challenge of the first order. Fortunately, a deceptively simple technique, generally known as "resting-state" functional magnetic resonance imaging (R-fMRI), has come into its own as a complement to traditional task-based functional imaging (Fox & Raichle, 2007). The chief advantages of R-fMRI are, first, that no specific task, other than remaining still, is required. Second, R-fMRI data turn out to be extraordinarily revealing of the latent functional architecture of the brain; that is, R-fMRI analyses delineate functional circuits in their entirety (e.g., Fox et al., 2005; Fox, Corbetta, Snyder, Vincent, & Raichle, 2006; Di Martino et al., 2008; Krienen & Buckner, 2009; Margulies et al., 2007, 2009; Roy et al., 2009; Vincent, Kahn, Snyder, Raichle, & Buckner, 2008; Vincent, Kahn, Van Essen, & Buckner, 2010). Third, R-fMRI indices are remarkably sensitive to developmental effects (Fair et al., 2007, 2008, 2009; Kelly, Di Martino, et al., 2009; Supekar, Musen, & Menon, 2009). Fourth, R-fMRI data, despite the lack of a constraining task, are surprisingly reliable over intervals as long as 4–16 months (Shehzad et al., 2009; Van Dijk et al., 2009; Zuo, Di Martino, et al., 2010; Zuo, Kelly, et al., 2010). Fifth, R-fMRI indices appear to be tightly linked to inter-individual variations in enduring traits (Di Martino et al., 2009). Finally, R-fMRI data are particularly amenable to aggregation across multiple imaging centers (Biswal et al., 2010; Tomasi & Volkow, 2010).

Further enhancing the feasibility of an ambitious collaborative plan of research, the brain regions that are most implicated in CU traits in the context of Conduct Disorder have all been mapped via R-fMRI in young adult participants. These include the amygdala (Etkin, Prater, Schatzberg, Menon, & Greicius, 2009; Roy et al., 2009), OFC (Tau et al., unpublished data), caudate nucleus (Di Martino et al., 2008), anterior cingulate cortex (Margulies et al., 2007), and posterior cingulate/precuneus (Margulies et al., 2009).

Thus the next step for the field will be delineating the developmental trajectories of the corresponding circuits as defined by functional connectivity and related techniques. In parallel, the field should begin to collect standard R-fMRI data sets in conjunction with any MRI research studies being conducted with youth with conduct problems with or without CU traits.

Specific imaging parameters must be determined locally in accordance with magnet and gradient coil properties. However, some guidelines can be provided based on optimization analyses (Van Dijk et al., 2009) and practical experience (Biswal et al., 2010). R-fMRI scans below 5 min in duration demonstrate substantial deterioration in test-retest reliability. In general 6 or 6.5 min are recommended to obtain at least 150 individual time points (also known as volumes), since the essence of the technique depends on analysis of those fMRI time series. Whenever possible, whole brain coverage, including the cerebellum, should be attempted. Examinations of the amygdala and OFC require particular attention to preventing signal drop out from the air-brain interfaces of the nearby sinuses. Finally, the lack of a task does not mean that R-fMRI is not influenced by prior experience. To the contrary, R-fMRI data appear to represent a complex integration of current, recent

(Barnes, Bullmore, & Suckling, 2009), and remote experience and influences (Achard & Bullmore, 2007; Kelly, de Zubicaray, et al., 2009). Thus, experimental control in terms of arousal level (eyes open or closed; awake or purposefully asleep), psychotropic medication use, and standardization of temporal placement during scan sessions are also strongly recommended.

In summary, differentiating Conduct Disorder based on the presence or absence of CU traits has now been thoroughly and compellingly established. The weight of evidence is being taken into account in the ongoing fifth revision of the DSM, and it is likely that such a distinction will be incorporated once again into the psychiatric nosology in 2013. In the meantime, the clinical and research importance of such a differentiation also compel continued progress. One area of particular potential traction is represented by the availability of a thoroughly validated instrument for quantifying CU traits. Combined with continued progress in genetics and task-based cognitive neuroscience, the exponentially growing field of "resting-state" fMRI provides the opportunity for a quantum jump in our ability to specify and test more accurate neuro-cognitive models. Such information, when combined with existing emotional, behavioral, and contextual data, will lead to more complete models of developmental pathophysiology. As noted above, when interventions have been linked to research findings on the unique characteristics of youth with CU traits, there is reason for optimism that a heretofore group of youths who were often viewed as "untreatable" may in fact be quite treatable; when the right treatment is employed.

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Chapter 5
A Multidimensional Approach to Disruptive Behaviors: Informing Life Span Research from an Early Childhood Perspective

Alice S. Carter, Sarah A.O. Gray, Raymond H. Baillargeon, and Lauren S. Wakschlag

The historical use of categorical diagnoses of disruptive behavior syndromes and disorders has been integral to clinical identification, treatment, and service utilization. The major nosological frameworks for classification have been the Diagnostic and Statistical Manual (DSM) (American Psychiatric Association, 2000) and International Classification of Diseases (World Health Organization, 2000). Increasingly, however, there is consensus that categorical approaches, which rely on an array of symptom criteria to classify an individual as having or not having a single disorder, may not fully capture clinical and developmental patterns of disruptive behaviors across the life cycle (Baillargeon, Zoccolillo, et al., 2007; Frick & White, 2008; Maughan, 2005; Rutter, 2003; Wakschlag et al., 2011). In contrast, multidimensional conceptualizations of psychopathology, which incorporate more than one domain or dimension of behavior and assess each domain/dimension along a continuum, offer many unique advantages to clinical characterization of disruptive behavior, including (1) improved characterization of heterogeneity, (2) provision of alternative strategies for understanding developmental course, (3) parsing the

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manner in which different components or dimensions of disruptive behavior may have varying associations with co-occurring symptoms, and (4) linkage of specific dimensions relevant to disruptive behavior to neurobiologic mechanisms as well as family and ecological contextual factors.

In this chapter, we propose a novel, developmentally based, multidimensional approach to disruptive behavior that can be applied across the life span to highlight the advantages of multidimensional versus dichotomous characterization. The specific dimensions identified within our multidimensional conceptualization of disruptive behaviors have strong support in the literature, but there is only preliminary work supporting the integrative approach that we present in this chapter. As a foundation, we first (a) highlight key findings in the history of categorical approaches to assessment of disruptive behavior disorders (DBDs), emphasizing research on subtypes that inform identification of salient dimensional components of disruptive behavior, (b) synthesize extant research and theory on dimensional approaches to disruptive behavior, and (c) review the advantages of adopting a multidimensional approach for deeper understanding of clinically significant disruptive behaviors. Following an elaboration of our multidimensional model of disruptive behavior, we conclude with a discussion of emerging areas of knowledge and critical next steps for scientific advancement. Although our approach is a life span framework, we focus particularly on early childhood to elucidate the framework—in part because of the particular complexities in the distinction between normative misbehavior and clinically concerning misbehavior in this period and in part because multidimensional inquiry about clinically significant disruptive behavior in early childhood has received more limited attention than inquiry about older children and adults.

A History of Categorical Approaches to Disruptive Behaviors

Diagnoses

Tracing the nosological history of DBDs highlights one challenge of developing an empirical knowledge base for investigating disruptive behavior. The shifting terrains of diagnostic conceptualizations have made it difficult to accumulate systematic knowledge about the prevalence and stability of disruptive diagnoses over time (Robins, 1999). The first edition of the DSM, published in 1952, included no childhood diagnoses. With the publication of DSM-II in 1968, disruptive behavior was captured in the diagnoses of runaway reaction, unsocialized aggressive reaction, and group delinquent reaction. Published in 1969, ICD-8 included the umbrella diagnosis of behavior disorders of childhood, which was further expanded in 1977s ICD-9 to include ten categories and one V-code. DSM-III (1980) saw the introduction of conduct disorder (CD). Oppositional disorder also first appeared in *DSM-III*, with "defiant" added to the clinical construct in the text revision. ICD-10 (1990) was modified to reflect DSM's formulation, with oppositional defiant disorder (ODD) under the larger umbrella of CDs. DSM-IV (1994) included further

modifications to definitional specification through revisions to symptom counts and descriptions (Costello & Angold, 2001; Robins, 1999).

DSM IV-TR parses DBDs into oppositional and conduct problems. ODD is defined by irritable disposition and resistant interactions with authority figures, whereas CD is defined more by disregard for social norms, rules, and the rights and wellbeing of others (as well as more physical aggression) (Wakschlag, Leventhal, Thomas, & Pine, 2007). The core diagnostic features of these disorders have stayed relatively steady over the past four editions of *DSM*, though changes in specific symptoms have led to fluctuations in prevalence rates. The newer diagnostic nosological system, developed for very young children by a consensus panel of experts in infant mental health (DC:0–3R multiaxial system) to address perceived gaps in the DSM and ICD systems, largely defers to the DSM when young children present with disruptive problems. However, there may be some overlap between the DSM-IV diagnoses of both ODD and CD and the DC:0–3 diagnosis of regulation disorders of sensory processing Type B—Negative/Defiant, particularly when coupled with a parent–child interaction disturbance. Of note, assigning a diagnosis of regulation disorders of sensory processing requires the presence of a constitutional or maturational etiology and at the present time specific criteria for determining subtypes are not available (Zero to Three, 2005).

Currently, the two diagnoses of behavior problems in the DSM—CD and ODD—are conceived of as a developmental sequence: a diagnosis of CD precludes a diagnosis of ODD because the assumption is that there is a developmental progression from ODD to CD. Longitudinal studies from clinic-referred samples of older children have supported this assumption: children diagnosed with ODD are at significantly increased risk of developing CD (Burns et al., 1997; Lahey, McBurnett, & Loeber, 2000; Pillow, Pelham, Hoza, Molina, & Stultz, 1998; Rowe, Maughan, Pickles, Costello, & Angold, 2002). They are also at increased risk for developing other disorders, such as anxiety and depression (Burke, Loeber, Lahey, & Rathouz, 2005). These findings highlight the importance of understanding, identifying, and intervening with clinically significant disruptive behavior early in its course.

The vast majority of studies of younger children have focused solely on ODD, because of concerns about the developmental applicability of CD to young children (Campbell, 2006; Keenan et al., 2007; Kim-Cohen et al., 2005; Wakschlag, Briggs-Gowan, et al., 2007). Thus, the validity of the ODD:CD distinction in young children remains unknown. Results from a factor analytic study of DSM symptoms support a single disruptive behavior syndrome in preschoolers (Sterba, Egger, & Angold, 2007). Further, the developmental sequence model makes little sense in young children when oppositional and conduct problems emerge simultaneously.

Subtypes

Early work on delineating the varied presentations of disruptive behavior focused on disruptive behavior "subtypes." Indeed, distinctions among disruptive behavior presentations are as old as the study of disruptive behaviors themselves. The parallel

between subtype and dimensional approaches is in their joint recognition of systematic heterogeneity within disruptive behaviors, which informs understanding of severity, course, and treatment. Moreover, identification of subtypes can inform selection of core defining features of disruptive behaviors. The difference between the two approaches is that subtypes focus on identifying subsets of individuals whereas dimensional approaches focus on identifying relevant subsets of behaviors.

Beginning with Hewitt and Jenkins's (1946) distinction between "socialized" and "unsocialized" delinquent behavior, researchers have described a host of potential subtypes of presentations of disruptive behaviors (Hewitt & Jenkins, 1946). Individuals with "socialized" and "unsocialized" delinquent behavior were described as distinguishable on perspective-taking, abstract reasoning, and empathy (Quay, Routh, & Shapiro, 1987). This distinction was presented in the *DSM-III* and in ICD-9 and -10 as a potential subtype.

A robust body of research addresses the delineation of CD subtypes based on age at onset (Moffitt, 1993). "Early onset" conduct problems (i.e., life-course-persistent) may have unique etiology and neurodevelopmental correlates from adolescent-limited conduct problems (Moffitt & Caspi, 2001). *DSM-IV* acknowledges this distinction as possible subtypes within the nosology of CD. The childhood versus adolescent onset distinction has been widely validated, replicated, and extended. Specifically, individuals with early onset of CD are more likely to have experienced perinatal complications, undercontrolled temperament, neurological abnormalities, and delayed motor development in early childhood. They are also more likely to have low intellectual ability, reading difficulties, low scores on neuropsychological tests of memory, hyperactivity, and slow heart rate in later childhood (Moffitt, 2006). Early versus late onset CD is more strongly associated with physical aggression and, by definition, a more persistent presentation (Lahey & Loeber, 1997).

Another subtype distinction that has been made is between presentations characterized by aggressive versus nonaggressive behaviors. This categorical distinction is supported by factor analytic work (Achenbach, Conners, Quay, Verhulst, & Howell, 1989; Frick et al., 1991; Tackett, Krueger, Sawyer, & Graetz, 2003). Aggressive conduct problems include fighting, physical cruelty, and violent behavior whereas nonaggressive conduct problems include nonviolent delinquent behaviors such as illegal acts and status violations (e.g., breaking curfew), and defiance. These two subtypes have been shown to have disparate etiologic correlates, with nonaggressive rule-breaking behavior appearing to be much more influenced by environmental factors than aggressive conduct problems (Tackett, Krueger, & Iacono, 2005). Person-centered analyses in a representative sample have further confirmed that persistent aggressive and nonaggressive disruptive behaviors tend not to overlap in boys, but the distinction is less clear for girls (e.g., only 12.6 % of boys but 43.3 % of girls with stable high aggressive behaviors were also stably high in nonaggressive behavior problems). Moreover, aggressive disruptive behavior was associated with unique environmental risk factors; among them were poverty, low parental supervision, and parental criminality (Maughan, Pickles, Rowe, Costello, & Angold, 2000).

Classic work by Loeber et al. distinguishes between three subtypes of disruptive behaviors in childhood: overt (e.g., confrontational, such as fighting); covert (e.g., concealing, such as stealing or lying); and "authority conflict" (e.g., disobedience or

defiance) (Loeber et al., 1993). In a prospective study of symptoms of CD, fighting—an overt behavior—was the best predictor of the onset of CD (Loeber et al., 1998). A further distinction in overt aggressive behavior between reactive and proactive aggression appears to have implications for the developmental course of disruptive behavior: proactive aggression appears particularly predictive of later maladjustment and diagnosis of CD (Loeber, Burke, Lahey, Winters, & Zera, 2000; Loeber & Farrington, 2000).

Finally, a seminal body of work by Frick and colleagues that addresses the roots of psychopathy in children's disruptive behavior has looked at callous and unemotional traits among a subgroup of children with disruptive behaviors as a possible causal pathway through which some children develop severe conduct problems (Frick et al., 2003). Callous and unemotional traits include a lack of empathy or concern for others, a lack of guilt over transgressions, and insensitive use of others for personal gain. These traits appear to be relatively stable across childhood and adolescence and are associated with a unique set of temperamental, physiological, and clinical attributes. These attributes include a temperamental style characterized by thrill-seeking and fearlessness, elevated reactivity to others as well as reactive aggression, and more severe conduct and aggression problems (Frick & White, 2008). These traits have also been linked to specific neurodevelopmental differences in the amygdala (Marsh & Blair, 2008).

These pioneering efforts have clearly demonstrated the heterogeneity of presentation of disruptive behaviors. However, despite identifying and focusing on a core feature of disruptive behavior that helps to clarify systematic heterogeneity in essential clinical characteristics (e.g., empathy, persistence), each subtype effort focuses on a single component of disruptive behavior. Thus, none of these frameworks adopts a multidimensional approach that attempts to capture multiple component features nor are developmental shifts in presentation considered.

Ideally, a more complete understanding of disruptive behavior might begin with characterization of normative and emerging developmental processes—of emotion regulation, empathy and conscience development, the balance of autonomy and compliance, and the modulation of aggression. Once normative understanding is established, a next step would be to determine the points at which and what goes awry in the process of development that leads to the combination of dimensions that cause us to conclude that the child's emotional and behavioral presentation is consistent with "disorder" status. We believe that a developmentally sensitive, multidimensional approach is uniquely suited for addressing these gaps.

What Do We Know About Disruptive Behavior in Young Children?

In terms of diagnostic nosology, there has been increasing acknowledgement that disruptive behaviors emerge in early childhood and are of sufficient severity in some children to meet diagnostic criteria (Baillargeon, Zoccolillo, et al., 2007; Carter, Briggs-Gowan, & Davis, 2004). Among preschoolers, diagnostic construct validity

is supported by findings such as that preschoolers meeting DBD symptom criteria are more than 20 times as likely to be impaired by parent report and more than twice as likely to be impaired by teacher report (Keenan et al., 2007). Moreover, DBD symptoms are consistent with observed behavior on developmentally sensitive assessments (Wakschlag, Briggs-Gowan, et al., 2007) and by young child self-report on the Berkeley Puppet Inventory (Kim-Cohen et al., 2005). DBD symptoms also demonstrate stability (Lavigne, Cicchetti, Gibbons, Binns, & DeVito, 2001).

We also know that continuous dimensional measurement can be applied to these disruptive behaviors reliably for toddlers as well as preschoolers (Achenbach & Rescorla, 2004; Carter, Briggs-Gowan, Jones, & Little, 2003). Disruptive behavior problems, when assessed continuously, are relatively stable and heritable (Chacko, Wakschlag, Espy, Hill, & Danis, 2009; Moreland & Dumas, 2008). Although Bennett et al. (1999) have argued that the positive predictive accuracy of these behaviors is relatively low, Baillargeon and colleagues have demonstrated more stability in these behaviors among younger children by correcting for attenuation (Baillargeon et al., 2004); for example, 80 % of children who exhibited physically aggressive behaviors on a frequent basis at 17 months were still doing so at 29 months of age (Baillargeon, Zoccolillo, et al., 2007). However, these differing findings highlight that there is both continuity and discontinuity in these patterns. DBD symptoms have also been shown to be responsive to empirically validated treatments for disruptive behavior (Webster-Stratton & Reid, 2007).

Investigators have approached the issue of distinguishing normative and nonnormative behaviors using both diagnostic and dimensional approaches. Identifying clinical concern in early childhood turns on "deviation from the norm," and increasing evidence from population-based samples and developmental research has helped outline the contours of these norms (Baillargeon, Zoccolillo, et al., 2007; Briggs-Gowan, Carter, Skuban, & Horwitz, 2001; Tremblay & Nagin, 2005). In very early childhood, dimensional work in large representative samples has demonstrated that normative misbehavior can be distinguished from atypical misbehavior through subjective frequency reports, as high frequencies of misbehavior ("often" as opposed to "never" or "sometimes") are atypical (Baillargeon, Zoccolillo, et al., 2007; Carter et al., 2003; Hay, Castle, & Davies, 2000; Tremblay et al., 2004). For example, in parent report of behavior of 17-month-old children in a population-based sample, approximately half of children are "sometimes defiant," whereas only 10 % of children are "often" defiant (Baillargeon, Normand, et al., 2007). In another large sample, less than 10 % of 2-year-olds "often" hit others (Carter et al., 2003). Investigators have defined deviation from the norm both as a chronic deviation, demonstrating a disruptive behavior more frequently than usual over an extended period of time (Tremblay, 2010), and as exhibiting many disruptive behaviors within a single domain (e.g., many aggressive behaviors) on a frequent/severe basis (Baillargeon, Zoccolillo, et al., 2007). For example, 5 % of boys and 1 % of girls in the general population exhibit a number of different physically aggressive behaviors on a frequent basis at 17 months of age (Baillargeon, Zoccolillo, et al., 2007). Similarly, 12.4 % of toddlers exhibit different oppositional defiant behaviors on a frequent basis at this age (Baillargeon, Sward, Keenan, & Cao, 2011).

Moreover, subtypes of disruptive behavior can be identified even before 2 years of age. Baillargeon et al. demonstrated that almost all toddlers with a significant aggression problem also exhibited oppositional defiant behaviors on a frequent basis, but only a minority of toddlers with a significant opposition-defiance problem also exhibited aggressive behaviors on a frequent basis, suggesting that even before 2 years of age, oppositionality and physical aggression are distinct components of disruptive behavior (Baillargeon et al., 2011). Such knowledge has been supported by advancements in measurement that provide the field with increasingly precise and developmentally informed tools for describing and measuring disruptive behaviors in younger children (DelCarmen-Wiggins & Carter, 2004).

Advances in statistical modeling of developmental trajectories have also supported more nuanced pictures of patterns of disruptive behavior into the earlier years of childhood (Nagin & Tremblay, 1999). Supporting a multidimensional approach to disruptive behaviors, trajectories of divergent components of disruptive behavior evidence unique developmental patterns, with, for example, trajectories of early physical aggression looking quite different from trajectories of early opposition-defiance (Tremblay, 2010). These divergences have led Tremblay to argue that the collapse of disruptive behaviors into one construct means the loss of important developmental data.

What Do We Know About Multidimensional Approaches to Disruptive Behavior?

A burgeoning body of work in disruptive behaviors is now focused on identifying the specific dimensions that constitute disruptive behavior in young children. Factor analytic methods among older children by Burke and colleagues have demonstrated two dimensions salient for ODD among boys (negative affect and oppositional behavior) and three dimensions salient for ODD among girls (oppositional behavior, negative affect, and antagonistic behavior) (Burke, Hipwell, & Loeber, 2010). These dimensions among boys and girls predict different diagnostic outcomes, with the negative affect dimension predicting later diagnosis of depression even after controlling for earlier depression. There is also evidence from a twin study that different factor analytically derived dimensions of CD might have unique etiologies, with nonaggressive rule-breaking showing more contribution from family environment and aggressive behavior showing more influence from genetic factors (Tackett et al., 2005).

Working from an a priori theoretical frame, Stringaris et al. have hypothesized three unique dimensions of oppositionality—irritable, headstrong, and hurtful—and have found these dimensions to be related to unique correlates and developmental diagnostic courses of disruptive behaviors among children between the ages of 5 and 16, with irritability predicting depression and anxiety, headstrong predicting ADHD and nonaggressive CD, and hurtful predicting aggressive CD (Stringaris & Goodman, 2009a, 2009b). As an explanation of these divergent trajectories,

Stringaris et al. propose a "convergence-divergence" model in which various etiological factors such as temperamental or biological predispositions to elevated activity and/or emotionality combine with environmental stressors to converge on the ODD diagnosis, and then diverge into distinct distal trajectories (Stringaris, Maughan, & Goodman, 2010). As this work demonstrates, employing a multidimensional model leads to a more nuanced clinical picture that captures the heterogeneity of children with disruptive behaviors relatively early in childhood and can begin to anticipate their developmental trajectories.

Ideally, rather than assuming a priori which dimensions are central to disruptive behaviors and subtyping based on one of these dimensions, children with clinically concerning disruptive behaviors can be subtyped based on their functioning across multiple dimensions that are relevant to the etiology and course of disruptive behaviors. Longitudinal data on large, representative groups of children could be gathered so that subgroups can be based on profiles of trajectories of dimensions found to be central to disruptive behavior. Stringaris et al.'s work highlights the promise of a multidimensional approach for predicting and capturing the heterogeneity of developmental pathways and clinical phenomenology. However, more developmental work is needed to ensure adequate representation of preschool-aged children and to capture the full disruptive behavior syndrome (i.e., expanding the work beyond a focus on ODD). Given the breadth of work on the components of disruptive behaviors, several distinct multidimensional models could be put forth as theoretically sound and based on extant empirical evidence. Thus, future work will be necessary to test the alternative multidimensional models that we anticipate will be proposed.

Advantages to Developmental, Dimensional Approaches to Disruptive Behavior

Although there has been tension between a categorical and dimensional approach to psychopathology for at least 60 years (Quay et al., 1987), there appears to be increasing emphasis on dimensional approaches to psychopathology, including preparations for DSM-V (Hudziak, Achenbach, Althoff, & Pine, 2007; Krueger & Bezdjian, 2009). In our proposed multidimensional model of disruptive behavior, we focus on capturing two axes: (1) Axis I comprises a single continuous dimension that addresses severity, irrespective of the specific disruptive behavior symptoms or the patterning of dimensions and (2) Axis II comprises the multiple interrelated components of disruptive behavior, each measured dimensionally. Both of these axes demand a developmental perspective or normative frame. The normative standards for quantifying severity of disruptive behavior shift across the life span. Consistent with the tenets of developmental psychopathology and expectations for heterotypic continuity within dimensions (Cicchetti & Rogosch, 1996; Rutter & Sroufe, 2000), the specific behaviors that comprise Axis II's core disruptive behavior dimensions and the contexts in which they are optimally assessed will change across the life span (see Table 5.1). Developmentally sensitive assessment of both

Table 5.1 Example of developmental manifestations of disruptive behavior dimensional components

	Early childhood	School age	Adolescent	Adult
Temper loss	Breaks or destroys things during "meltdowns"	Has frequent temper tantrums	Often has outbursts in response to routine requests	Is explosive
Noncompliance	Has a "reflexive no"—i.e., says "no" even before hearing what's asked	Pervasively resists completing schoolwork	Flagrantly disobedient	Is frequently argumentative with supervisors
Aggression	Pinches/hurts other children when adult is not looking	Starts fights with peers when "unprovoked"	Bullies others	Has aggressive relationships

severity and multiple dimensions of disruptive behavior is critical to understanding the etiology, course, and treatment of clinically significant disruptive behaviors.

A Developmental Framework for Conceptualizing Disruptive Behavior

The developmental psychopathology approach defines psychopathology as deviations from normative patterns. This approach necessitates grounding the study of disruptive behaviors within normative developmental expectations. Fundamentally, this requires distinguishing between normative misbehavior (i.e., age-typical manifestations of the components that characterize disruptive behaviors) and clinically significant maladaptive patterns that indicate that the child's development is at risk or of clinical concern (Wakschlag, Briggs-Gowan, et al., 2007). However, to date, this approach has largely been theoretical and has not been systematically applied to clinical classification systems (Wakschlag, Tolan, & Leventhal, 2010).

Adopting a developmental frame is critical to understanding disruptive behavior: behavior that is normal or expected during one developmental stage might be considered clinically of concern at another age, and vice versa (Hudziak et al., 2007). In the relatively adevelopmental categorical framework of DSM, however, as we have previously noted, approximately one-fourth of CD symptoms are *developmentally impossible* (e.g., forcible sexual activity, truancy); approximately one-third of CD symptoms are *developmentally improbable* (e.g., fire-setting, stealing); and the remaining symptoms are largely *developmentally imprecise* due to high normative

base rates of occurrence (e.g., "often loses temper") (Wakschlag, Leventhal, et al., 2007). Reliance on a diagnostic nosology that lacks developmental specificity has meant that clinically significant behaviors in early childhood have often been neglected and heterotypic continuity has been difficult to trace through time.

In contrast, framing core components of disruptive behavior dimensionally and in a developmentally meaningful way across periods has the potential to capture varying developmental manifestations while still tapping into the same fundamental atypical processes. For example, the specific symptoms of truancy, a behavior consistently associated with a clinical diagnosis of CD in adolescence, might be conceptualized as falling into a broader dimension of "non-compliance." At other points in the life span, manifestations might include such behaviors as a "reflexive no" in preschool (i.e., the child who is posed to say no—even before hearing what is being asked of him or her) and/or an inability to take direction from supervisors in adulthood. A true life span approach would empirically test for such continuities over time along multiple dimensions, capturing changes in overall severity (Axis I) as well as continuities within and across each of the dimensional components (Axis II) (Wakschlag et al., 2010). Such a developmentally sensitive multidimensional approach permits assessment of within-dimension and disorder heterotypic continuity that might otherwise be missed if the same criteria are employed through the life span.

Of particular relevance to our understanding of disruptive behavior are the following core developmental processes of early childhood: emotion regulation (particularly anger regulation), empathy and conscience development, the balance of autonomy and compliance, and the modulation of aggression. These developmental processes, all at their root directly implicated in social conflicts and therefore implicated in disruptive behaviors, can each be assessed along a continuum from normative to clinically concerning throughout the life span. Children's cognitive, linguistic, and inhibitory skills develop exponentially across early childhood, and with greater maturation, children are thrust into increasingly demanding social situations that require both increasing autonomy and regulation (Wakschlag & Danis, 2009). It is through these processes that the more diffuse reactivity of early infancy is transformed into the more intentional and directed (mis)behaviors of the toddler period (Hay, 2005).

Advantages to Dimensional Assessment

The advantages to dimensional assessment of the severity of psychopathology have been well enumerated in the literature (Hudziak et al., 2007; Krueger & Bezdjian, 2009). While it is appropriate for a life span approach, conceptualizing psychopathology dimensionally has particular relevance for capturing the full range of clinical manifestations of clinically concerning disruptive behaviors in *early* childhood. First, emergent manifestations may be milder and less likely to be captured by rigid symptom thresholds, particularly because clinical symptoms often emphasize the most severe forms of behavior. Moreover, given the relatively adevelopmental criteria of current diagnostic criteria, children with *early* manifestations of disruptive

behavior (e.g., prolonged temper tantrums that are characterized by intense, angry mood) may not fall under the umbrella of symptom criteria for the categorical diagnoses as currently written. Specifying behavior developmentally and along a continuum from normative misbehavior to clinically at risk to of clinical concern enables a more nuanced examination of the point at which typicality and atypicality are demarcated. Further, as has been noted (Campbell, 2006), since misbehaviors are more common at preschool age than in older childhood, it is the *constellation* of behaviors present as well as their frequency and severity that demarcate the threshold of clinical concern, not just the presence or absence of any one behavior.

Advantages to Assessment of Dimensional Components or Multidimensional Assessment of Disruptive Behavior

To better capture constellations of behaviors, the second axis of our model looks beyond a single severity dimension (Axis I) to identify specific dimensional components of disruptive behavior (Axis II). Focusing on multiple specific dimensional components, rather than looking at the broad disruptive behavior syndrome, enables greater specificity in description. Narrowband dimensions of disruptive behavior can be conceptualized in relation to disruptions in specific developmental processes. For example, in the developmental process of emotion regulation, young children's response to frustration may vary along a continuum from autonomously regulated emotions, to expectable outbursts at times of transition, to highly dysregulated temper tantrums in low demand contexts (Belden, Thompson, & Luby, 2008; Kochanska, Coy, & Murray, 2001).

Defining narrowband components of disruptive behavior developmentally may also provide an empirical basis for testing the construct of heterotypic continuity, the notion of underlying latent traits that take on different expressions across development based on capacities and demands (Rutter & Sroufe, 2000). Though often cited, heterotypic continuity has rarely been systematically demonstrated in studies of clinically significant disruptive behaviors (Maughan, 2005; Wakschlag et al., 2010). To the extent to which subtyping based on multidimensional profiles of disruptive behaviors contributes to a more comprehensive and developmentally attuned understanding of disruptive behaviors, it offers promise as well to capture the heterogeneity of symptom presentation over time. While we know that disruptive behaviors in childhood are predictors of future conduct problems, the diagnostic specificity of this prediction is limited. In a study of 251 nonclinical children in kindergarten and first grade, the positive predictive value of externalizing behaviors to a diagnosis of the low-prevalence CD 30 months later was below 50 %, though the positive predictive value increased when contextual risk factors such as maternal psychopathology were taken into account (Bennett et al., 1999). In other words, simply measuring externalizing behaviors in kindergarten does not meet the standards of prevention science to advocate universal screening and targeted intervention because misclassification is likely to occur.

Part of the explanation for this poor prediction may be that categorical diagnoses may not capture the full range of meaningful behavior or may not capture behavior with adequate specificity. For example, a study of the 5-year predictive validity of CD found that a majority of children diagnosed with CD at age 5 no longer had CD symptoms at age 10. However, these children continued to demonstrate behavioral difficulties and psychoeducational impairment (Kim-Cohen et al., 2009). This finding suggests that the current diagnostic category of CD may not be capturing one set of stable behaviors over time, but may be indicative of a future course that takes on a different, but still impairing form (Kim-Cohen et al., 2009). Further, the lack of stability may also reflect the fact that many children who will later meet diagnostic criteria for CD may be misclassified (i.e., not meeting the diagnostic criteria) at age 5 due to the adevelopmental frame of the current nosology, which would explain the presence of false positives at age 5 contributing to the observed low positive predictive value.

Research on specific components of disruptive behavior shows promise in identifying potential heterotypic manifestations of disruptive behavior. For example, work by Shaw and colleagues documents that fearlessness at age 2 predicted conduct problems in early and middle childhood (Shaw, Gilliom, Ingoldsby, & Nagin, 2003). This same study highlights how careful measurement along the range of a normative developmental process (here, fear/fearlessness) at a particular point in development can aid in identifying youth at risk for later psychopathology. Conceptualizing psychopathology and/or clinically significant behavior problems in terms of deviation from normative processes as well as with respect to extreme or deviant forms of behavior provides an overarching framework that may help to understand the heterogeneity of symptom presentation over the life span. By looking at specific components of disruptive behavior, we are able to increase the specificity with which we describe deviation in these processes.

Advantages to Understanding Etiology and Context Using Multidimensional Assessment

Multidimensional approaches also provide opportunities to consider how contextual factors such as gender, age, or culture might inform different aspects of disruptive behaviors (Krueger & Bezdjian, 2009). It is highly likely that contextual factors will influence different components of disruptive behaviors to a different degree, possibly dependent on the age and developmental level of the individual. Twin studies may be particularly informative in understanding the role of genetic and environmental mechanisms at different points in development. For example, there is evidence that the influence of context varies between subtypes of CD: aggressive behaviors are more influenced by genetic factors, and nonaggressive rule-breaking is more associated with environmental factors (Tackett et al., 2005). Similarly, parenting is not a predictor of callous/unemotional patterns but is strongly linked to other forms of disruptive behavior (Dadds & Salmon, 2003). Weems and Stickle describe the development of disordered behavior as “an interlocking network of

constructs and processes, as opposed to a single disease process or risk" (Weems & Stickle, 2005). These interlocking processes might include individual risks within the child (e.g., child sex, temperament), as well as contextual factors such as family risk (e.g., parental psychopathology, exposure to intimate partner violence) or sociodemographic risk (e.g., exposure to poverty or parental incarceration), all of which interact over time in complicated transactional processes to produce and maintain maladaptive behavior patterns. Multidimensional approaches that incorporate both severity and specific components of disruptive behavior (measured dimensionally) may shed light on clinically significant disruptive behavior by providing further specificity with which to examine their unfolding as well as opportunities to consider recently developed statistical modeling methods (Tremblay, 2010).

That CD and ODD are currently the only diagnoses in the DSM nosology that reflect disruptive behaviors means that many different behaviors and combinations of behaviors are subsumed under these two categories. For example, the categorical diagnosis of CD requires that an individual manifest only 3 of 15 symptoms (with no criteria regarding the types of symptoms required within the broad range of behaviors covered; this is in contrast to other developmental syndromes such as autism). As a result, children with very different symptom profiles, and children whose problems may have differential etiologies (e.g., aggressive versus rule-breaking), receive the same CD diagnosis. Although subsumed within a shared diagnostic classification, these subtypes reflect unique etiologies and courses, which may have critical implications for prevention and treatment (Krueger & Bezdjian, 2009; Tremblay, 2010). Moving beyond a priori subtypes to describe behavior in relation to patterning of multidimensional components or profiles may enhance understanding of etiological and developmental pathways. It is likely that etiological and contextual factors will vary across these dimensional components, just as they do across subtypes such as socialized versus unsocialized delinquent behavior or early versus late onset CD.

Quantitative and Empirical Advantages to Multidimensional Approaches

Multidimensional measurement of disruptive behaviors also offers quantitative advantages. First, even within dimensional components, there is the advantage of assessing along a continuum. Children's behavior is often assessed from a variety of informants, including teachers, parents, and children themselves, whose ratings often show only modest agreement (De Los Reyes, Henry, Tolan, & Wakschlag, 2009). These sources of variance add additional "noise" to the clinical formulation of children—variance that might better be accounted for in dimensional approach rather than a categorical diagnosis of "sick" versus "well" (Hudziak et al., 2007). Looking dimensionally within narrowband components—or looking multidimensionally—offers additional quantitative advantages beyond continuous measurement (Achenbach, 1981). In addition to characterizing core components of disruptive

behavior and identifying individual child profiles of behaviors across multiple dimensions, it is possible to subtype children empirically based on their varying profiles across the multiple dimensions, either at one point in time or through development, by subtyping based on individual profiles of functioning across multiple dimension trajectories (e.g., aggression, noncompliance).

A multidimensional approach is also likely to be critical to efforts to understand neural circuitry and/or genetic risk factors that contribute to particular forms of psychopathology. It is likely that identification of relevant neural circuitry and genes will depend on careful developmental specification of components of clinical behavior as well as concurrent examination of environmental risk factors associated with these components. This strategy has been effective in other genetics research on psychological phenomena, such as reading disability (Petryshen & Pauls, 2009). Dimensional assessments often provide greater statistical power than categorical characterization for elucidating such associations (Hudziak et al., 2007). Thus, rather than seeking a one to one correspondence between disorder status and a particular brain structure or activation pattern or between disorder and one or more genes, identification of brain—and gene—behavior associations will likely be expedited through assessment of developmental phenotypes, which comprise trajectories of specific dimensions in combination with attention to critical contextual factors (i.e., gene by environment interactions).

Emphasis in clinical nosological systems is increasingly on classification of psychopathology based on etiology and pathophysiology (Charney et al., 2002). From the perspective that psychiatric disorders are in fact reflective of perturbations in brain function, developmental neuroscience may offer an alternative perspective to identifying meaningful subgroups of children who evidence clinically significant disruptive behavior. A diagnostic system that is reflective of brain structure and function may seem far afield, but neuroscientific epistemologies can and should inform diagnostic understandings. Elegant work grounding diagnostic classification in neuroscience knowledge has been done in the realm of childhood anxiety, in which neuroscientific understandings of processes like attention, learning, and memory have been used to extrapolate to mechanistic distinctions between diagnostic classifications such as MDD and anxiety (Pine, 2007).

Knowledge from neuroscience seems particularly relevant in seeking out relevant mechanisms along the developmental pathway of disruptive behaviors. As an example, callous/unemotional traits are linked to specific neurodevelopmental differences in the amygdala; children (ages 10–17) with these traits demonstrated reduced amygdala activation while processing fearful expressions in stimuli compared to children with ADHD and control children with no diagnoses (Marsh & Blair, 2008). Further work by Blair has revealed that deficits in processing facial affect, particularly recognition of fear cues, have been demonstrated in adults and youth with psychopathic or callous tendencies across a wide range of samples and methods. Such deficits are theorized to interfere with the internalization of basic rules like inhibiting misbehavior (Kochanska & Aksan, 1995). Thus, youth with deficits in processing facial fear cues may have downstream difficulties with negative arousal and empathy that result in a lack of inhibition and aggression (Blair, 2006; Blair, Peschardt, Budhani, Mitchell, & Pine, 2006). This example demonstrates how

multidimensional approaches may be particularly crucial to fostering discovery of neuroscientific mechanisms of disruptive behaviors—and how a multidimensional perspective enables further specification.

Clinical Advantages to Multidimensional Approaches

Empirically derived multidimensional subtyping offers significant promise for improving treatment effectiveness. Effectiveness of the most widely used empirically based disruptive behavior interventions is modest, and better differentiation and earlier identification may enhance targeting of treatments (Brestan & Eyberg, 1998; Dishion & Patterson, 1992). Given that most intervention studies target children as globally disruptive, little is known about differential treatment response based on differing patterns of disruptive behavior. Evidence from subtype research suggests that labeling components of disruptive behavior and tailoring treatments to match subgroups of children who vary along these components may lead to more effective interventions. For example, boys categorized as callous/unemotional were found to be less responsive to a parent-training intervention than boys without this trait (Hawes & Dadds, 2007). The increased clinical specificity offered by a multidimensional approach that parses the heterogeneity of disruptive behaviors would allow for a more careful tailoring of treatment. Increasing usage of psychopharmacology among preschool children with disruptive behaviors (Gleason et al., 2007) also highlights the need for a stronger empirical basis for clinical discrimination.

A Developmental, Multidimensional Approach to Disruptive Behavior: A Two-Axis Model

In this proposed model of multidimensional assessment, assessment might be thought of as taking into consideration two axes, both dimensional. Axis I is a *severity axis* that cuts across specific dimensions or types of disruptive behavior and focuses on the extent to the set of behaviors the individual presents deviates from normative development with respect to frequency, intensity, persistence within or across contexts; the breadth of behavioral repertoire; and the quality of specific behavioral manifestations. Axis II comprises relevant *components* of disruptive behavior, focusing on the distinct attributes that constitute the disruptive behaviors (e.g., temper loss, noncompliance). Using a multidimensional approach, the core components that comprise the disruptive behavior syndrome can be assessed concurrently to form a profile of an individual's disruptive behavior functioning. This second axis is designed to reflect the full scope of disruptive behaviors, and is conceptually akin to the polythetic nature of DSM/ICD diagnoses. That is, diagnoses are defined by multiple problem areas and this variation is clinically meaningful (Krueger & Bezdjian, 2009). Measuring multiple components of disruptive behavior dimensionally is an attempt to better characterize this variation systematically.

While frequency, intensity, and duration are common ways of characterizing behavior, we have also highlighted the importance of quality of behavior as a critical aspect of clinically significant behavior, particularly in early childhood. Drawing on developmental science, we have operationalized *quality* in terms of the extent to which behavior is modulated, and expectable in context (Cole, Michel, & Teti, 1994; Wakschlag, Briggs-Gowan, et al., 2007). *Modulation* has three components: (1) *intensity*, or a behavior's strength and force; for example, among preschool children, mild aggression is normative, but *intense* aggression is associated with more persistent aggression over time (Brownlee & Bakeman, 1981; Cummings, Iannotti, & Zahn-Waxler, 1989; Hay et al., 2000); (2) *flexibility*, or how stubbornly entrenched a behavior is, as opposed to responding to environmental cues; this has also been shown to be a clinical indicator in disruptive disorders (Angold & Costello, 2000); and (3) *organization*, or the pacing, duration, and predictability of sets of behaviors; for example, tantrums of a few minutes that are not highly dysregulated are normative for preschoolers (Potegal, Kosorok, & Davidson, 2003), but destructive tantrums are more common among children with a range of clinical disorders (Egger, 2003). *Expectable in context*, also an element of quality, refers to the extent to which a behavior is normatively elicited within a particular context. For example, mild aggression may be typical for children in the context of peer disputes or rough and tumble play (Hay, 2005), but aggression directed towards adults is not expectable in context and thus viewed as qualitatively distinct. Research on quality of disruptive behaviors has largely proceeded by examining a specific component of disruptive behavior in isolation (e.g., looking at aggression or noncompliance in isolation), rather than identifying the quality of multiple components of behavior within the same child (Wakschlag & Danis, 2009).

Quality is critical to understanding the severity axis of disruptive behavior. For example, in the domain of temper loss, a tantrum that is highly dysregulated but short in duration is qualitatively more severe than a more regulated and short tantrum, but less severe than a highly dysregulated tantrum that lasts for 20 min. Moreover, quality also informs the range of behavioral elements that are included within the second domain axis in which components are specified. Low base-rate behaviors are often not included in dimensional scales designed to assess the continuum of behavior. However, building a comprehensive model of disruptive behavior that captures the full scope of disruptive behavior will mean including low base-rate, qualitatively distinct behaviors, that when present may be highly informative in terms of both the severity axis and the dimension that they represent.

The Four-Factor Multidimensional Model of Disruptive Behavior Across the Life Span

Some of us have previously (Wakschlag et al., 2010; Wakschlag et al., 2012; Wakschlag et al., 2011) proposed a four-factor dimensional approach to disruptive behavior that is theoretically, developmentally, and empirically grounded.

These four core dimensions of disruptive behavior are: (1) aggression, (2) noncompliance, (3) temper loss, and (4) low concern for others. These four dimensions are theoretically based on: (a) a developmental psychopathology approach, emphasizing individual differences and developmentally based conceptualizations along four core normative developmental processes that are relational in nature: (1) the modulation of aggression, (2) the balance of autonomy and compliance, (3) emotion regulation (particularly anger regulation), and (4) empathy and conscience development; (b) a clinical understanding of the heterogeneous ways early emerging disruptive behavior presents itself; and (c) prior conceptual and empirical work that has looked at characterizing disruptive behavior. This comprehensive four-dimensional model seeks to move beyond aggression as a central organizing frame and to integrate bodies of work that have sought to describe specific components of disruptive behavior (e.g., callous/unemotional) into a unified model that captures the full disruptive behavior spectrum.

The *aggression* dimension characterizes a tendency to respond aggressively across a variety of contexts, ranging from expectable self-protection to severe violence. The *noncompliance* dimension captures failure to comply with directions, rules, and social norms, ranging from developmentally expectable resistance to pervasive and provocative rule-breaking. The *temper loss* dimension encompasses overt expression and management of anger, ranging from mild expressions of frustration to rage and extreme and dysregulated temper loss. The *low concern* dimension captures active disregard of others, including lack of guilt for transgressions and lack of concern for others' feelings. Behaviors along this dimension may include mild insensitivity within expectable contexts to extreme and persistent disregard of others' needs and feelings.

In three independent samples (two early childhood and one adolescent), this four-dimension model has demonstrated a superior fit compared to traditional models including: (a) a DSM-based (ODD/CD) model and (b) a two-dimensional model distinguishing a general disruptive group from a group high on the low concern dimension, along the lines of the callous/unemotional subtype described and extensively studied by Frick and colleagues. The superior model fit was demonstrated across child age and sex. Concurrent and predictive validity were also demonstrated (Wakschlag et al., 2011).

Aggression

Normative aggression appears in infancy as a natural way of expressing anger; attaining "aggressive competence" is viewed as a normative developmental event (Hay, 2005, p. 125) as young children learn to respond to frustration (e.g., loss of a toy to another child) with instrumental aggression that achieves a functional goal (e.g., retrieval of the toy) (Tremblay et al., 2004). While some aggression is normative in early childhood, landmark longitudinal studies of patterns of aggression across early childhood have demonstrated that normative levels of aggression are

low-moderate in early childhood and begin a marked decline in frequency between 36 and 42 months of age (Shaw, Lacourse, & Nagin, 2004; Tremblay et al., 2004). In the current DSM-IV, aggressive behaviors are captured in multiple CD symptoms (e.g., "often initiates physical fights"). These symptoms are intended to be evaluated with respect to normative development, although no specific developmental criteria are offered. Aggression is the most studied of the disruptive behavior dimensions and has often been considered the hallmark of DBDs.

Population-based research on aggression in young children has demonstrated that the quality of aggression may be an important clinical indicator. For example, 19 % of 2-year-olds and 15 % of 3-year-olds are often "aggressive when frustrated," but only 1 % of children at either age "hurt others on purpose" (Carter et al., 2003). Moreover, observed reactive aggression with peers is not associated with high maternal ratings of aggression, but proactive aggression is (Hay et al., 2000). *Normative* manifestations of aggression in toddlers include mild aggression when frustrated and rough and tumble play (Hay, 2005). Clinical manifestations may include intense, driven aggression; dysregulated, destructive aggression; and aggression directed towards adults (Hay, 2005; Zahn-Waxler & Radke-Yarrow, 1990).

A great deal of work in social cognition documents that aggression is associated with hostile attribution bias, i.e., the tendency to attribute hostile intent to others in neutral or ambiguous situations. As deficits in social cue detection fail to provide information that would promote adaptive social problem-solving and diffuse angry/retaliatory responses, hostile attributions may increase rates of aggression (Dodge, 2006). From preschool through adolescence, hostile attribution bias has been associated with disruptive behavior in general and with increased aggression specifically (Coy, Speltz, DeKlyen, & Jones, 2001; Runions & Keating, 2007). Hostile attribution bias also appears to be present in youth prenatally exposed to cigarettes who are at heightened risk for DBDs (Wakschlag et al., 2009). Supporting a causal mediating role in the maintenance of disruptive behavior, interventions designed to reduce hostile attribution bias have resulted in corollary reductions in youth aggression (Hudley & Graham, 2008).

Noncompliance

Like aggression, noncompliance has developmental roots in a normative process, here negotiating rules and directives and a movement towards autonomy. Indeed, learning to say "no" is a normative developmental milestone on this path (Crockenberg & Litman, 1990). Normative assertions of autonomy exist on a dimensional continuum of severity with their clinical counterparts of pervasive and persistent disregard of rules and norms. Using detailed observations, researchers were able to distinguish normative noncompliance (e.g., a child negotiating to get his/her own way) from overt defiance that involves active and definitive refusal, with the latter associated with elevated risk of disruptive behavior (Kuczynski & Kochanska, 1990).

Noncompliance has been examined developmentally as disregard for rules (Petitclerc, Boivin, Dionne, Zoccolillo, & Tremblay, 2009) and as defiance (Baillargeon et al., 2011) in toddlers; as "resistance to control" in young children (Bates, Pettit, Dodge, & Ridge, 1998); and as serious norm violation in delinquent youth (Loeber & Farrington, 2000). In DSM-IV, noncompliance is diagnostically captured in ODD symptoms of defiance and argumentativeness as well as in CD symptoms that reflect rule violation. Normative manifestations in young children include autonomy assertions, negotiated noncompliance, and noncompliance in response to fatigue or limit (Drabick, Strassberg, & Kees, 2001). Possible clinical indicators in young children include intense and insistent noncompliance, a "reflexive no," sneaky misbehavior, and noncompliance that predominates even in positive social contexts (Kuczynski & Kochanska, 1990). There is epidemiological evidence that preschoolers who are very difficult to manage are more likely to present DBDs (Moffitt, Caspi, Rutter, & Silva, 2001).

Authors have also stressed the possible adaptive nature of toddlers' noncompliant behavior for learning the ranges of possible behaviors that are legitimate, or open to him or her (Breger, 1974; Dubin & Dubin, 1963). Noncompliance can be used adaptively to negotiate the boundaries between what is within the toddler's area of personal preferences and choices, and what falls within the purview of socially prescribed norms of interpersonal conduct, moral obligations, and health/safety prescriptions (Nucci, Killen, & Smetana, 1996). It can also be used as a step in the process of internalizing rules of conduct (Hoffman, 1983). In addition, Stifter and Wiggins (2004) refer to "assertive noncompliance" and Wenar (1982) to "healthy/realistic negativism."

Neurocognitively, noncompliance may be related to response perseveration deficits, which reflect a failure to inhibit behavior in response to "punishment" cues because of heightened sensitivity to immediate reward. This inflexible response pattern under conditions of high motivation has been theorized as a neurocognitive substrate of disruptive behavior (Nigg & Casey, 2005; Van Goozen, Cohen-Kettenis, Swaab-Barneveld, & Van Engeland, 2004) and has corollary behavioral manifestations in the intransigent patterns of noncompliance exhibited by children with ODD symptoms. Response perseveration has been associated with youth disruptive behavior in community samples (Goodnight, Bates, Newman, Dodge, & Pettit, 2006) and ODD in clinic samples (Matthys, Van Goozen, Snoek, & Van Engeland, 2004; Van Goozen et al., 2004).

Temper Loss

Temper loss has normative roots in the developing skill of emotion-related behavior regulation (Eisenberg & Fabes, 1992), specifically overt expressions and management of anger (Cole, Martin, & Dennis, 2004). Dimensionally, it might be seen along a spectrum from normative mild-moderate expressions of anger in response to frustration to extreme, dysregulated temper. The developmental emergence of

anger has been studied during infancy in the context of emotion differentiation, emerging even before 4 months of age (Sternberg & Campos, 1990). Anger has also been studied in the context of examining individual differences in temperamental predispositions to reactivity and regulation of negative emotion (Rothbart, Posner, & Hershey, 1995). Anger is also one of the primary components of tantrums (Potegal et al., 2003). Episodes of moderate anger are normative (Calkins & Johnson, 1998), but anger dyscontrol heightens risk for DBDs and serious antisocial behavior across the life span (Bates, Bayles, Bennett, Ridge, & Brown, 1991; Cole, Teti, & Zahn-Waxler, 2003; Eisenberg, 2000; Gilliom, Shaw, Beck, Schonberg, & Lukon, 2002). Within the DSM-IV nosology, temper loss is reflected in multiple ODD symptoms (e.g., loses temper, angry/resentful). It is not specific to ODD, and may be a marker of multiple DSM-IV disorders (e.g., irritability in depression) (Leibenluft, Cohen, Gorrindo, Brook, & Pine, 2006; Stringaris et al., 2010).

Normative manifestations in young children include intermittent tantrums and temper loss in response to frustration (Potegal et al., 2003). Parent ratings of the frequency of distinct anger-related behaviors indicate marked variability in early development. For example, for children at 17 months of age, only 22.1 % of boys and 18.7 % of girls are described by parents as "having a hot temper or temper tantrums" (Baillargeon et al., 2011). Clinical indicators of temper loss for preschool disruptive behavior include destructive and prolonged tantrums, multiple daily tantrums and easily precipitated temper loss (Egger, 2003; Needleman et al., 1991; Wakschlag et al., 2011). Whereas episodes of moderate anger are normative (Calkins & Johnson, 1998), anger dyscontrol heightens risk for DBDs and serious antisocial behavior across the life span (Bates et al., 1991; Cole et al., 2003; Eisenberg, 2000; Gilliom et al., 2002). There is limited evidence from epidemiological studies that temper loss predicts to antisocial acts further down the developmental trajectory, for example, that frequent and/or severe temper tantrums at age 3 years predict violent crimes at 23–24 years of age (Stevenson & Goodman, 2001). Notably, though, destructive tantrums are not specific to DBDs. Rather, they are a clinical indicator for several disorders in the preschool period (including separation and other anxiety disorders) (Egger, 2003). Therefore, dimensional assessment of destructive tantrums, or anger, is likely to contribute to the severity axis but will need to be examined as part of a multidimensional profile that includes additional disruptive behavior related behaviors to obtain prediction of disruptive behaviors with high specificity and sensitivity.

Neurocognitively, temper loss has correlates in deficits in effortful or "inhibitory" control (i.e., the ability to inhibit a prepotent or dominant response in accordance with rules or instructions) (Aksan & Kochanska, 2004; Carlson & Wang, 2007), which have been associated with young children's difficulties regulating negative emotions and to predict disruptive behavior (Brophy, Taylor, & Hughes, 2002; Eisenberg, Fabes, Nyman, Bernzweig, & Pinuelas, 1994; Kochanska & Knaack, 2003; Rueda, Posner, & Rothbart, 2005; Spinrad et al., 2007). Effortful attentional shifting and response inhibition importantly underlie distress regulation (Rueda et al., 2005); thus, children with impaired effortful control are more likely to exhibit the core temper loss features of ODD, particularly difficulty modifying or

inhibiting the expression, intensity, and temporal features of negative emotion in response to environmental demands (Carlson & Wang, 2007; Cole et al., 2003; Spinrad et al., 2007).

Low Concern

Dimensionally, low concern for others reflects variations in responsiveness to the feelings of others, including modifying behavior based on negative response from others, extent of remorse after angering or displeasing others, and sensitivity to others' feelings. It ranges normatively from mild insensitivity within contexts of stress or conflict to extreme and persistent callous disregard of others across a range of social interactions and contexts (Wakschlag et al., 2010). In developmental studies, this dimension has been studied in multiple streams of research including the development of prosocial behavior such as empathy and attentiveness to others' feelings (Hay & Cook, 2007) and multiple facets of conscience, including early moral emotions (i.e., discomfort following wrongdoing/guilt) that influence responsiveness to punishment (Kochanska & Aksan, 2006). Although these various facets have been studied as separate, interrelated behaviors developmentally, here we propose that from a clinical perspective they are considered as elements of a single low concern for others' dimension that coalesces in a coherent set of behaviors reflecting active disregard of others' feelings, in keeping with the extensive work on callousness in older youth (Frick et al., 2003).

Concern for others develops in the first years of life, including the emergence of empathic responses to others' distress and spontaneous prosocial behaviors (Carter et al., 2003; Chase-Lansdale, Wakschlag, & Brooks-Gunn, 1995; Eisenberg & Fabes, 1998; Zahn-Waxler, Radke-Yarrow, Wagner, & Chapman, 1992). For instance, in the study by Baillargeon, Normand, et al. (2007), 62.4 % of children—the same percentage for boys and girls—were estimated, at 17 months of age, to have comforted a child who is crying, at least on an occasional basis. Extensive work by Kochanska and colleagues on the development of conscience has demonstrated its emergence even in very young toddlers (Kochanska & Aksan, 2006). For example, even very young children have internalized basic rules, such as inhibiting misbehavior and refraining from prohibited activities even when an adult is not present (Kochanska & Aksan, 1995). Further, young children also exhibit remorse including guilt about misbehavior, apologizing, gaze aversion, and attempts to restore good feelings (Kochanska, 1994). Lack of concern has been widely studied in older youth by Frick and others within the framework of "callous/unemotional traits" (Frick et al., 2003; Kotler & McMahon, 2005) but has not been a focus of attention in studies of preschool disruptive behavior. Consistent with this argument, Frick et al. have reported links between callous/unemotional features and proactive aggression in a small sample of preschoolers (Kimonis et al., 2006). Laboratory observations of preschool children's lack of concern for others' simulated distress has also been shown to moderate the stability and severity of preschool disruptive behavior in

developmental studies (Hastings, Zahn-Waxler, Robinson, Usher, & Bridges, 2000). In DSM-IV, low concern is reflected in ODD (e.g., spitefulness, blaming) and CD (e.g., bullying, cruelty) symptoms. Normative manifestations in young children may include mild insensitivity to peer distress, occasional blaming of others to avoid negative consequences, and refusing to share and mild taunting or teasing (Wakschlag et al., 2012). We hypothesize that clinical manifestations may include indifference to punishment or consequences, being unfazed by parental anger, disinterest in pleasing others, and taking pleasure in others' distress.

Neurocognitively, low concern may be related to processing of fear cues. Deficits in processing facial affect, particularly recognition of fear cues, have been demonstrated in adults and youth with psychopathic or callous tendencies across a wide range of samples and methods (Marsh & Blair, 2008). Such deficits are theorized to interfere with internalization, because others' fear and distress are negatively arousing, elicit empathy, and lead to inhibition of aggression (Blair, 2005; Kochanska, Gross, Lin, & Nichols, 2002).

Critical Next Steps for Advancement

Working from a bottom-up, developmental psychopathology framework to build multidimensional understandings of disruptive behaviors will require the use of a variety of research designs and methods. To disentangle normative misbehavior from clinically significant manifestations of disruptive behavior will require epidemiological, population-based, longitudinal studies that begin in early childhood. While much advancement has been made in this field (Baillargeon, Normand, et al., 2007; Briggs-Gowan et al., 2001; Tremblay et al., 2004), developmental specification of dimensional manifestations of disruptive behaviors will require greater knowledge about normative manifestations of a broad range of these behaviors in longitudinal, multi-method, population-based studies. In many ways, this descriptive work has only begun and multi-method studies that include observational methods across multiple contexts are needed.

Relatedly, as we have argued, looking at behaviors in a dynamic and organized manner to consider *quality* is crucial to describing the full spectrum of disruptive behaviors. Studying clinical or clinically enriched populations may be extremely helpful in characterizing the severe end of the spectrum of disruptive behaviors. Further research on the quality of disruptive behavior, done from a developmental perspective, will help to distinguish what is typical from what is atypical across the life span.

In addition to work that seeks to locate the early childhood roots of these dimensions, a life span conceptualization demands looking beyond early childhood to understand the unfolding of these potentially linked behavior patterns across time and context (i.e., examining heterotypic continuity). Further work on the trajectories of early disruptive behaviors—and on the children early identification may currently be *missing*—will help to enhance the sensitivity and specificity of our measurement. Although we are advocating a multidimensional approach, we

concurrently believe that categorical diagnoses will continue to serve useful functions, especially in relation to clinical practice and public health initiatives. Moreover, once disruptive behaviors are characterized by multiple dimensions, we will need to document the relation of these dimensions to diagnosis as well as how the specificity and sensitivity of disruptive behaviors change with age within the general population. For instance, due to the rapid decline in frequency, biting peers may be a perfectly sensitive behavior for assessing physical aggression in children under 2 years of age, but may not be a sensitive marker among 4- to 5-year-olds.

Moreover, if we are truly attempting to capture the spectrum of behavioral manifestations of disruptive behavior, more sensitive work that evaluates the influence of context is required. As Dodge has argued, "any assessment of behavior always represents the individual in context" (Dodge, 1993). Indeed, disruptive behaviors are conceptualized as existing *only* within a relational framework—one cannot be "defiant" without an *other* to defy. Thus far, our only real diagnostic conceptualization of context is that we require the presence of a behavior or behaviors within a dimension to occur across multiple contexts to determine that the behavior is pervasive, an indicator of severity. Although we do not routinely assess the degree to which contexts such as school and home are varied with respect to the demands placed on the individual, we presume that the occurrence of disruptive behaviors across contexts reflects both pervasiveness and inflexibility of response. For example, if a child is defiant across multiple contexts—at school, at home, with peers—his behavioral response pattern is more rigidly maladaptive and therefore perhaps more "severe" (De Los Reyes et al., 2009).

The question of contextual manifestations of disruptive behavior also has implications for diagnosis and assessment, which highlights the critical importance of assessment tools. According to the current diagnostic formulation of ODD, defiant behaviors need only occur in one context to meet criteria for diagnostic categorization. If one is inflexibly defiant with a parent, for example, one is eligible for the same diagnosis as if one is inflexibly defiant with a parent, at school, and in unfamiliar situations. However, these clinical profiles could require distinct assessment as well as intervention. Novel approaches to diagnostic assessment of young children that take the varied demands of interactional context as well as the rigidity and pervasiveness of behavior into account are needed. For example, new research from the Disruptive Behavior—Diagnostic Observation Schedule (DB-DOS) (Wakschlag et al., 2008), an observational assessment of disruptive behavior that includes both examiner and parent contexts, reveals that while scenarios with an unfamiliar adult are the most diagnostically informative for boys, it is with *parents* that girls with DBDs are demonstrating diagnostically informative disruptive behavior (Sarah et al., 2012); thus, the same lab assessment, without both parent and examiner contexts, would not capture the underlying disruptive behavior of boys and girls. This surprising finding reminds us that our knowledge of the varied landscape of disruptive behavior is only as specific as the tools with which we measure it.

The above-cited finding about sex differences in contextual manifestations of disruptive behavior fits into a large body of theorizing in which questions are raised regarding whether the current diagnostic conceptualization of disruptive behaviors, which have largely grown out of research on boys, is appropriate for capturing the

varied ways that young *girls* may be demonstrating impairing and maladaptive disruptive behaviors (Zoccolillo, Tremblay, & Vitaro, 1996). Current knowledge of disruptive behavior dimensions draws largely on studies of male youth; however, burgeoning evidence suggests sex difference in expressions and patterns of disruptive behavior even in early childhood (Baillargeon, Zoccolillo, et al., 2007; Crick, Ostrov, & Werner, 2006; Hipwell et al., 2007; Moffitt et al., 2001). Consistent with early studies of young children that attempted downward extensions of adult and older child assessment tools, studies that *have* included girls have often sought to confirm the fit of male models for girls rather than working from an a priori frame that assumes that female manifestations may look different (Ostrov, 2008). Thus, building up a body of knowledge that creates space for female-typical manifestations of disruptive behavior—and how the specificity and/or sensitivity of the relation of dimensions of disruptive behaviors to disruptive disorders vary between boys and girls at a given age—will be a crucial part of characterizing the full spectrum of disruptive behavior dimensions.

In addition to a focus on boys, literature on disruptive behavior has focused perhaps disproportionately on aggression. The large role that aggression has played in clinical research on disruptive behaviors means that our knowledge base is more expansive in that domain. Moving forward, it will be important to increase our understanding of each of the salient component dimensions that constitute the full range of disruptive behaviors (e.g., temper loss, noncompliance) in order to build a consistent knowledge base.

Finally, disruptive behavior cannot be understood without looking at homo- and heterotypic comorbidity, or co-occurring problems within and across diagnoses. Comorbidity has been postulated to relate to the severity of disruptive behaviors. It may also be reflective of unique etiological processes; for example, it has been found consistently that the presence of comorbid ADHD and CD is associated with earlier onset of disruptive behavior than a diagnosis of CD alone (Loeber et al., 2000). Moreover, it has been hypothesized that the specific dimensions of ADHD (e.g., impulsivity, hyperactivity, inattention) may uniquely relate to dimensions of disruptive behavior. For example, among 13-year-olds, aggressiveness when combined with motor restlessness predicted more strongly to adult criminal behavior than either alone (Magnusson, 1998). As this finding suggests, comorbidity may also relate to heterotypic continuity. Looking multidimensionally, the developmental relationship between the severity and domains of comorbid psychopathology (e.g., inattention, hyperactivity, depression) and the severity and domains of disruptive behavior (aggression, temper loss) is a field ripe for exploration.

Conclusion

Multidimensional approaches, which we have conceptualized here as incorporating two axes (one axis addressing severity and a second axis that comprises multiple components that reflect the most salient features of disruptive behavior), offer many

advantages to the study of disruptive behavior. Given an interest in early manifestations of disruptive behaviors, a central advantage is increased developmental specificity, particularly in terms of charting heterotypic shifts in the behaviors that comprise disruptive behaviors through time. In addition, dimensional approaches typically offer greater statistical power than categorical approaches and, due to their focus on more narrow sets of behavior, are also more likely to shed light on neural circuitries and/or genes that are linked to these behaviors

Building on prior work, this chapter highlights a life span multidimensional model with four core disruptive dimensions. This model is based on preexisting developmental science, focusing on the four normative and relational developmental processes of (1) emotion regulation, (2) empathy and conscience development, (3) the balance of autonomy and compliance, and (4) the modulation of aggression. The four proposed domains of disruptive behavior include the range of normative presentations and the ways in which these processes go awry—in temper loss, low concern for others, noncompliance, and aggression.

Critical to the advancement of dimensional approaches to disruptive behavior will be continuing to chart the normative developmental course of these domains as well as deepening understanding of how normative development shifts towards and away from psychopathology. Attention to age and gender differences in their typical and atypical expression is also crucial. Often overlooked in current research is attention to how the quality intersects with frequency, duration, and intensity of disruptive behaviors, which is likely critical for understanding the full manifestation of disruptive behaviors over development and capturing heterotypic continuity. Multimethod, longitudinal studies that begin with representative sampling of both boys and girls and that assess core dimensions through parent and teacher reports and observation are needed. However, such studies will be limited unless researchers begin to also link individual variation in profiles determined based on trajectories of multiple disruptive behavior dimensions to neurocognitive, genetic, and broader familial and community contextual risk factors. As our current intervention strategies leave considerable room for improvement, we can hope that elucidating mechanisms of change over time will yield important clues for enhancing preventive and targeted interventions.

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Chapter 6
Gender and the Development of Aggression, Disruptive Behavior, and Delinquency from Childhood to Early Adulthood

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It is well established that only a very small proportion of children become persistently serious delinquents or adult psychopaths. Also, many boys and girls showing some aggression and disruptive behavior in earlier childhood will not progress to more serious conduct problems by adolescence. Thus, the study of conduct problems and serious outcomes requires knowledge of the age-normative problem behaviors and their course over time for boys and girls, and why some children and youth deviate from these normative patterns. The pattern, developmental course, and their causes are somewhat different for girls compared to boys, which is the main topic of this chapter. For example, most of the violence committed by adolescent girls, in contrast to boys, is directed at relatives, especially their mother or a dating partner. Assault rates by girls have increased over the years, but it is debatable to what extent these increases are a result of improved police work, and prosecution, and the reporting of simple assaults by the police (Zahn, 2007).

There are several reasons why we understand relatively little about the development of disruptive and delinquent behaviors (such as symptoms of oppositionality, aggression, and theft) in girls versus boys and, therefore, have limited ability to understand the role of gender. The small number of longitudinal studies, particularly ones starting in infancy with data for both boys and girls, limits our

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understanding of the developmental course of problem behaviors by gender. This is largely due to the fact that higher levels of delinquency and crime in males led to a focus on boys (e.g., see Blumstein, Cohen, Roth, & Visher, 1986). In recent years girls' delinquent behavior has received more attention (e.g., Chesney-Lind, 1997; Jackson, 2004; Maccoby, 2004; Moffitt, Caspi, Rutter, & Silva, 2001; Moretti, Odgers, & Jackson, 2004; Pepler, Madsen, Webster, & Levene, 2005; Putallez & Bierman, 2004; Zahn, 2009). Much new knowledge comes from major longitudinal studies with sizeable samples of girls (e.g., Costello et al., 1996; Hipwell et al., 2002; McConaughy, Stanger, & Achenbach, 1992).

However, there are substantial issues in the empirical literature that constitute challenges for the explanation of gender differences in aggression, disruptive behavior, and delinquency, particularly explanations that examine the contributions of both socialization and genetic or biological factors. The problems are compounded by arbitrary divisions between different scholarly disciplines, such as psychiatry, developmental psychology, and criminology (Zahn-Waxler & Polanichka, 2004), and by the concentration on boys in the literature so far. This paper attempts to cross different disciplines and present a view from "above" to reconcile and integrate different approaches in a critical fashion. We begin by a discussion of the approaches of the different disciplines to understanding disruptive and delinquent behaviors.

The psychiatric approach is focused on the classification of individuals according to diagnostic categories for clinical purposes. Oppositional defiant disorder (ODD), conduct disorder (CD), and antisocial personality disorder (APD) are the most relevant for this chapter. DSM-IV specifies ODD as involving "a recurrent pattern of negativistic, defiant, disobedient, and hostile behavior toward authority figures," whereas the key features of CD are "a repetitive and persistent pattern of behavior in which the basic rights of others or major age-appropriate societal norms or rules are violated" (American Psychiatric Association, 1994, p. 91). APD, an adult diagnosis, is defined as a pervasive pattern of disregard for and violation of the rights of others occurring since age 15 years (p. 649).

In contrast, the discipline of developmental psychology has focused more on dimensional conceptualization and measurement of disruptive behavior, using distinctions such as overt and indirect aggression (e.g., making prank phone calls, writing critical notes or e-mails about a person behind their back), callous-unemotional behavior (an early form of psychopathy), and delinquency, implying that these related behaviors are the extreme end of an underlying continuous distribution of liability (Watson, 2005; Widiger & Samuel, 2005). Developmental psychologists have focused more on developmental patterns of, and relations among, different disruptive and delinquent behaviors to form predictive and explanatory models.

Criminological approaches to deviant behavior share with the developmental psychological approach to deviant behavior an emphasis on behavioral manifestations (e.g., delinquency, frequency of violent acts) but use categories of behaviors based on law breaking (e.g., Wolfgang, Figlio, & Sellin, 1972). Sometimes criminological approaches focus on categories of individuals, such as career criminals, which are different from psychiatric nosology; but these approaches often are about the same individuals, albeit typically each at different developmental periods and

with different emphases in deviant behavior. The three approaches, however, share recognition of the possible severity of the problem behaviors in terms of long-term sequelae, including the repeat victimization of others, impairment of functioning of the perpetrator in several areas other than the deviant behavior, and the possible persistence of the problem behaviors over long periods of time in a subpopulation of youth (e.g., Loeber, Farrington, Stouthamer-Loeber, & White, 2008). Each has produced valuable information about gender and disruptive behavior.

There is less agreement among the disciplines about causation, including the causes of gender differences in aggression, disruptive behavior, and delinquency. The psychiatric approach is based on a disease model and has put more emphasis on biological processes than individual features (such as self-control) and social factors. Developmental approaches have focused on individual and social factors but in recent decades also have embraced a variety of biological factors and their interaction with social factors. Criminology, in contrast, is largely (but not exclusively) preoccupied with individual factors (such as self-control) but also macro-environmental conditions that foster delinquency (e.g., poverty, neighborhood differences, collective efficacy, and deterrence effects of incarceration). To be fair, many researchers representative of each of the disciplines have pursued a medley of different levels of causation.

This chapter addresses gender differences in the development of aggression, disruptive behavior, and delinquency, and a selection of their causes particularly focused on individual characteristics, family socialization factors, and peer factors (a full review of all putative socialization factors is outside the purview of this chapter). Findings based in the tradition of developmental psychology, which focuses on continuous or dimensional measurement of aspects of conduct problems are discussed, as well as on findings related to the dichotomous clinical diagnoses of ODD and CD. This chapter aims to address the following questions: (a) When is the onset of boys' and girls' disruptive and delinquent behavior, and when does desistance (or cessation) occur? (b) To what extent is there continuity of different kinds of disruptive and delinquent behavior symptoms for each gender? (c) What are explanations for developmental differences in disruptive behaviors in boys and girls? These questions will be addressed for different forms of aggression and violence. Brain developmental as a cause of gender differences in conduct problems, aggression, and delinquency is covered elsewhere in this volume and, for that reason, is not included here.

Onset, Prevalence, and Manifestations of Aggression in Childhood

Aggression is normative in the first few years of life, and then decreases (Loeber & Hay, 1994; Tremblay et al., 2004). In recent years, Tremblay and colleagues (e.g., Tremblay et al., 1999) have argued persuasively that developmental models, which view physical aggression by children as solely due to social learning (e.g., Bandura, 1973), have not given adequate consideration to the fact that very young

children—almost as soon as they are capable of independent motion and prior to substantial language—engage in physically aggressive behaviors. Tremblay et al. (1999) found that by age 17 months the onset of physical aggression was reported for close to 80 % of children. It was initially thought that there are few or no gender differences in infancy and toddlerhood (Keenan & Shaw, 1997; Loeber & Hay, 1994), but new studies have changed this picture (see also review by Archer & Coté, 2005). For example, Baillargeon, Tremblay, and Willms (2005) examined gender differences at ages 2–3 years. They argued that one of the factors accounting for discrepant findings in the literature regarding the association of gender and physically aggressive behaviors in early childhood was that physical aggression was defined and operationalized differently across studies. Using the National Longitudinal Survey of Children and Youth (NLSCY) in Canada, involving parental reports, Baillargeon et al. (2005) examined the frequency of three types of aggression; namely, getting in many fights; reacting with anger and fighting to accidental bumps; and kicks, bites, and hits other children. Findings indicated that boys were more likely than girls to get into fights and to kick, bite, and hit other children frequently (see also Archer & Coté, 2005). For example, among 2 year olds, 33.1 % of girls and 37.7 % of boys occasionally kicked, bit, or hit, and 2.4 % of girls and 4.8 % of boys engaged in such aggression often. Thus, whereas the prevalence of any such behavior occasionally was relatively similar for boys and girls, boys were twice as likely to engage in such behavior frequently. Boys were also more likely to get in many fights, but not more likely to react aggressively to accidental contacts.

Tremblay, Masse, Pagani-Kurtz, and Vitaro (1996) examined the developmental trends in frequent versus occasional physical aggression (hitting, biting, and kicking) from ages 24 months to 12 years for boys and girls. The highest levels occurred in both sexes at age 2 years and declined over time. Thus, evidence was found supporting the hypothesis that the normative pattern is for children to improve in inhibitory control, and by extension social skills, with age.

There are large individual differences in aggression early in life for both girls and boys; Loeber and Hay (1994) proposed that among these differences, intensity, reactivity, and pervasiveness are critical dimensions in the continuity of the behaviors over time. Boys generally showed higher levels of physical aggression than girls, but only a very small proportion of boys or girls continued to show frequent aggression after age 5 years. For those showing aggression during the preschool years, girls seemed to improve more rapidly than boys after age 4 years (Maccoby, 2004). In a study of expulsions in kindergarten, Gilliam (2005) shows that the average number of expulsions per 1,000 preschoolers was 4 times as high for boys as for girls (10.5 vs. 2.3), showing the relative rarity of extreme disruptive behaviors for girls in that age group.

Keenan, Wroblewski, Hipwell, Loeber, and Stouthamer-Loeber (2010), reviewing past studies, showed that the age of onset of symptoms of ODD and CD did not statistically differ between boys and girls. An early age of onset of conduct problems predicts later serious delinquency in both boys and girls (Loeber & Farrington, 2001; Zahn-Waxler & Polanichka, 2004). By age 5 years, some parent-reported gender differences appear, including more boys hitting others with dangerous objects, fewer girls bullying or threatening other people, and more boys engaging in

theft (Kim-Cohen, Moffitt, Taylor, Pawlby, & Caspi, 2005). However, observations in school playgrounds show that between Grades 1 and 6, the level of aggression of boys and girls toward their peers is very similar (Pepler & Craig, 2005).

It should be kept in mind that there are gender differences documented for types of behavior related to delinquency and disruptive behavior: girls tend to show more empathy than boys, demonstrate higher affiliative behaviors, display more collaborative play, show more tend-and-befriend behaviors, and when angry have shorter anger outbursts (Zahn-Waxler & Polanichka, 2004). Girls who are low on empathy, show low affiliative behaviors, engage in little collaborative play and little tend-and-befriend behaviors, and display long anger outbursts are more extreme outliers for their sex relative to boys (Loeber & Hay, 1994).

Prevalence of Disruptive Behavior Disorders by Age

Does the prevalence of disruptive behavior disorders change with age? Unfortunately, most studies have not looked separately at prepubertal children and adolescents, so it is difficult to establish how such prevalence rates change with age. Studies of preschool children, however, are not inconsistent with those of older children. In the two studies (Egger et al., 2006; Keenan, Shaw, Walsh, Delliquadri, & Giovannelli, 1997) using DSM-IIIR or DSM-IV criteria and standard interview methods, the rates of CD were, respectively, 3.3 % and 4.6 %, and the rates of ODD were 6.6 % and 8.0 %. Evidence for increases in delinquency and antisocial behavior in adolescence is strong (National Research Council and Institute of Medicine, 2001) but the patterns for CD and ODD are much less clear. ODD appears to have a fairly constant prevalence across childhood and adolescence, but many studies report increases in conduct disorder or CD symptoms in adolescence (reviewed in Maughan, Rowe, Messer, Goodman, & Metzler, 2004), which is consistent with the peak age for delinquency in mid to late adolescence (Blumstein et al., 1986).

Despite the variability among individual studies—a meta-analysis (National Research Council and Institute of Medicine, 2001) combining across childhood and adolescence for studies using DSM-IIIR, DSM-IV, ICD-9, or ICD-10—the estimates of prevalence have a reasonably narrow range: median 2.9 % (inter-quartile range 1.2, 4.2 %, $N = 27$ studies) for CD and median 2.5 % (inter-quartile range 1.3, 2.9 %, $N = 21$ studies) for ODD. It is estimated that in elementary school 2 % of girls and 7 % of boys meet a diagnosis for CD (Offord, Boyle, & Racine, 1991), ODD being more common at this age. The prevalence of conduct disorder for males is found consistently to be higher than that for females. Most of the difference is seen in symptoms related to causing physical harm to others. Estimates of prevalence rise through early to midadolescence to about 4–15 % of boys and girls (Offord et al., 1991). The gender difference remains but is smaller in most studies.

Despite the fact that much research, until quite recently, has focused on boys, CD is the second most common psychiatric diagnosis in girls, particularly in adolescence, indicating that it is a substantial mental health concern for girls. Conduct problems show a continuous distribution among both boys and girls at any one point in time,

although the individual symptoms change in prevalence, frequency, and severity throughout childhood and adolescence. One developmental change that has been noted in several studies is girls' increased use of verbal rather than physical aggression and covert forms of delinquency. For example, Pepler and Craig (2005) reviewed studies showing a decrease in girls' physical aggression with age but a subsequent increase in verbal and social aggression. Adult criminal records indicate that women are frequently arrested for nonaggressive, covert forms of delinquency, such as shoplifting and fraud (Ogle, Maier-Katkin, & Bernard, 1995; Rutter & Giller, 1983).

Different Forms of Psychopathological Co-determinants

The search for early psychopathological co-determinants distinguishing age-normative from serious antisocial youth has turned to different forms of psychopathology. Scholars have pointed out that aspects of psychopathology, often co-occurring with disruptive and delinquent behavior, were thought to be co-determinants for the persistence and increasing severity in some and not in other youth. Initially, the search focused on attention-deficit hyperactivity disorder (ADHD; American Psychiatric Association, 1994), with evidence that the most seriously affected youth scored high on both early antisocial acts and symptoms of ADHD (or its former diagnosis of ADD; e.g., Loeber, Burke, Lahey, Winters, & Zera, 2000). This then led to the idea that ADHD was a key component in the prediction of which youth would develop into the most antisocial or delinquent individuals. However, once longitudinal data became available and better statistical controls were introduced, it became evident that ADHD (or its pattern of symptoms) did not consistently predict later CD or serious forms of delinquency if prior CD or delinquency was taken into account (Loeber et al., 2000).

Disenchanted with ADHD, researchers became convinced that psychopathy in adulthood could have psychopathy-like antecedents in childhood and adolescence (Frick, Cornell, Barry, Bodin, & Dane, 2003; Lynam, Caspi, Moffitt, Loeber, & Stouthamer-Loeber, 2007; Pardini, 2006), and hypothesized that early signs of psychopathy could aid in the discrimination between those who were and those were not at highest risk of later antisocial outcomes (see Frick, Blair, & Costellanos, this volume). Most of the research, however, has focused on boys rather than girls, and it remains to be seen to what extent early forms of callous-unemotional behavior predict serious delinquency by late adolescence or early adulthood in girls.

Homotypic and Heterotypic Continuity

Are girls' disruptive and delinquent behaviors as stable as boys'? Loeber, Burke, and Pardini (2009), examined girls' disruptive behaviors across ages 5–12 years and showed that year-to-year stability of factor scores for behaviors, including oppositional behavior/conduct problems, relational aggression, and callous-unemotional behavior, was high for parents' reports (ICC = 0.7–0.88) and slightly lower for

teachers' reports (ICC = 0.56–0.83). Other studies showed that the stability of mental health problems, including externalizing behaviors, in girls is either as high as in boys or higher (Costello, Mustillo, Erkanli, Keeler, & Angold, 2003; Tremblay et al., 1992; Verhulst & van der Ende, 1991; Zoccolillo, Pickles, Quinton, & Rutter, 1992). It should be noted that some variation between genders may exist, in that girls compared to boys may hold grudges longer and that boys go through more frequent cycles of conflict and reconciliation than girls (Maccoby, 2004). The difference, however, is that on average girls tend to outgrow or desist from delinquent behavior at a younger age than boys (Rutter, Giller, & Hagell, 1998), although the persistence of delinquency is lower for girls than boys (see review and analyses by Lanctôt, Émond, & Le Blanc, 2004).

One of the features of disruptive and delinquent behavior that has bedeviled its study is that the behaviors change in manifestations with age. This is not only true for aggression typical for the preschool years, compared to violence (robbery, rape, and homicide) from late adolescence onwards but also for behaviors such as shoplifting, car theft, and breaking and entering, which are practically unknown in the preschool years and gradually increase with age. We will review several aspects of this heterotypic development, particularly as they differentiate boys and girls. The study of these developments can be conceptualized in at least three ways. First, does condition A predict condition B? Second, to what extent is A a necessary precursor to B (i.e., do most individual with B develop A first)? The third question concerns the stability of individual symptoms over time.

ODD and CD as predictors of later deviance. Turning to the first question (does condition A predict condition B?), tests of a predictive, developmental relationship between ODD and CD have provided somewhat mixed support. In the Developmental Trends Study—a sample of clinically referred boys followed from ages 7–12 to 18 years—ODD was predictive of later CD, with no reciprocal (i.e., CD→ODD) relationship (Burke, Loeber, Lahey, & Rathouz, 2005). In a mixed-sex general population sample, however, dimensional measures of oppositionality at ages 4–7 years showed negligible prediction to dimensional measures of later conduct problems (at ages 8–13 years) once initial levels of conduct problems were controlled (Lahey et al., 2009).

The final step in the developmental model posits prediction not only from ODD to CD but also from CD to a diagnosis of APD in adulthood. Here, current evidence is generally more consistent: APD is more typically an outcome of CD than of ODD (Burke, 2008), though one study has reported an independent relationship between ODD and APD (Langbehn, Cadoret, Yates, Troughton, & Stewart, 1998).

Not all disruptive and delinquent behaviors are equally stable. Research on the stability of male violence from ages 7–25 years in the Pittsburgh Youth Study shows that violence is much more stable over time than serious forms of theft; trajectory analyses show that desistance from theft tends to occur earlier than desistance from violence (Loeber et al., 2008). Whether this applies equally to girls is not known.

Once established, there is evidence that girls' aggression predicts later aggression over several developmental transitions, and stability looks similar for girls and boys (Cairns, Cairns, Neckerman, Feguson, & Gariep, 1989; Caspi & Silva, 1995; Prior, Smart, Sanson, & Oberklaid, 2001). Caspi and Silva (1995) found that difficult temperament at age 3 years predicted conduct disorder in both boys and girls at age

15 years, and Prior et al. (2001) found that the strongest predictors of adjustment at age 12 years for both boys and girls were previous behavior problems along with self-regulation and maternal rating of overall child difficulty. The long-term consequences for girls persistently engaging in such behaviors are quite serious (e.g., Giordano, Cernkovich, & Lowery, 2004; Lewis et al., 1991). Lewis et al. (1991) followed up 21 female delinquents from a correctional facility and found that most were seriously impaired neuropsychiatrically. They were likely to be heavy substance users, suicidal, enmeshed in violent relationships, and unable to care for their children. In addition, mortality rates were high.

Girls and boys with a disorder are not equally at risk for comorbid conditions. The *gender paradox* states that the gender with the lowest prevalence of a disorder is at higher risk of developing another, relatively rare, comorbid condition than the gender with the higher prevalence of a disorder (Eronen, Hakola, & Tiihonen, 1996; Loeber & Keenan, 1994). This is also consistent with Robins' (1986) observation that in adulthood "an increased rate of almost every disorder was found in women with a history of disruptive behaviors" (p. 399), including ADHD, anxiety disorders, and mood disorders (see also Costello et al., 2003; Teplin, Abram, & McClelland, 1997; Zoccolillo, 1993). Eronen et al. (1996) found that the risk of alcoholism and APDs in adult Finnish convicts for homicide was 3 times higher for females than males. It is likely that because conduct disordered girls are a smaller group than are conduct disordered boys, that these girls are more comparable in severity to the more extreme boys. In summary, female gender may carry a protective effect for mild disruptive and delinquent behavior, but the most severe girls are at significant risk for serious general maladjustment and comorbid disorders.

ODD as a predictor of other outcomes. In the Great Smoky Mountains Study, a longitudinal population study following 1,420 children from age 9 years to adulthood—a follow-up of the sample into early adulthood (Copeland, Shanahan, Costello, & Angold, 2009)—showed that (independent of other adolescent disorders) ODD in adolescence was associated with increased risks of anxiety and depression in early adult life but that CD showed no similar predictions to internalizing disorders. Differential prediction to internalizing and externalizing outcomes has also been noted in the Developmental Trends Study (Burke et al., 2005), possibly echoing the more diffuse patterns of comorbidity in ODD than CD reported in childhood and adolescent samples (Simonoff et al., 1997). These studies suggest that the symptoms of ODD, which focus on irritable, headstrong, and vindictive behaviors, do not predict the development of aggression, at any rate as measured by CD.

Developmental Patterns

We will consider three aspects of the development of boys' and girls' disruptive and delinquent behavior and examine when and where gender differences occur: developmental progressions, developmental types, and developmental trajectories.

Developmental progressions. The study of developmental progressions investigates whether condition A (i.e., a disorder or a behavior) is a necessary precursor for the development of condition B. For instance, is ODD a necessary condition for the emergence of CD, and is this different for each gender? Costello and colleagues (Costello et al., 2003; Rowe, Maughan, Pickles, Costello, & Angold, 2002) examined the association between CD and ODD across childhood and adolescence in the Great Smoky Mountains Study. Almost 20 % of boys and 10 % of girls met criteria for ODD or CD at least once between the ages of 9 and 16 years: CD was diagnosed on at least one occasion in 8.6 % of youth (3.7 % girls, 13.2 % boys) and ODD in 9.7 % (7.8 % girls, 11.6 % boys). Among youth who ever met criteria for CD, for example, substantial proportions (57.2 % of boys and 46.9 % of girls) never met criteria for ODD at any study wave; for disruptive girls, "ODD only" was also a common pattern. Among youth who did meet criteria for both disorders, the most common pattern was for the two disorders to onset at the same assessment wave, and the next most common for CD to be diagnosed before ODD. The expected progression from ODD to CD was comparatively rare, especially in girls.

Around one half of boys and almost three quarters of girls with ODD never met full criteria for CD, and the majority (55 %) of CD cases never received a diagnosis of ODD, although in both cases youth in these "pure" diagnostic categories had higher levels of subthreshold symptoms of the other diagnosis than children who never met criteria for CD or ODD. These findings are consistent with results from other epidemiological studies (Loeber et al., 2009) in showing that in community samples CD and ODD are less closely associated than was suggested by data from referred groups at the time that DSM-IV was formulated. These findings suggest that, especially in girls, aggression as measured by CD is part of a different dimension of behavior from that captured by ODD, in most cases. However, at the higher levels of disruptive behaviors seen in clinical samples, the two types of behavior more frequently co-occur.

Another line of research has focused on investigating developmental pathways of specific categories of disruptive and delinquent behaviors. Two key issues are, first, whether individuals' development to serious delinquency is orderly and, second, whether it can be best represented on a single or on multiple pathways. After initial research comparing single and multiple pathways, Loeber et al. (1993) formulated a model of three pathways for boys that best fitted the data, and which was replicated across the three samples in the Pittsburgh Youth Study (Loeber et al., 1993; Loeber, DeLamatre, Keenan, & Zhang, 1998). The three pathways were: (a) An *Authority Conflict Pathway* prior to the age of 12 years, that starts with stubborn behavior, has defiance as a second stage and authority avoidance (e.g., truancy) as a third stage; (b) *A Covert Pathway* prior to age 15 years, that starts with minor covert acts, has property damage as a second stage and moderate to serious delinquency as a third stage; and (c) *An Overt Pathway*, that starts with minor aggression, has physical fighting as a second stage and more severe violence as a third stage. Tolan, Gorman-Smith, and Loeber (2000) have replicated the pathway findings in a sample of African American and Hispanic adolescent males in Chicago and in a male and female nationally representative the US sample of adolescents. Replications have also been undertaken in

the Denver Youth Survey and the Rochester Youth Development Study (Loeber, Wei, Stouthamer-Loeber, Huizanga, & Thornberry, 1999).

Developmental pathways from less to more serious behaviors have also been documented in girls (Gorman-Smith & Loeber, 2005). Gorman-Smith and Loeber (2005) conducted a partial test of the applicability of these developmental pathways to girls by using self-report data from the National Youth Survey. They found some support for the role of ODD symptoms as stepping-stones to CD, but not as strong as for boys. It is possible that developmental pathways in disruptive and delinquent behavior in girls are more complex than formerly thought. For instance, Loeber, Stouthamer-Loeber, Hipwell, Burke, and Battista (in press) found evidence that girls' irritability and anger increased with age. The authors identified a developmental pathway starting with irritability that in a minority of girls evolved into anger during preadolescence. Angry emotionality in girls is an antecedent to both later conduct problems and depression for Caucasian but not African American girls (Burke, Hipwell, & Loeber, 2010).

In sum, there is a normative tendency to the undercontrol of physical aggression in the toddler years (although not for all children) and to normative improvement through late childhood, with girls improving more rapidly than boys. Girls tend to engage in more indirect aggression, which is a more frequent behavior in later childhood, presumably because it requires more verbal and cognitive skill. In contrast, the stability of disruptive behavior over time seems to be similar for boys and girls.

Developmental types of delinquency and gender. Two of the oldest and most widely accepted conclusions regarding delinquency and crime are first that involvement in crime diminishes after late adolescence, and second that males are more likely than females to offend at every age (Steffensmeier & Allan, 2000). The number of youth arrested increases dramatically in early adolescence; for example, arrest rates almost double from age 14 to 15 years, and decreases sharply after late adolescence. Peak ages at arrest (across the entire life span) in 2008 were at ages 18 and 19 years for both boys and girls in the USA (Uniform Crime Reports, 2009). The number of girls becoming involved in antisocial and delinquent behaviors appears to have increased in recent years (American Bar Association and The National Bar Association, 2001), but boys still show considerably higher arrest rates, with girls accounting for just 24 % of arrests at the peak ages of 18 and 19 years in 2008 (Uniform Crime Reports, 2009).

Dual taxonomy models of delinquency—notably Patterson's model of early and late starting delinquents (Patterson & Yoerger, 1993) and Moffitt's (1993) widely cited model of (a) early-onset/life course persistent offenders and (b) adolescent-limited offenders—have been highly influential in the field of delinquency development for males. Fontaine, Charbonneau, Vitaro, Barker, and Tremblay (2009) reviewed the empirical literature to examine the extent to which gender differences applied to these types and reported that early-onset persistent cases in girls, although rarer than in boys, do occur. Unlike the Moffitt (1993) theory, Fontaine et al. (2009) reported that adolescent-limited girls may present adjustment problems in adulthood at prevalence levels that undermine the notion that their problems are transitory only. Also, Fontaine's review highlighted that a proportion of women (3–4 %)

started their delinquent or criminal career in adulthood. They noted that it is unclear to what extent late-onset females are "truly problem free earlier in life" (p. 375).

There is increasing evidence both due to the studies of conduct problems and delinquency that have extended recently well into the decade of the twenties, and to the more sophisticated modeling techniques that have become available (Muthén & Shedden, 1999; Nagin, 1999), that the life-course persistent/adolescent-limited model is not a strong fit for males either. Similar to Fontaine et al.'s (2009) findings for females, males show ongoing problems and crime in adulthood, even if they were not in the most persistent trajectory in adolescence, also show a group who onset for crime in adulthood (Capaldi, in press; Wiesner, Capaldi, & Kim, 2007). Further, the most severe offenders in adolescence show the strongest relative trends to desistance in adulthood relative to lower frequency offenders. Studies on boys also clearly show that there are many variations on the life-course type (e.g., Loeber et al., 2008), and it is expected that longitudinal studies on girls will also demonstrate variants on the life-course type. Thus, many recent findings indicate that the dual taxonomy models need considerable revision for both boys and girls, and that it is rather adding to confusion in the field to assume that the model is a good fit for boys and that any deviation from the model for girls is due to gender differences.

Developmental trajectories and gender. Whereas most work on the formulation of developmental types initially was conceptual, the study of developmental trajectories refers to the empirical classification of individuals according to their behavior development over time. Thus, quantitative methods are used to determine which individuals have a high probability of belonging to one rather than to other developmental types (examples are provided below). There are fewer studies on developmental trajectories in girls than boys (Fontaine et al., 2009). In a study examining heterogeneity in trajectories of physical aggression from ages 2 to 8 years, Côté, Vaillancourt, Barker, Nagin, and Tremblay (2007) identified four groups; namely, consistently high (15 %), moderate desisters (44 %), low desisters (36 %), and low (5 %). Boys were overrepresented in the two higher groups (e.g., the consistently high group was composed of 54 % boys and 46 % girls, and the low desister group was composed of 53 % girls and 47 % boys). Thus, both studies indicated higher levels of physical aggression by boys in childhood. As can be seen, however, there were considerable gender similarities in this study and in the Tremblay et al. (1996) study previously described. In Zahn-Waxler and Polanichka's (2004) study, boys and girls showed a similar developmental trend toward improvement or desistence with age. In Côté, Boivin, et al.'s (2007) study, gender representation in the four developmental patterns identified showed only a modest imbalance (e.g., high indirect aggression and high physical aggression: 12.5 % for boys and 11.0 % for girls).

Increasingly, researchers are documenting the development of indirect aggression (essentially, undermining someone behind their back; e.g., spreading false rumors), which has been hypothesized to be a type of aggression more characteristic of girls than boys. Côté, Boivin, et al. (2007) identified two trajectories of indirect aggression from ages 4 to 8 years: persistently low versus initially high and then increasing (the latter being 32 % of the sample). The high-increasing group comprised 58 % girls and 42 % boys. Thus, indirect aggression showed a different

developmental trend from physical aggression in increasing across childhood and being more characteristic of girls, although boys also engaged in this behavior. The gender representation was not, however, highly unbalanced. A missing piece in the research on young children seems to be on direct verbal conflicts, and it is unclear if gender differences would be found in this area.

It may be argued that, as regards physical aggression at least, the early childhood period involves increasing control or learning to inhibit such behavior by around school entry (discussed in more detail below). The picture also involves developmental changes in physical strength (i.e., an 8-year-old can hit harder than a 2-year-old), cognitive abilities (e.g., the language skills required for more subtle forms of aggression such as relational aggression), and types and severity of aggression employed (e.g., involving a weapon).

Explanation of Gender Differences

Below we review key issues regarding evidence for gender similarities and differences in the development of disruptive and delinquent behaviors.

Inhibitory control and temperament. One of the chief factors theorized to account for early differences among children in levels of aggression, and also for the differences across boys and girls, is the temperamental dimension of inhibitory control and relatedly impulsivity (Cole & Zahn-Waxler, 1992; Keenan et al., 2010; Prior, Smart, Sanson, & Oberklaid, 1993). In an influential theory of aggression and conduct problems, namely the general theory of crime, Gottfredson and Hirschi (1990) posit that poor inhibitory control established in childhood, caused by temperamental risk factors and poor parenting that fails to promote self-control, is the only explanation needed for such problem behaviors. Others consider that this theory overstates the case and that there are a number of factors, such as contextual and social influences, affecting aggressive behaviors across the life span, including key factors such as deviant peer influences (Dishion & Patterson, 2006). Nevertheless, there is much support for the view that poor inhibitory control is a major causal factor and that there are gender differences in this risk factor.

Temperament is generally considered to be inherited or genetically related across generations. Genetic loading may be associated particularly with dimensions of temperament that relate to brain activity and, thus, to neural pathways (Hill, 2002). Two behavioral systems, the Behavioral Activation System (BAS) and the Behavioral Inhibition System (BIS), are hypothesized to be critical to the way an individual responds to environmental stimuli that offer reward or punishment (Gray, 1987). The BAS is posited to activate behaviors in response to likely rewards, and the BIS to inhibit behaviors when punishment cues are present. During behavioral activation, the dopaminergic system in the brain is believed to be facilitating approach responses. The noradrenergic and serotonergic systems are believed to be associated with behavioral inhibition (Rogeness & McClure, 1996). Temperamental tendencies related to problematic aspects of approach and withdrawal include poor inhibitory control (i.e., poor self-control), a relatively high activity level, a greater

vulnerability to feelings of negative affect and anger, and sensation seeking or a higher tolerance for risk taking (e.g., Rothbart, Posner, & Hershey, 1995).

There is evidence of gender differences in inhibitory control in early childhood. Snyder, Prichard, Schrepferman, Patrick, and Stoolmiller (2004) examined impulsivity and inattention in children entering kindergarten. They found that, according to parent report and observer ratings, boys showed significantly higher levels of such problems than girls. In addition, boys were observed to show significantly higher amounts of time off task during academic work periods in the classroom. Romano, Tremblay, Farhat, and Côté (2006) examined the development of hyperactive symptoms from ages 2 to 7 years in a population-based Canadian sample (NLSCY). Boys were more than twice as likely as girls to be in the group of 7 % of children who showed high and persistent levels of hyperactivity across this age period.

Hormonal contributions. As discussed by Capaldi (in press)—in considering the large difference between adolescent males and females in aggression, in conjunction with the sharp peak of aggressive and related behaviors in adolescence—hormonal differences in females and males should be considered. Differences in levels of sex hormones, in particular, may be involved. On the average, a man produces 40–60 times the level of testosterone as a woman. Book, Starzyk, and Quinsey (2001) conducted a meta-analysis of 45 independent studies of the association of testosterone and aggression. Findings supported a weak positive relationship *within* men or women, and the association was found to be stronger at younger (ages 13–20 years) than at older ages. In a longitudinal study, van Bokhoven et al. (2006) found that boys who developed a criminal record had higher testosterone levels at age 16 years, and testosterone was associated with aggression and self-reported delinquency. In a review of studies of the association between hormones and aggression in children and adolescents, Ramirez (2003) concludes that the origin of gender-based differences in aggression must lie in neuroendocrinological events occurring during prenatal or early postnatal life and that testosterone has a complex and indirect effect on aggression. Mong and Pfaff (2003) found that the lifetime curve of murders of unrelated men by men follows the testosterone curve in increasing and decreasing across the life span in a variety of cultures and that both testosterone and its metabolites, as well as serotonergic projections to the forebrain, play roles in the neurobiological controls over aggression. Kuepper et al. (2010) found evidence of interactive effects of testosterone and serotonin on aggression in men but not women. Thus, it appears that testosterone plays a role in aggressive conduct problem behaviors and relates to gender differences in levels of conduct problems.

Socialization in the family. There is a general consensus that the development of antisocial behavior involves a prolonged process of interplay between the characteristics of the individual youth (e.g., temperamental characteristics related to approach and inhibition) and their key social environments (e.g., Baltes, 1983; Cairns & Cairns, 1995; Elder, 1985). These environments include those created by family, by school personnel and students, by peer groups, and by pertinent community members. The social interactions that occur within each environment may affect antisocial behavior across the life span. The topic of differential socialization of boys and girls regarding externalizing behaviors has been covered

extensively elsewhere, and detailed treatment of this topic is not within the scope of the current chapter (see e.g., Keenan, Loeber, & Green, 1999; Keenan, Stouthamer-Loeber, & Loeber, 2005; Kroneman, Loeber, Hipwell, & Koot, 2009; Loeber et al., 2009; Pepler & Craig, 2005; Putallez & Bierman, 2004; Zahn-Waxler & Polanichka, 2004). We focus here on the family environment. Conduct problems in later childhood show many similar family risk factors for boys and girls (e.g., harsh parenting, low socioeconomic status, parental risk behaviors). For example, hostile parenting has been found predictive of high and persistent hyperactivity (Romano et al., 2006).

Similar to boys, disruptive behavior in girls is more common in dysfunctional than in well-functioning families (e.g., Caspi & Moffitt, 1991; Keenan et al., 2005; Kroneman et al., 2009) and in families where maltreatment and a high degree of conflict occurs (e.g., Giordano & Cernkovich, 1997; Widom, 1978), and more girls than boys in such environments direct their aggression at family members (e.g., Pepler & Craig, 2005). However, more seriously disruptive girls—i.e., those involved in delinquency—tend to come from more problematic home environments than do delinquent boys. Thus, girls within the juvenile justice system tend to have experienced particularly high levels of family disruption, including multiple father figures or foster care and high levels of sexual abuse (Leve & Chamberlain, 2004; Smith, Leve, & Chamberlain, 2006). These findings could indicate that, given that girls tend to be less at risk temperamentally for disruptive behaviors, it takes stronger environmental risk for girls than for boys to become involved in substantial levels of delinquency. Leve, Kim, and Pears (2005) found evidence that girls may be particularly at risk from a combination of harsh discipline and low inhibitory control (high impulsivity and low fear/shyness). These findings should be viewed within the context that far fewer girls than boys are involved in the juvenile justice system.

Parenting is one of the major foci of research on gender differences in socialization. More knowledge is available about mothers' than fathers' parenting because of lack of focus on fathers in most studies. A meta-analysis by Lytton and Romney (1991) found that the only socialization area that displayed a significant gender effect in North American studies was encouragement of sex-typed activities by mothers and fathers. Studies from Western countries other than North America indicated that boys experienced more physical punishment from parents than girls. The authors concluded that gender differences in aggression in North America, at least, were likely not due to differential parenting practices, such as less harsh punishment in response to girls' versus boys' aggression. However, a later review by Keenan and Shaw (1997) concluded there is some evidence that parents interact with boys and girls differently. They found that during early childhood (through age 5 years) mothers encouraged their girls more than their boys to have concern for others, share toys with their peers, and behave prosocially. In addition, there was some evidence that girls rather than boys were responded to positively for being shy. Teachers were found to give less attention to girls than to boys. When girls did receive positive attention from teachers, it tended to be for less active play and for dependent behavior.

There is evidence that girls may be more sensitive to disruptions in home environments than boys, for example, that they are more affected by parental divorce (Keenan et al., 1999). Conflict in mother–daughter dyads may be particularly important for girls at risk for conduct disorder (Pepler, 1995). On the other hand, Webster-Stratton (1996) found that parents of girls versus boys aged 3–7 years referred to a clinic for behavior problems did not differ in their observed patterns of interactions, suggesting that parenting of disruptive boys and girls is quite similar. In sum, although there is evidence for some differences in parenting of boys and girls related to conduct problem behaviors, these differences do not appear to be large.

A reciprocal relationship has been found between girls' conduct problems and parental punishment and warmth. Hipwell et al. (2008) showed that both parental punishment and warmth were uniquely predictive of changes in girls' conduct problems, and that girls' conduct problems predicted changes in mothers' harsh punishment over time.

Peer factors. Girls' greater orientation to interpersonal relations relative to boys has been well documented (e.g., Keenan & Shaw, 1997; Moretti, Holland, & McKay, 2001; Turner, Dindia, & Pearson, 1995; Zahn-Waxler, Schiro, Robinson, Emde, & Schmitz, 2001). Much of girls' social lives revolve around dyadic or small group interactions with friends who are similar to themselves (e.g. Hartup, 1996). Such homophily is likely to occur as a result of both initial choice of friends (selection) and the process of mutual influence over time (socialization). Although association with a deviant peer group appears to be important for antisocial females (Aseltine, 1995), little is known about the nature of these relationships, or the mechanisms by which peer relations are linked with later maladjustment. For example, the association between susceptibility to deviant peer influence and the emergence of CD in girls has rarely been tested.

Intimate and supportive aspects of romantic relationships appear to be particularly meaningful to adolescent girls (Buhrmester & Furman, 1987). Adolescent conduct problems are also associated with difficulties forming and/or maintaining supportive and harmonious relations with an intimate partner (e.g., Bardone, Moffitt, Caspi, & Dickson, 1996; Capaldi & Clark, 1998). Breakdown of these relationships is associated with an increased risk of crime and other problem behaviors among high-risk individuals (Laub, Nagin, & Sampson, 1998). To date, few studies have focused on the risks for CD of relationship failure among females, and research is also needed to understand the protective function that a supportive intimate relationship may have for high-risk girls.

Rejection by peers is known to be associated with CD, particularly aggression (Coie, Terry, Lenox, & Lochman, 1995; Dodge et al., 2003; Miller-Johnson, Coie, Maumary-Gremaud, & Bierman 2002). Unlike boys who may experience both peer approval and disapproval for bullying (Milich & Landau, 1988), female bullies are likely to be rejected by the peer group (Pepler, King, Craig, Byrd, & Bream, 1995). Downey and colleagues' work also shows that children's sensitivity to rejection places them at risk for behavioral and emotional problems (Feldman & Downey, 1994). In a proportion of cases, rejection sensitivity experienced in a peer context is a continuation of feelings of rejection by parents, which are often exacerbated by

the parents' physical maltreatment or emotional neglect (Feldman & Downey, 1994). We consider rejection sensitivity to be an important parameter in the etiology of both CD and MDD, in that it is often accompanied by *either* anger or anxiety, depending on situational and temperamental factors. Although withdrawn and submissive behaviors generally predate peer victimization, risk is enhanced when girls already have behavior problems (Schwartz, McFadyen-Ketchum, Dodge, Pettit, & Bates, 1999).

In summary, there are a number of possible explanations—temperamental, familial, and peer factors—for the differential patterns of physical aggression observed for boys and girls in childhood. In addition, girls tend to be developmentally more advanced than boys in some key areas, needing less correction by parents. For example, girls tend to show more rapid development of language (Kimura, 2000), and being able to verbally express desires and emotions more readily may relate to greater self-control and less expression via physical aggression. This is likely to be associated with more rapid brain development in girls than in boys until adolescence (see further below).

Genetic factors and correlates of CD and ODD. Simonoff (2001) reviewed genetic influences on sensation seeking, impulsivity, and physical aggression. There are promising findings of associations of several candidate genes and implicated brain metabolic pathways (Susman & Pajer, 2004). Associations with neurotransmitters hypothesized to be associated with low behavioral inhibition have been found, including lower noradrenaline (Rogeness et al., 1984) and serotonin (Kruesi et al., 1990; Moffitt et al., 1997) metabolic levels. Genes affecting dopamine function have been found to be associated with hyperactivity (Thapar, Holmes, Poulton, & Harrington, 1999). Caspi et al. (2002) examined the association of child maltreatment and a genetic variant that results in brain monoamine oxidase levels being too low to break down some neurotransmitters (e.g., norepinephrine, serotonin, and dopamine) that may become overactive because of maltreatment. Findings indicated an interaction effect between this polymorphism and maltreatment in predicting antisocial behavior. Taylor and Kim-Cohen's (2007) meta-analysis is relevant in that it demonstrates that across studies the *interaction* between genetic and environmental factors results in a risk that is far higher than the sum of the individual risk factors. However, the results show little agreement among the studies about the observed interaction mechanisms. Importantly, Hicks et al. (2007) found increasing genetic variability and heritability of externalizing disorders in men, but a decreasing genetic variability and increasing environmental effects for women.

Concluding Comments

The findings of this review indicate that there are more similarities than differences in developmental patterns and stability of conduct problems and aggression in girls and boys, particularly in the prepubertal years. The largest gender differences in such behaviors in girls and boys appear to be in the differences in frequency of

delinquent acts at adolescence, at least at the level that reaches official attention, and in lower levels of physical aggression to persons outside the family among girls (e.g., stranger assaults). Physiological protective factors for girls are implicated in these gender differences, particularly more rapid brain maturation for girls and differences in sex hormones—particularly lower levels of testosterone. The strongest difference in risk factors related to individual behavior appears to be differences in inhibitory control, and in socialization appears to be encouragement of gender-typed behaviors by parents. Girls' relative resilience to physical aggression however, is tempered by the findings that CD is the second most common psychiatric diagnosis for girls, and second, that long-term outcomes for girls with higher levels of aggression and conduct problems include pervasive and severe psychosocial problems affecting themselves and their families for decades.

This review by necessity was selective. On the one hand, research findings on gender-related aspects of the development of disruptive and delinquent behavior have advanced much over the past decades, partly aided by the increasing availability of longitudinal data and partly by the increasing availability of sophisticated analytic and statistical tools. As illustrated here, many primary research questions remain. Importantly, the research base for disruptive and delinquent behavior in girls remains small in comparison of that for boys. Relatedly, the knowledge of risk and protective factors and the use of such factors in prevention and intervention studies to reduce individuals' and population level of these problems behaviors is inadequate. Evaluation studies are much needed that prevention and interventions work best for girls to reduce both less serious and more serious outcomes over the life span (Hipwell & Loeber, 2006).

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Chapter 7
Tracking the Multiple Pathways of Parent and Family Influence on Disruptive Behavior Disorders

Patrick H. Tolan, Kenneth Dodge, and Michael Rutter

Parenting and related family characteristics are perhaps the most studied and documented contributors to risk for disruptive behavior disorders among children. They are also the most salient protective factor against such problems. Family-focused interventions (both preventive and treatment) are among the most effective for disruptive behavior disorders (Dishion & McMahon, 1998). Indeed, their effectiveness underscores the importance of family factors in the cause and solutions for this problem.

Parenting is a broad construct encompassing multiple components—and the focus of a voluminous research literature (Parke & Buriel, 1998). Within this literature, there is considerable variation in how family relationship characteristics have been conceptualized and studied, yet remarkably little attention to the specifics of their interdependence or conceptual relation. Parenting practices and family relationship qualities are also related to other familial influences, such as genetic transmission of personality and behavioral characteristics of the parents, the extended family, and familial cross-generational consistency in behavior and risk and protective influences, as well as the social context of childrearing and family development (see Parke & Buriel, 1998, for a cogent summary of the broad ecological perspective). Within each of these broad domains, researchers have formulated elemental constructs and theories of interrelations of these elements and effects on

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development, risk, and expression of problems. Some of these constructs have considerable reference and use, but many arose because of specialized interests and only have meaning among a small set of researchers. A major issue of interest is the distinction between genetic influence and parenting influence on disruptive behavior disorders. The possibility of genetic influence tempers many reported findings on parenting in the literature because these two influences are correlated. Furthermore, discoveries of gene-by-environment interactions indicate that each must be considered in tandem with the other.

Encompassing and summarizing all or even most of the essential findings of the field is well beyond a single chapter and much broader than is pertinent to this volume. However, we do attempt to locate within a broad biopsychosocial and ecological perspective important avenues for parental and family influence on disruptive behavior disorder. To do so, we first describe some of the key conceptual considerations in understanding how parenting and family relationship characteristics can influence disruptive behavior problems. Next, we describe the current state of knowledge about several of the key characteristics or processes of influence. In the final section we outline important areas of further study, including an agenda for moving the field forward and improving our ability to determine best interventions to prevent and treat and perhaps lead to cure of disruptive behavior disorders.

Parents and Families as Developmental Influences on Disruptive Behavior Risk

The multiple avenues of influence for risk for disruptive behavior disorders add complexity to any attempt to understand this process. There is not, and cannot be, one single cause or even a primary or more critical cause of disruptive behavior disorders (see Academy of Medical Sciences, 2007). The multifaceted origins also mean that the mix and balance of influences can vary from individual to individual. Thus, models can serve for general description, but they may not be similarly applicable to a given subgroup or person. Yet to be established are processes that capture the multiple avenues of parental influence on disruptive behavior, the specificity and distinction of these processes, and the conditions under which influence on subgroups or individuals varies.

Characterizing Disruptive Behavior

Disruptive behavior is far from homogenous or easily characterized, and many of the characteristics of the disorder are common in the overall population. Even individuals who evidence an identifiable clinical pattern may present different symptoms of the disruptive behavior disorder (Cicchetti, 2006; see Carter et al. in this volume, and Frick et al. in this volume). The relation of these distinctions to

parenting and family influences and to the avenues for such influence also varies. Thus, for example, antisocial behavior associated with psychopathic features has a higher heritability (i.e., a stronger genetic liability) than that not associated with psychopathy (Viding, Larsson, & Jones, 2009). It also carries a poorer outcome and probably a different response to family influences (Dadds & Rhodes, 2009). Similarly, antisocial behavior accompanied by overactivity/inattention or attention deficit disorder with hyperactivity (ADHD) also involves a stronger genetic component than antisocial behavior without these features (Silberg et al., 1996). Nevertheless, it appears that the psychopathy is not due to associated overactivity/inattention (Viding et al., 2009). In addition, molecular genetic research has shown that COMT (Catechol-O-methyltransferase gene) is not associated with either ADHD or antisocial behavior as such, but is significantly associated with antisocial behavior in individuals with ADHD (Caspi et al., 2004).

Numerous studies have shown the strong co-occurrence of oppositional-defiant disorder (ODD) and conduct disorder; it is also clear that there is a substantial shared genetic liability (Kimonis & Frick, 2010). On the other hand, there is growing evidence that the irritability component of ODD is different in its association with affective disturbance and suicidal behavior (Pickles et al., 2010; Stringaris, Cohen, Pine, & Leibenluft, 2009; Stringaris, Maughan, & Goodman, 2010). These findings underscore the limitation of these categorizations in distinguishing causes, even when they can be differentially related to various family contributions.

Developmentally framed typologies provide seemingly more useful distinctions, although the findings testing their validity are still limited and not always consistent. Thus, Moffitt (1993) has argued for a split between life course-persistent antisocial behavior (meaning a variety that begins in childhood and persists into adult life) and adolescence-limited antisocial behavior. It is well established that the former is much more likely to be associated with neurodevelopmental impairment (Odgers, Caspi, et al., 2007) and with family dysfunction (Odgers, Milne, et al., 2007). However, it remains unclear whether the difference is categorical or dimensional (i.e., whether adolescent-limited antisocial behavior involves no family influences or only different influence, or whether it involves the same family features but with a weaker association). Barker and Maughan (2009) found that early maternal anxiety, harsh parenting, and child activity all differentiated children with early-onset persistent conduct problems from those with childhood-limited conduct problems.

In another approach, Tremblay (2003) argued for a distinction between physically aggressive and nonaggressive varieties of antisocial behavior. Although there is much evidence to support this differentiation, it is not known whether the forms of aggression differ with respect to family influences. In a similar vein, Wakschlag, Tolan, and Leventhal (2010) suggested that disruptive behavior disorders could be differentiated by key symptoms along dimensions of aggression, noncompliance, temper loss or anger, and low concern for others (see both Carter et al. and Frick et al. in this volume for further deliberation).

Clearly, the extent of and multidimensionality of family influences on disruptive behaviors are complex and may vary by behavior of interest. As we consider other further elaborations that are important in considering family influences, this initial

complexity may be important for how specific research endeavors might be formulated as well as how theoretical linkage of forms of influence would be organized.

A Multisystem, Transactional, Developmental Process of Effects

Another complicating factor in the interplay of timing and variety of family influences on disruptive behaviors adds is the bidirectionality of relationships: parents influence children and children influence parents (Kerr, Stattin, & Burk, 2010; Laible & Thompson, 2007; Pettit & Arsiwalla, 2008). Bidirectionality has been suggested using a variety of research strategies including experimental designs (Anderson, Lytton, & Romney, 1986) and the effects on parents of changing child behavior (Brunk & Henggeler, 1984; Schachar, Taylor, Wieselberg, Thorley, & Rutter, 1987).

As Parke and Buriel (1998) note, an interactive systemic perspective is needed to relate components and the overall influence of families on child development. Yet, as they further delineate, it is important to attend to the individual, dyadic, and multiperson levels of influence as well. That is, individual characteristics can evoke different reactions or responses to environmental influences. Similarly, dyadic relationships between parents, and between each parent or parent figure and a child, have specific influences on development that are not simply a reflection of the family system or reducible to the sum of the two personalities in the dyad. For example, Cowan, Cowan, Schulz, and Heming (1994) documented that marital interaction quality, particularly conflict and hostility level, predicted child risk for externalizing symptoms. Family discord and unresolved conflict, particularly of parents, are other examples of this type of influence. Rutter (1971) compared happy and unhappy marital separations in predicting antisocial behavior in the child, finding that risk for antisocial behavior did not increase in happy separations. Similarly, the risk for antisocial behavior was much greater in the case of divorce than parental death. Fergusson, Horwood, and Lynskey (1992), using the Christchurch longitudinal study, found that risks for behavior problems were much more strongly associated with family discord than family separation. Mother-child and father-child relationships may also have differing influences. For example, it appears paternal influences on risk may be tied to the quality of the marital relationship more than maternal parenting influences (Belsky, Youngblade, Rovine, & Volling, 1991). Also, the impact of paternal involvement may differ from maternal involvement (DeGarmo, 2010).

Family influences are also irrevocably intertwined with social context (Rutter, 1999). Practices, values, beliefs, and other social influences of the overall society also affect how family influences disruptive behavior risk. For example, conditions of poverty, including fewer parenting resources and greater threats to child well-being, make effective parenting more difficult (Conger, Neppl, Kim, & Scaramella, 2003). In addition, it is possible that family influences differ for children growing up in a high-risk neighborhood (Peeples & Loeber, 1994; Stouthamer-Loeber, Loeber, Wei, Farrington, & Wikstroem, 2002; Tolan, Gorman-Smith, & Henry, 2003).

For example, close parental supervision and control may be more necessary when the neighborhood risks are high (Gorman-Smith, Henry, & Tolan, 2004).

There is also uncertainty from empirical information to date about the variation by child gender or ethnic and cultural group in parenting and other family factors as effects on disruptive behavior disorders (see Loeber et al. in this volume for a discussion of the gender patterns and contributors issues). For example, analysis of Dunedin longitudinal data suggests that, rather than differential family influences, it is the greater frequency of neurodevelopmental impairment in males that contributes to their markedly higher rates of antisocial behavior (Moffitt, Caspi, Rutter, & Silva, 2001). Other research, too, has shown that although the developmental trajectories are broadly similar in males and females, a life course-persistent pattern is much more common in males (Fontaine, Carbonneau, Vitaro, Barker, & Tremblay, 2009). Nevertheless, there are some indications of sex differences in other research. For example, the severe empathy deficit associated with psychopathy in males is less evident in females (Dadds & Rhodes, 2009).

Similarly, country or ethnic group differences may limit the generalizability of findings (Rutter & Tienda, 2005). For example, Deater-Deckard, Dodge, Bates, and Pettit (1996) showed that the association between child aggression and physical discipline applies only to European-American children and not African-American children, while other studies have shown broader applicability in this association (Deater-Deckard et al., 2011). Understanding the generality and specificity of these relations is challenging because of the difficulties in disentangling cultural differences from relative poverty rates, differences in political power and exposure to discrimination, and other explanations for parent and family influences on disruptive behavior. For example, certain ethnic minority groups living in poverty have elevated rates of crime and violence while other groups do not (Morenoff, 2005; Pople & Smith, 2010). Also, there is evidence of cultural differences in how family dynamics influence risk among ethnic groups of similar economic status. For example, Gorman-Smith, Tolan, and Henry (1999) found that among inner-city U.S. Latino male adolescents, elevated emphasis on family closeness and responsibility was associated with risk for delinquency, while for African-American youth the opposite was the case. Parke and Buriel (1998) also describe the importance of viewing families as embedded within a variety of social systems and cultural traditions, including extended family ties, neighborhood norms and conditions, work experiences, and variations in access to and utility of educational, medical, social, and political systems. These multiple levels and wide array of potential influences on development of disruptive behavior disorders, as carefully described by Sameroff (1994), feed into a cumulative transactional process that also affects and is affected by environmental conditions to then affect subsequent development. As children advance along their developmental course, a coinciding developmental course of family priorities and tasks emerges as well. Thus, an adequate understanding of family influences must incorporate both child development and parent and family systems development. Further, all of these interrelated influences occur within secular trends and larger cultural and societal mores and social conditions. For example, the growing number of children growing up with only one biological parent may

alter how influential parenting is on child development as well as affect how the parent–child dyad influences individual tendencies of the child (Tolan, 2002).

What these complexities imply is that we must assume that, in most instances, the link between parenting and child behavior will reflect both a parent effect and a child effect, operating as part of an ongoing *transactional* process (Sameroff & Chandler, 1975). While described often and recognized by most, there is still limited incorporation of such principles into research design and interpretation of effects. Modeling such theorized multilevel multivariate growth relations can challenge current design and analytic capabilities (and many research budgets). Yet, there is value in pursuing work that is informed by this framework even if by necessity only focused on a piece of the overall processes of influence thought to be at work. Research should track and test a diversity of interdependent family influences and the emergence of problems over time (Forgatch & Patterson, 2010; Maccoby, 2000; Maccoby & Martin, 1983).

Diverse Family Influence Processes

Family influences may affect disruptive behavior and subsequent interventions through different psychological processes. Rothbaum and Weisz (1994), in a meta-analysis, reported that the relations of parenting approaches to behavior were additive; that is, they had stronger correlations when combined rather than individually. Grusec and Davidov (2010) argued that several parenting approaches can be differentiated and associated with different child outcomes, including responsiveness or sensitivity to the child's needs and communication; how protective the parent is of the child; level of controlling behavior; guided learning; and group participation or quality of the relationship (see also Maccoby, 2007). Parke, Burks, Carson, Neville, and Boyum (1994) theoretically distinguished three levels of parental influence: (1) parent as interactive partner with the child; (2) parent as direct instructor and manager of child behavior; and (3) parent as provider of developmental opportunities or shaper of context. This model augments another useful distinction by Darling and Steinberg (1993) between parenting style or emotional qualities of the parent–child relationship (Baumrind, 1991) and parenting practices or the methods and habits of parenting in the teaching, shaping, and managing of child development (Darling & Steinberg, 1993).

These and other formulations have wrestled with the distinction between basic and derivative parent influence processes (Tolan, Gorman-Smith, Huesmann, & Zelli, 1997), how processes overlap (Dunn, 2010), the importance of bidirectionality (Turiel, 2010), and whether supposed domain-specificity implies a modularity of effects (Gelman, 2010). However, with respect to disruptive behavior problems, these domains of family influence by no means exhaust the possible modes of influence or clarify the source of these influences (Rutter, 1989). For example, effects may be transmitted genetically, through perinatal environmental harm such as exposure to maternal alcohol or other substances (e.g., D'Onofrio et al., 2007; Lester,

LaGasse, & Seifer, 1998), or high levels of maternal stress (Davis & Sandman, 2010). They may stem from overt parental psychopathology (Eaves, Prom, & Silberg, 2010; Rutter, 1989) or from abuse or neglect (Jaffee et al., 2004; Kendler et al., 2000). Applying designs that can help differentiate forms of genetic and environmental influence is important to achieve clarification and greater certainty about the relations among including which are basic and which are derivative (Silberg, Maes, & Eaves, 2012). Similarly, while there is some scientific understanding that can be gained from studies that are limited to statistical adjustment for potentially confounded parenting processes of influence, these cannot determine causality or clear differentiation of relative primacy and derivative effects. Silberg, Maes, and Eaves (2010a, 2010b) point out that incorporating genetic influences in the research design is necessary to differentiate these possible modes of mediation and the need for designs that do not confound environmental effects with direct and indirect genetic influences (Silberg et al. 2012). This view can be extended to the challenge of differentiating multiple forms of parenting influence (Marceau & Neiderhiser, this volume).

Applying a Gene-Environment Interplay Perspective

There are many compelling reasons that family influences on disruptive behavior disorders have to be viewed through the lens of gene-environment interplay. The topic of gene-environment interplay framework and pertinent studies is considered in more detail in Chap. 2, but it is worthwhile to note critical features here. To begin, environments, through epigenetic effects, influence gene expression (Meaney, 2010); this transmission is crucially important because genes can be influential only if they are expressed. Environments may also become biologically embedded through other routes, as illustrated by the effects of maltreatment on immune mechanisms (Danese, Pariante, Caspi, Taylor, & Poulton, 2007). In addition, it has been shown that abuse and neglect have neuroendocrine effects (Gunnar & Donzella, 2002). It has still to be determined whether these could account for behavioral consequences.

Gene-environment interplay also involves gene-environment correlations (rGE; Kendler & Baker, 2007), implying that family features that index the rearing environment might also involve a degree of genetic mediation (Plomin & Bergeman, 1991). It is not, of course, that genes have effects on the environment. Rather, indirectly, via effects on proteins, the genes affect behavior, and the need is to study the processes involved in the effects of child behaviors on the environment; the extent to which such behaviors are genetically influenced is a secondary consideration.

Gene-environment interactions (G×E) are even more important (Dodge, 2009; Rutter, Moffitt, & Caspi, 2006). For example, Caspi et al. (2002) showed that a variant of the MAOA gene moderated the effect of child abuse on antisocial behavior. In the absence of the relevant genetic variant, even definite child abuse had a negligible risk effect for antisocial behavior. One implication is that some genetic effects

operate through influences on environmental susceptibility. There has been a tendency to consider the finding in terms of a genetic influence on vulnerability to adverse environments. However, evolutionary considerations suggest that it is more likely that the influence is on responsiveness to both good and bad environments (Belsky, 2005; Boyce & Ellis, 2005).

Parents can influence child risk through genetic and environmental transmission and as shared traits or tendencies or as products of between family members (Blaze, Iacono, & McGue, 2008; Dodge & Sherrill, 2007). Within these basic differentiations of influences, there are multiple processes that have been implicated as pertinent in family influences on disruptive behavior. For example, genetic liability because a parent manifests a substantial antisocial behavior is different from the genetic liability from parenting tendencies and/or child reactivity to such parenting (Rice et al., 2009). This influence also differs from liability deriving from genetic behavioral tendencies toward maladaptive reactions to environmental conditions or sensitivity to risky environments, including parenting practices in one's family (Rutter, 2010). Distinct from this risk (and protective) influence traceable to genetic similarity, socialization features of parenting practices and family relationship qualities are acting on child development in many forms, with the transactional development between tendency and experience accumulating into enhanced or dampened functional capabilities (Sameroff, 1994). Clearly, advances will be greatest when studies can better understand the relative contributions of these different components and the interrelations among them. One example is a multivariate twin design study examining the effect of parents and siblings' negativity toward the child (Pike, McGuire, Hetherington, Reiss, & Plomin, 1996). The study partialled genetic and environmental contributors. The findings showed that although genetics mediated a portion of the effect on the children's antisocial behavior, environmental effects as mediators were stronger. Use of this design with our more current understandings of key parent and family processes would be fruitful. Mills-Koonce et al. (2007) offer an example of how parent–child genetic interplay might inform child risk for antisocial behavior. They genotyped parent and child dopamine receptor D2 (DRD2) polymorphisms and sorted the sample into groups by the presence or absence of the risk polymorphism in mother or child. They then identified any relation between child behavior problems and parental sensitivity, which is thought to be related to DRD2. They found an allele thought to be related to lower parental sensitivity in children also was more common in their children. However, they also found that this pattern related to child evocation of less responsive and positive behavior in addition to explaining parental lower sensitivity. Notably, they did not find a relation between this pattern and harsh or negative parenting per se, but specifically to less sensitive responding by parents.

These many considerations create a picture of genetic liability and capabilities intertwined with environmental conditions. Some environmental conditions are truly exogenous, but others are created through the infant's interaction with the environment. These influences are affected further by ongoing, and not unrelated, parenting practices such as developmental and individual adjustment to child capabilities and needs, monitoring and predictable and consistent discipline methods,

within-family relationships such as emotional warmth or felt support and cohesion, and surrounding micro and macro systems such as economic and social resources, interpersonal networks, and life stress (Patrick, Snyder, Schrepferman, & Snyder, 2005). To adequately formulate how family and parenting in particular is related to disruptive disorders, research must incorporate this complex set of potential influences, which cannot be presumed to be simply reducible but are likely distinct, interdependent, and acting over time toward some stability of personality and behavior (Moffitt, 2005). This overall transactional process is also not simply so varied and individualistic as to negate the value in identifying key components and relative saliency of different forms of influence. Tremblay et al. (2004) have argued that the early years are most important because it is then that parents need to help children learn *not* to use physical aggression as a problem-solving strategy. However, important changes in different aspects of disruptive behavior occur later in childhood and adolescence, and it is implausible that family influences do not operate then as well. Similarly, Belsky, Steinberg, and Draper (1991) suggest that the first 5–7 years is when a child learns the expected predictability of resources in the environment, the trustworthiness of others, and an understanding of how enduring close relationships are formed. While later experiences, especially traumatic experiences, can shift these mental schemas, these early experiences persist in affecting risk for most children.

These considerations point to the value of tracking how interdependently and over time a cascading set of influences on disruptive behavior disorders develops. This information can then be formulated into theoretical models of differential risk and testable causal hypotheses (Dodge & Pettit, 2003; Patterson, Reid, & Dishion, 1992). This perspective implies that theories and related empirical tests will fall short if they are not formulated within an understanding that family influences are transactional, multilevel, and cumulative.

Organizing Parenting Influences

Within this broad and complex transactional developmental framework, numerous processes of influence can be identified, although much more work is needed to fully understand them. Five parenting practices emerge as most empirically supported and potentially useful as components in a multidimensional understanding of the influence of family processes on disruptive behavior disorders. The five are: (1) attachment relationships, (2) discipline methods, (3) monitoring of child safety and well-being, (4) warmth/hostility in the parent–child relationship, and (5) maintaining cohesion in the face of stress.

Attachment relationships. It is usual for children to develop multiple attachment relationships, although it is also usual for there to be an attachment hierarchy (Cassidy & Shaver, 2008). From a biological perspective, it is clearly adaptive for this to be the case in order to ensure that social development can continue normally even if the main caregiver dies. But this does not mean that benefit increases with a roster of changing caregivers or even a large number of caregivers. It does mean that

there is likely a primary attachment figure for most children but not a singular figure and meaningful attachment relationships are confined to no more than three or four attachment figures who are consistently present.

Understanding of the relation of attachment to disruptive behavior disorders involves five main issues. First, there is the question of long-term stability of assessments. Grossman, Grossman, and Waters (2005) brought together the findings of the studies extending from infancy into adult life and showed that attachment security in infancy constituted a very weak predictor of adult functioning, accounting for only some 5 % of the variance. By contrast, when combined with other social measures at somewhat later ages, social relationships constituted a powerful predictor of adult functioning, accounting for nearly half the total variance (Rutter, 2006). Second, there is the question of the differences in findings on stability of attachment relationships for low-risk samples and high-risk or clinical samples (DeKlyen & Greenberg, 2008). Stability has been found to be higher in the high-risk samples. However, this has also led to the finding that the main psychopathological risk derives from the combination of attachment insecurity, family adversity, and ineffective parenting. This risk relation for disruptive behavior, however, seems to be greater in boys than girls. Third, occurrence of disorganized attachment shows a stronger association with child psychopathology, as well as a stronger association with maltreatment and with institutional care (van Ijendoorn, Schuengel, & Bakermans-Kranenburg, 1999). Nevertheless, disorganized attachment occurs in some 15 % of children from low-risk samples, so that although it is involved with a probabilistic increased risk for psychopathology it is not strongly deterministic. Fourth, although attachment insecurity and disorganized attachment are associated with a moderately increased risk for psychopathology, this risk is diagnostically nonspecific (DeKlyen & Greenberg, 2008). It does not seem to be more associated with disruptive behavior than other maladaptive outcomes. Fifth, few of the studies of the association of attachment to disruptive behavior have been prospective and longitudinal so causal inference is necessarily uncertain. It may be the relation is transactional. For example, Kochanska, Barry, Aksan, and Boldt (2008) and Kochanska, Barry, Stellern, and O'Bleness (2009) produced empirical findings that were consistent with a bidirectional process in which the delineation of parental behaviors might be important in studying the pathways for early social relationship to disruptive behavior disorders (see Burke, Loeber, & Birmaher, 2002; Guttman-Steinmentz & Crowell, 2005 for a discussion of the interplay between attachment features, social context, and family stress).

Discipline methods. Given that a major feature of disruptive behavior problems is disobeying adult directives, a key interest among researchers is how discipline practices meant to shape behavior and curb aggression contribute to disruptive behavior (Barkin, Scheindlin, Ip, Richardson, & Finch, 2007). A central tenet has been that consistency in rules and expectations about behavior is important, as is a proportional response to misbehavior and compliance, such that more serious transgressions are treated differently from less serious transgressions. In addition, the methods of discipline are thought to be important, including use of physical punishment, psychological coercion, and/or positive and supportive comments to reinforce desired

behavior (vs. negative reinforcement or ignoring undesired behavior) (Dishion & McMahon, 1998). The coercive interaction model of Patterson et al. (1992) is the most influential. That model relates inconsistent rules and parental responses to child resistance to a pattern of subsequently fewer attempts at control by parents, which, paradoxically, promotes the misbehavior through negative reinforcement (Snyder, Cramer, Frank, & Patterson, 2005). Often this exchange is marked by abrupt and intrusive parenting that evokes child resistance and an emotionally charged exchange of parental imposition rather than corrective guidance, which has been labeled a "coercive exchange" (Patterson, 1997). Over time, the model has incorporated parents' hostile attribution about motivations of the child, and the child's proclivities toward noncompliance or aggression as a spur for greater parental control, which in turn can strain parental capabilities. For example, O'Connor, Deater-Deckard, Fulker, Rutter, and Plomin (1998) compared 38 adopted children with a genetic risk for antisocial behavior with 50 children with no risk. Parenting was consistently more likely to be negative when children were at genetic risk, but the stimulus for negative parenting behavior was the child's negative behavior.

This model has evolved to emphasize a transaction with multiple potential contributors and the need to consider child as well as parental attributes in attempts to alter the dysfunctional exchange. Also, as noted by Patterson (1997), parenting inconsistency can be expressed as variation in type of response (e.g. disinterested and then angrily disapproving) as well as level of response (mildly disapproving to very angrily disapproving). The inconsistency also can work through withdrawal of initial control efforts. Each inconsistency contributes to a likely increase or persistence of the undesired child behavior. For example, if parental substance abuse leads the parent to strongly react to a child's noncompliance while under the influence but leads to a tempered response when sober, the inconsistent response pattern, as well as any coercive cycle patterns, can reinforce the child misbehavior. In fact, Patterson and colleagues explicitly note that harshness can be conceptualized as having impact because it is expressed intermittently and so is inconsistency in response to misbehavior (Patterson et al., 1992).

The use of physical punishment has also been of great interest for its potential role in disruptive behavior. The relation has not been clearly determined in part because of variations in what is being measured. There has been an unfortunate tendency in the literature to treat corporal punishment and physical maltreatment as milder and more severe varieties of the same phenomenon. The study by Jaffee et al. (2004) showed this is mistaken. Maltreatment involved very little genetic liability and had a strongly adverse effect on the child. Moreover, any genetic liability was environmentally mediated. By contrast, corporal punishment had a substantial genetic component that seemed to indicate that it mainly arose as a response to the child's disruptive behavior rather than serving as a cause for it. In addition, the same study showed that a frequent recourse to corporal punishment was associated with an increased possibility of escalation to maltreatment. Thus, rather than being two parts of a continuum they are different in basis and how they arise in the transactions of development. They are associated, and given the particular association of increased escalation with frequent corporal punishment to maltreatment, it is clear that parental use of frequent corporal punishment is not advised. Even if giving the

impression of short-term effectiveness or actual suppression of child disruptive behavior, it is likely to have ill effects in the long term. Extreme physical abuse has been shown to relate to increased child aggression, although not simply in a linear and unidirectional fashion (Dodge, Bates, & Pettit, 1990; Lansford et al., 2005). In addition to these empirical findings that suggest the complex relation of corporal punishment and maltreatment and disruptive behavior, it seems to ignore the human rights concern in allowing serious physical punishment of children when that is illegal if done to adults or children other than one's own.

Within normal ranges of physical punishment, the correlation to disruptive behavior is still significant, but most of the relation can be explained by parents' response to child behavior rather than parental influence (Jaffee et al., 2004). The vast literature on physical punishment suggests generally that it exacerbates antisocial behavior when it is inconsistent and harsh, particularly if there is a time delay in the punishment (Nelson, Hart, Yang, Olsen, & Jin, 2006). The majority of studies in Gershoff's (2002) meta-analysis also found that physical punishment is correlated with less internalization of intended moral lessons, self-control, and empathic tendencies, especially when there is heightened emotion and limited communication about the reason for punishment (Gershoff, 2008).

Several studies have examined whether the adverse effect of physical discipline generalizes across cultural groups. In Western culture, parents' use of physical discipline connotes displeasure and disappointment with the child, which could lead the child to comply immediately but adopt a defensive and combative response that grows into antisocial behavioral patterns. Yet if the interpretation is different in other cultures, the impact might differ. In a U.S. sample, Deater-Deckard et al. (1996) showed that the effect of physical discipline on increasing aggression applied more strongly to European-American children than to African-American children, which the authors attributed to different cultural norms. In a further examination of this same sample, Pinderhughes, Dodge, Bates, Pettit, and Zelli (2000) found that African-American parents were more likely to use physical punishment. African-American parents were also more likely to make hostile attributions about the child's misbehavior and to fear that the child's misbehavior would lead to long-term problems. Thus, the use of physical punishment by African-American parents was warranted from their perspective and designed to prevent problem outcomes, and it was less strongly correlated with child antisocial behavior.

Lansford et al. (2005) tested the hypothesis that the relation between punishment and child antisocial behavior might vary across cultural groups in a study of parents and children in Italy, China, India, Kenya, Philippines, and Thailand. They found significantly different correlations across cultures. In those cultures in which physical punishment had higher base rates, its adverse effect was lower than in cultures in which punishment occurred more rarely. Nevertheless, although the effects of disciplinary practices vary according to the ways in which they were viewed in the particular culture, it cannot be assumed that practices that simply because a practice is acceptable in a culture, they are without risks. For example, female circumcision may be normative in some cultures. So far as we know, the effects on disruptive behavior have not been studied systematically but they clearly lead to harm through physical mutilation.

Others have linked more use of coercive parenting to economic stress and to lower socioeconomic status (Ceballo & McLoyd, 2002; Conger et al., 1992; Tolan, Gorman-Smith, & Henry, 2002), including tests that link increased economic stress to changes in parental coercion and subsequent child antisocial behavior (Conger et al., 2003; Schonberg & Shaw, 2007). Together these findings suggest that the harmful effects from coercive transactions are consistent across cultural and economic level groups, but that the saliency for disruptive behavior can vary by ethnic group and socioeconomic status.

Monitoring. The complement to discipline consistency and harshness is monitoring. Parental monitoring has been among the more consistent empirical correlates of disruptive behavior and in parenting programs is a frequent target for change (Dishion & McMahon, 1998). Originally conceived as parental attention to and knowledge about a child's behavior, social relations, and motivation in middle childhood and adolescence, the concept has been expanded to include safety, direct interaction, and attention to peer relationships (Dishion & McMahon, 1998). In the past 10 years, questions have been raised about the content validity of most measures of monitoring. Stattin and Kerr (2000) provided some of the most careful criticism of the concept as measured, noting considerable emphasis in measures on information provision by the youth to parents and/or reference to personal closeness between adolescent and parents. They also noted that once disclosure and youths' perceptions that they communicate well with their parents were controlled for, the "pure monitoring" items were no longer significantly related to youth delinquency. Other studies have shown similar patterns, but point to the need to augment monitoring with other parent–child relationship characteristics, such as positive or reinforcement parenting, better communication, and emotional warmth or receptivity during communication (Tolan, 2002). In addition, there are hints that the role of monitoring as narrowly defined is more important in childhood than either in infancy or adolescence. For example, Lahey, Van Hulle, D'Onofrio, Rodgers, and Waldman (2008) tested the Stattin and Kerr (2000) contention that most of what is considered monitoring is of adolescents' willingness to share with parents details about their lives. They found that while adolescent disclosure did explain parental knowledge about child activities and experiences in relation to delinquency risk, there was also an independent effect of parental limit setting or control through monitoring. This perspective is consistent with Fletcher, Steinberg, and Williams-Wheeler's research (2004) that found monitoring was dependent on knowledge and information sharing but also was related to felt warmth and control efforts by parents. This analysis showed that parental actions to monitor a child's whereabouts and to obtain knowledge of the child's activities consistently predicted less child antisocial behavior.

The concept of monitoring as a distinct parental effort changes significantly across the child's life course, yet many of the items used to assess monitoring do not reflect this changing meaning. For example, checking on a child's whereabouts outside the home is not meaningful for assessing monitoring of infants and young children. Yet by early adolescence it is central to the concept. For this reason, some have suggested

that monitoring should be reconceptualized to be a developmentally specific parenting role during the late elementary and middle school years. Another view is that measurement should be developmentally informed so that care to ensure safety and constant awareness in early infancy can be understood as developmentally appropriate monitoring, keeping the child within sound and site while playing is appropriate for preschoolers, and other efforts appropriate to early and later adolescence are used. The conceptual thread is an active understanding of a child's activities, views, and experiences when not with the parent. This then could be related to rather than confounded with communication quality (Stattin & Kerr, 2000).

Warmth. Parental warmth was included in Baumrind's (1971) seminal formulation of parenting's impact on child development. Parental warmth also pervades in studies of attachment, discipline methods, and caregiving; it is conceived as an "emotional tone" affecting these processes (Darling & Steinberg, 1993). As Darling and Steinberg (1993) noted, parental warmth overlaps with parenting practices but is usefully differentiated as a positive receptivity toward a child's needs and tendencies and a positive disposition toward the child (Deater-Deckard, 2000). Warmth has also been viewed as the absence or low rates of discipline methods that rely on threat, disparagement, rejection, or forms of emotionally abusive interpersonal orientations (Dodge, Pettit, & Bates, 1994). Thus, one can find warmth applied as a direct, overt parenting practice; an approach to parenting practices; or a subsuming characterization of more desirable parenting, with linkage to disruptive behavior documented for each conceptual base (Domitrovich & Bierman, 2001). Finally, warmth has been shown to be a "base" of security within the family relationship when youth face developmental challenges such as peer acceptance and social competency (Patterson, Cohn, & Kao, 1989; Steelman, Assel, Swank, Smith, & Landry, 2002). This pattern of findings may suggest that discipline methods and warmth might not only differentially affect risk but also vary in how genetic and environmental components contribute to risk.

More recently researchers have attempted to differentiate warmth within a multivariate model of parenting influence. Deater-Deckard, Ivy, and Petrill (2006) tested the role of warmth in moderating the relationship between physical punishment and child externalizing problems. Although use of physical discipline and child problems were moderately correlated, maternal warmth moderated the relation, such that the greater the warmth, the weaker the relationship between physical punishment and child problem behaviors. Warmth and discipline methods were also quite modestly related, suggesting that discipline practices and emotional warmth between parent and child are relatively independent. Both are therefore valuable in assessing the impact of parenting on disruptive behavior (Barkin et al., 2007).

Similarly, Stormshak, Bierman, McMahon, and Lengua (2000), in seeking to link coercive discipline practices to disruptive behavior in general, found a more specific link in low parental warmth. This study suggests that parent–child warmth may set the stage for when coercive parenting is most harmful. Feinberg, Button, Neiderhiser, Reiss, and Hetherington (2007) demonstrated genetic contributions to parental warmth (defined as closeness and rapport with the child) and negativity (defined as use of punitive and coercive parenting) depended on the child's behavior.

Feinberg et al. (2007) found that the effect of parental negativity on antisocial behavior strengthened as antisocial behavior increased, but the extent to which that negativity was due to genetic similarity was relatively lower at higher levels of antisocial behavior. Warmth, on the other hand, did not significantly moderate the genetic and shared environmental contribution to antisocial behavior. It did moderate the correlation for a non-shared environmental contribution.

Similarly, Tolan et al. (2002) compared discipline practices, monitoring, and parental warmth/harshness as mediators of parental partner violence on youth behavior. They found that each was significantly related in a multivariate model to youth violence. In addition, warmth and monitoring mediated the parental violence relation to youth violence, whereas disciplinary practices did not. This pattern of findings suggests potential differences in how varying parental practices transact with other family characteristics. In one informative study, Richmond and Stocker (2006) added to these interaction perspectives by documenting the unfolding transaction over time between parental warmth/hostility and child aggression. They found that those children who exhibited more aggression initially were more likely to evoke parental hostility, and that over time those with more hostile parents showed greater growth in disruptive disorders. Maternal hostility levels also differed by families and were related to overall child externalizing behaviors. The similarity in findings of these two studies points to the possible role of warmth as a distinct and important contributor to parenting influences on risk, albeit with more understanding needed about how these processes develop and interact over time to affect risk trajectories.

Family systems characteristics: Cohesion. A family systems focus moves from the dyadic parent–child level to the triadic and larger family set of relationships. It views the family as one, if not the, essential unit of interest (Cox & Paley, 1997; Tolan, 2002). For example, links between parental conflict and disruptive behavior are also well documented and were summarized succinctly in a recent systematic review by Rhoades (2008). In a meta-analysis of parental conflict and child problem behavior (internalizing and externalizing), she noted that it was exposure to the between parent conflict that was related to externalizing behavior, whereas rumination about parental conflict was only related to internalizing behavior. Rhoades (2008) argues that parental problems affect children through decreasing security and increasing affective and cognitive stress as well as a lessening of soothing parental responses or those that promote self-control. Notably she suggests focusing interventions on lessening exposure to overt conflict but also on child cognitive, affective, and physiological reactions that could lessen the harmful impact of conflict.

Among the many key constructs of family systems, lack of cohesion in the family has emerged as one with more empirical support as associated with risk for disruptive behavior disorder, including evidence that it is changes in cohesion that mediates the effects of some family intervention programs on disruptive behavior (Henggeler, Melton, & Smith, 1992; Tolan, Gorman-Smith, & Henry, 2004). Cohesion can be defined as an ability to maintain an emotional connection among family members in the face of stress and conflict (Sturge-Apple, Davies, &

Cummings, 2010). In addition, cohesion is thought to involve more positive and cordial family relationships, which promote well-being and constructive problem-solving during moments of conflict. Family cohesion measured at preschool predicted increased prosocial interactions with peers during middle childhood (Leary & Katz, 2004). Lack of cohesion, on the other hand, has been linked to behavior problems in middle childhood and preadolescence (Kerig, 1995; Lindahl, 1998). Studies have also identified cohesion as a mediator of family stress on risk (El-Sheikh & Buckhalt, 2003; Lindahl, Malik, Kaczynski, & Simons, 2004; Vandewater & Lansford, 2005). For example, Sturge-Apple et al. (2010) found that children in the least cohesive families had the highest average number of problems and increasing problems over time.

Cohesion may be particularly relevant to disruptive behavior disorders (Fosco & Grych, 2008). The insecurity and lack of positive family engagement that constitutes low cohesion may have a particularly precipitant role in how aggressive tendencies develop toward disruptive behavior disorders. Richmond and Stocker (2006) reported that low cohesion explained adolescent externalizing behavior even when parent–child hostility was taken into account, and it added to the explanatory power of each child's behavior within a family and between family differences. Multilevel modeling indicated an independent, significant relationship of low cohesion and externalizing problems in addition to parent–child hostility, consistent with the view that parenting effects occur within overall family relationship qualities (Jenkins, Rasbash, & O'Connor, 2003; Tolan et al., 2003).

Family cohesion also was found to moderate the relation between testosterone and disruptive behavior in adolescents. Under conditions of low family cohesion, free testosterone was positively associated with disruptive behaviors among boys, whereas in families with high cohesion no association was observed. In contrast, free testosterone was negatively associated with disruptive behaviors among girls in low-cohesion families (Fang et al., 2009). This study also illustrates the interplay of a possible genetic predisposition and family system characteristics in affecting risk for disruptive behavior disorders.

Advancing Knowledge About Family Influence Processes

This summary of the field's understanding of the relations between parenting and family characteristics and disruptive behavior disorders reveals many critical considerations moving forward. Perhaps most fundamental is the need for research with design qualities that can permit discrimination of the various forms of genetic and environmental family influences and clarification of which processes have a basic role from those that function more as augmenting of primary influences and from those that are derivative or provide no additional explanation once other correlated processes are considered (Marceau & Neiderhiser, this volume; Rutter, 2012; Silberg et al., 2012). At the same time, theoretical clarifications and elaborations that locate causal understanding with a developmental framework that can consider

variations in timing of effect, immediacy of evidence of effects and relative permanence of effects and that relate different levels of ecological influences and can incorporate a transactional process will be very important in advancing what is best to study in descriptive and causal studies.

One important area of limited study to date is the simultaneous effect of multiple family influence processes. A meta-analysis points to similar effects for multiple processes whether in discriminating between disorder features (Rothbaum & Weisz, 1994; Wakschlag et al., 2010) or interventions (Eyberg, Nelson, & Boggs, 2008; Wyatt, Valle, Filene, & Boyle, 2008). For example, Wyatt et al. (2008) found that teaching parents to use time outs and the importance of parenting consistency resulted in consistently larger effects than interventions teaching parents problem-solving skills or how to promote children's cognitive, academic, or social skills. However, they also note that most programs involve multiple target processes, often without specifying which aspects are meant to affect which skills or how an effect on one process might relate to an effect on another. Thus, disentangling the importance of various parenting and family processes in multivariate studies is needed. The task of partialling the unique importance of each parenting strategy could include various research approaches, including mediational analyses to model differential influence (Baron & Kenny, 1986; MacKinnon, Fairchild, & Fritz, 2007). In such an approach, mediation is used not only to test for expected intermediaries between intervention exposure and change in target behavior, but also to test processes not thought to the intermediaries, to show that they do not mediate outcomes (MacKinnon et al., 2007).

Sorting contributors to intervention effects on parenting practice and family relationship influences will also be facilitated by advances that permit more sensitive and more complex mediational analyses. This research should include cross-level mediation, moderated mediation, and multiple mediators, with recognition that partial mediation is more likely than full mediation (see Fairchild & MacKinnon, 2009; MacKinnon et al., 2007; Rutter & Sonuga-Barke, 2010). However, Kazdin (2007) has pointed out that more than a single statistical model is needed to test for mediation. The starting point is the same, namely, the identification of a theoretically sound and empirically supported mediator and ruling out alternative processes, but five more steps are required. As Kazdin notes, consistency across replication; experimental tests that manipulate the mediator to determine the effects on child outcome; the establishment of a time line between the mediating and mediated effects; determination of a gradient of dose effects; and establishment of the plausibility of mediation in terms of a broader evidence base (including biological studies in humans and the use of animal models). Formulating a model and a proper sampling for parental and family processes is, in other words, rather daunting. As far as we know, there are no published examples using all six steps, but the recommendation is sound and there are examples in which some of the steps have been used to test mediation. While ultimately it is experimental manipulation of the theorized mechanism that is needed, such statistical methods can provide important direction about important parenting and family processes. This approach can help promote refinement of interventions and can also suggest valuable emphases for subsequent

research to help sort various forms of genetic and environmental influence and to suggest the components of transactional models of development of disruptive behavior.

Researchers can also use a range of natural experiments to test the causal pathways of influence (see Rutter, 2007, 2012). Studies could focus on the possibility of genetic mediation of parenting practices or effects using twin, adoptee, and other strategies that create a quasi-control and experimental group. An example is the use of assisted reproductive technologies (ART) (see Rice et al., 2009; Thapar et al., 2009). Some varieties of ART involve genetic liabilities shared between mother and child (as with donated sperm) and others do not (as with donated eggs). This strategy showed, for example, that it was unlikely that maternal smoking during pregnancy contributed to an increased risk of antisocial behavior or ADHD among children. Sibling comparisons (between offspring exposed to maternal smoking in pregnancy and those not) led to the same conclusion (D'Onofrio et al., 2008; Obel et al., 2011).

The Next Generation of Research on Parenting

Even though a literature review reveals a great deal about the processes, impact, and antecedents of parenting behavior, much is still to be learned. Furthermore, new developments in our understanding and measurement of genetics are leading to evolving frameworks for understanding influence. Additionally, the rapid shift and extent of impact of information technology on children's daily lives and children's exposure to new cultures may be fundamentally shifting how parenting and related family characteristics influence child development, including risk for disruptive behavior disorder. The final section of this chapter identifies six issues facing the next generation of research on parenting. This list is not meant to exhaust the six most critical issues, but identify issues that are, in addition to advances in technology and methods of science and results from specific studies, important considerations for research aiming to improve understanding about the relation of parenting and associated family characteristics to disruptive behaviors.

Direction Bias in Sampling and Designs

One of the important challenges is the problem of directional bias in parenting research studies. As first noted by Bell in 1968, the alternative hypothesis to the claim that parenting contributes to child behavior problems is that child behavior elicits particular parenting behaviors. Although longitudinal studies restrict correlations to temporally precedent ones, most theories of parent–child relationships suggest reciprocal relations over time, which acknowledge selection biases as at least partial explanations. Furthermore, advances in heritability

studies suggest that genetic factors that might underlie many child behavior endophenotypes, such as impulsivity, might also underlie parenting behaviors, such as inconsistent harsh discipline. In the 45 years since Bell's re-interpretation, this challenge has not been conclusively surmounted. As advances in specific knowledge about gene processes and heritability of more specific parenting and child behaviors advance, and more refined statistical tools suggest more promising foci of research, particularly sampling that is organized to permit better differentiation of genetic and environmental influences, the critical features of the parent–child transactional relationship should become better understood. Thus, designs that can control for or minimize confounding of different forms of parenting and do not bias directionality of influence are critical for advancing knowledge (Silberg et al., 2012).

Differences Across the Life Course

Just as child behaviors change across the life course, so, too, do the tasks of parenting change. During infancy, the major tasks are to provide for the infant's survival through food and warmth and to provide a secure attachment for the infant's comfort. During the toddler years, the task of parenting shifts to providing consistent responses to misbehavior so that the child learns which behaviors are acceptable in a social world and which are to be avoided. During early adolescence, when the child naturally explores peer groups and seeks new experiences which may include risks such as substance use, a parent's task moves to monitoring the child's whereabouts, supervising activities, and limiting access to harmful environments (such as exposure to alcohol and substances).

For scholars of developmental psychopathology, an important question to pursue focus in this line of knowledge development is the differing impact of parenting behaviors on a child's development at different ages (and the child's behavior impact on parenting behaviors), particularly how transactional impact may vary as a function of age and related needed parenting. Surprisingly little is known of these contours, however. Too many empirical findings are presumed applicable across ages, which future research should rectify. For example, the meaning of corporal punishment likely changes as the child gets older and begins to understand whether a parent's behavior is deviant by cultural standards, yet we do not know whether this parenting style has different effects at different stages of development nor how that might depend on child understanding of the style as deviant or atypical. In the next generation of research we should work to better understand how the multiple effects of a given parenting behavior pattern vary across development. In doing so, it seems important to consider that effects, bidirectional or unidirectional, can be immediate or delayed and temporary or long lasting. It is possible, for example, that corporal punishment in early years can evince immediate and temporary compliance by the child but leave residual ill effects on identity and sense of competence over the long term.

Fathers and New Family Configurations

Although Parke (1996) lamented years ago that not enough is known about fathers' effects on child development nor is this given adequate attention in research, this gap in knowledge continues today. The particularity and the additive role of fathers takes on new meaning given the growing proportion of child births to single mothers, the increased divorce rates, and the lesser but emerging rates of single fathers. Some studies have begun measuring fathering influence cognizant of the similarity of fathering to mothering but also that there are meaningful distinctions (see DeGarmo, 2010 for one such example). Fathers remain involved in a child's life even when not living with the child. How these different living arrangements affect fathering and alter the impact of father behavior on child disruptive behavior disorders is not yet clear. With growing independence between mothers and fathers comes the potential for more independent parenting styles and family rules. Therefore, it will be important to learn more about how mothering and fathering interact in non-intact families. Consistency between parents would seem to be important in mitigating child disruptive behavior, although it is plausible that one parent's warmth could protect a child from the adverse impact of the other parent's harshness, and the growing ease of independence could mean greater hope for a child to become free from the ill effects of one problematic parent (DeGarmo, 2010).

These shifts in family configurations touch all demographic groups and across societies. About 40 % of births in the United States are to single mothers, with figures above 50 % for Western European and Scandinavian countries. Furthermore, couples increasingly delay marriage even after child-bearing and living together (Gibson-Davis, 2009). As single-parent families and other forms that blur the distinction between ascribed gender-based parenting roles reach levels of commonality, it is likely there will be shifts in not only what is culturally normative, but how family structure and risk are to be understood. At a more basic level, scholars are challenged to better organize measurement of fathering, father influence, and describe how, why, and under what circumstances single-parenthood, divorce, nonmarried parents, and other forms of family structure alters a child's risk for disruptive behavior.

Parenting in Context

Findings that parenting affects child disruptive behavior in different ways at different ages in different family configurations point to a broader need to understand parenting in context. While consideration of cultural, ethnic, and national norms has only recently been incorporated into developmental studies, there is evidence that contextual variables can play an important role in how parenting and child disruptive behavior relate. This contextual moderation might whether a given practice is culturally normative context (Lansford et al., 2005) or it might be the access to extended family and others to provide emotional and instrumental aid for the

parent–child relationship (McLoyd & Smith, 2002). Perhaps one of the most significant findings over the past decade is that while there seems to be some constancy in parenting impact, there is also considerable variation depending on context. Equally significant is how these findings reveal the subtlety and complexity of contextual influences. For example, there is much need for studies that examine how microsystem and mesosystem influences can facilitate parenting, particularly of children with risk or early evidence of disruptive behavior. How important is access to extended family or neighborhood resources? Similarly, there is need for more extensive and carefully formulated cross-cultural comparisons of the relative roles of key parenting processes identified in this chapter. We do not know yet *how* parenting is affected and its impact on and from child behavior depends on more micro and more macro contextual characteristics. The field needs to incorporate thoughtful and specific formulations of context into framing of research, just as there is need to incorporate genetic and nongenetic processes in such framing.

In addition to building on work conducted to date that describes potential roles of context and suggest variations in patterns, we suggest attention to three ways in which context can be important. First, context alters which parenting styles are possible or at least plausible. For example, even if past findings might suggest that infants are better off if a parent stays at home full time, this may not be feasible for many families in American society and elsewhere. Financial demands and increased valuing of work outside the home for each parent seem to make this less feasible. Similarly, if raising children in a violent and economically deprived community, it may be that parenting that promotes child exploration and opportunity to learn through experience is not viable; it may carry serious and lasting harm to the child (e.g. through eating lead on windowsills in substandard housing or through potential injury if playing near an area where gunfire occurs). Both of these examples suggest that parenting research will be well served by examining how parenting occurs in common context and varies in plausibility across contexts. It might be that under different cultural and economic constraints, the optimal parenting style changes.

Second, context alters the meaning of parenting behaviors. The impact of a parent's behaviors on a child cannot be reduced to a schedule of rewards and punishments that reinforce certain antisocial and prosocial behaviors. Culture and ethnic group meaning ascribed to family engagement, deference, and respect varies, and this variation may have influence on how parenting and associated family characteristics relate to risk. For example, greater family involvement was positively related to delinquency among Latino males growing up in inner-city communities, whereas it was negatively related for African-American males from similar communities (Gorman-Smith, Tolan, & Herry, 1999). Similarly, how common and appropriate a given parenting practice is seems to affect how it is related to disruptive behavior (Lansford et al., 2005). At the microsystem level, there can be variations in meaning attached to a given behavior. As parents of teenagers often experience, sometimes praising a child for a certain behavior in front of his or her peers *reduces* that child's desire to continue that very behavior. Each of these examples illustrates that context influence on meaning is an important consideration for future research. Among the key topics will be the relation of meaning variation to parenting practice use and

whether there is considerable or limited covariation in these by context. That is, if meaning variation is considered, is the explanatory value of practice accounted for (or vice versa)? Thus, studies of observed parenting behaviors and their effects on child outcomes are likely to yield inconsistent findings if the broader context is not described, measured, and taken into account as a moderating influence.

Third, new information technology is creating new contexts in which parent–child relations are being influenced (internet access, instant communication), as well as likely having impact on parent–child relations, including risk for disruptive behavior disorders. Past studies have shown that monitoring and supervision of adolescents are crucial factors in protecting them against antisocial behavior, and even critiques of this research point to communication between adolescent and parent as the alternative explanation. However, the methods available for monitoring and the immediacy of ability are evolving. Video and GPS monitors cannot be installed in vehicles to help parents track with certainty child driving practices. Cell phone records, internet postings, and other methods of more direct understanding of child behavior are now readily accessible and used with greater frequency. At the same time, such media provide opportunities for broader social engagement and exposure that may well shift how central parent–child relationships, particularly for older children. Further, access to on-line information and support may provide parents with aid, reminders, and social connection, even if physically isolated when stressed about parenting. While the potential impact of these and other aspects of the new electronic contexts is still being grasped, it is evident that consideration of these as contexts of and potential influences on parent–child relationships, including risk for disruptive behavior, warrants substantial attention.

While there are likely other aspects of context that are important for future research, these three seem to be valuable in a more elaborate and useful understanding of the role(s) of context in understanding family influences on child disruptive behavior risk. In general as well as for utility for those interested in this relation, a critical task of the next generation of research will be to provide systematic theoretical organization for study of contexts and thorough description of context considerations in which parenting-child behavior linkage is studied, so that critical features can be discovered. Accompanying digging into the multiple aspects of genetic influence and various relations between gene and environmental influences, research to better capture theorized pertinent aspects of context is essential.

Parenting Interventions

The final innovation in the next generation of research will emerge from interventions to change parent behaviors. Although some of clinical psychology's greatest successes have come from parenting interventions (Patterson et al., 1992; Tolan, 2002), the utility of experimental manipulations of parenting influence on child behavior to advance knowledge can be much greater. For example, design of

interventions that are more specifically tied to a gene-environment modeling of risk, that have procedures more directly and specifically formulated as expression of a causal theory, and measurement regimen that permits more thorough testing of the processes of effects and variations in effects by participant characteristics are all likely to expedite and deepen understanding for more effective interventions, but also about causes of disruptive behavior (Tolan & Gorman-Smith, 2002).

In addition, the intervention design and research field will likely change rapidly with the ability to utilize new methods and more interactive technology for communication between clinicians and parents, for improved data-gathering, and for incorporation of technologies into interventions. For example, parents will be able to more reliably and validly complete daily diaries of their behavior and the child's response through electronic entry on smart phones and similar devices, "push" technologies can prompt parents to implement specific parenting strategies, and synchronized reporting from cell phones can provide simultaneous data on the perspectives of parents and children. Internet resources including libraries of modeling of effective parenting, personal stress management, or support systems may augment or even fundamentally shift how preventive and treatment of disruptive behavior disorders through parent focus occurs. This can occur through resources for parents and for adolescents, but also in helping providers to provide more effective methods with greater fidelity.

Thus, we can expect novel parenting intervention technologies, engaging interventions with potential for prescriptive organization dependent on parent and child needs, substantially more data more easily accumulated, collated, and utilized, and new technology as part of parenting and parent training and intervention. Whether these innovations lead to greater intervention efficacy and serve to expedite scientific understanding of the role of family influences in disruptive behavior is to be seen. We expect so, but we offer a caveat. Like many other consequences of twenty-first century technology, we suspect that the emphasis will move toward immediacy; immediacy in focus and in utility. There will be increased opportunity for immediacy of parent interventions and increased emphasis on immediate impact on the child. However, as was noted at the outset of this chapter and is abundantly evident in the vast literature on parenting influences on child development, the effects of a given potential influence are not simply determined and easily disentangled from other co-occurring influences, with important critical and fundamental aspects of genetic and environmental forms of influence still to be discovered and fully understood. Also, parenting occurs across a life course of many years, and the impacts of parenting are both direct and indirect, immediate and deferred. A challenge for the next generation of scholarship will be to figure out how to incorporate the challenges of consideration of multiple genetic and environmental influences, context as an important consideration, the shifting patterns of family organization, and the best use of new technologies and advances in methodology to understand the complete impact of parenting behavior on child development and the optimal interventions that parents can employ in a rapidly changing cultural context.

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Chapter 8
Advancing Our Understanding and Interventions for Disruptive Behavior Disorders

Patrick H. Tolan and Bennett L. Leventhal

This volume has served as a focused review and discussion of key issues in advancing our understanding of Disruptive Behavior Disorders (DBDs), particularly Conduct Disorder (CD) and Oppositional Defiant Disorder (ODD). The October 2008 symposium on which this book is based asked senior scientists to report on a single topic. That presentation and the ensuing discussions are reflected in the chapters in this volume. Each chapter summarized current knowledge about a key topic, and together they provide an understanding of patterns of occurrence of DBD; the likely contributing influences on its emergence, expression, and course; and important avenues for treatment, prevention, and eventual curative efforts. The chapters in this volume also have identified many exciting and important areas for investigation that could substantially advance our knowledge and improve our ability to identify, treat, and ultimately prevent these serious problems of childhood. Here we summarize the overall themes and implications emerging in the areas considered during the symposium.

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Disruptive Behavior Disorders Are Prevalent and Have Serious Harmful Impact

As noted in the introduction to this volume, DBDs are among the most common brain-functioning problems affecting children and adolescents. In many epidemiological studies, they are the most prevalent DSM diagnosis (National Research Council and Institute of Medicine, 2001). The harmful impact of these disorders begins early in life, often as early as toddlerhood, and extends well beyond childhood (Dodge, Coie, & Lynam, 2006). Continued aggression and conflict can contribute to school failure, marital instability, employment conflicts, and criminal involvement or create risk factors for other mental illness patterns and other life problems (Offord, Boyle, & Racine, 1991). It is estimated that, if untreated, each child with a conduct disorder will eventually impose a cost to society of $3 million, excluding lost productivity. Much of that cost is attributable to criminal justice and special education costs related to the problem behavior (Cohen & Piquero, 2009). Clearly, these disorders rank among the most serious and costly health problems, and they warrant full scientific attention in understanding the causes, best management practices, and prevention.

The best scientific knowledge points to a complex picture, with multiple patterns of manifestation, substantial heterogeneity among those with the same diagnosis, multiple risk factors with varying levels of empirical evidence about causes, and yet to be demonstrated efficacy and utility of interventions to prevent or treat the disorders (Loeber et al., 1993). It seems evident, on the one hand, that current diagnostic categories and criteria are of limited utility and should yield to those with more specificity and greater differentiation in presentation features, developmental pathways, and etiological bases. On the other hand, DBD is one of the most studied mental illnesses, with some of the most sophisticated and robust results. Thus, although the need for continued advancement remains, the research also represents a model for developmental psychopathology research and practice. DBD is a prime example of the developmental psychopathology principles of equi-finality (multiple causes leading to the same manifestation) and multi-finality (the same risk factor implicated in multiple disorders). Like many child mental disorders, DBD is not identifiable with certainty by pathognomonic symptoms or risk markers, nor do the required criteria for identification provide much certainty about likely seriousness, course, or treatment need and responsiveness. There is thus an urgent need for systematic attention to the topics in this volume. This work can also inform work on other disorders that are less extensively studied.

Descriptive and Experimental Efforts Must Share a Common Developmental-Ecological Framework

As noted in several chapters in this volume, a primary issue is whether variations in risk patterns (population distribution) and in symptom configurations represent different manifestations of the same disorder (a single type of DBD) or are indicative

of different disorders. Determining this is constrained by the lack of epidemiological studies of scope and depth necessary and by conceptual limitations within the current nosology as well as controversies about what alternative markers, configurations, and classification should be applied.

In addition, there is need for clinical studies that can identify distinct disorders or meaningful variations and describe the related variations in developmental pattern, presentation, reaction to interventions, and etiological features. Among the richest avenues of investigation would be careful and in-depth assessment to tie epidemiological pattern analysis to multiple risk factors and potentially important differences in symptom presentation and configuration. Several chapters have suggested next steps for such work and pointed to the value of connecting such efforts so as to relate the findings across these efforts.

This primary concern is not new. In fact, it might be said that moving beyond gross distinctions in identifying DBDs and their impact has eluded research and clinical theorists for decades. Multiple descriptive studies have focused on what constitutes DBD, and several longitudinal, developmental-ecological studies have traced the contributors to DBD (see Carter, Gray, Baillargeon, & Wakschlag, 2013; Dodge et al., 2006; Frick, Blair, & Castellanos, 2013; Loeber, Capaldi, & Costello, 2013 this volume for summaries). In addition, numerous experiments have attempted to isolate causal factors, specify genetic and other predisposing conditions, and provide animal models of mechanisms theorized to cause DBD (see, e.g., Dishion & McMahon, 1998). Yet each advance adds additional complexity, subtlety in relationships among influences, and more refined but not simpler explanations.

The most robust contribution is in the value of tracing how individual characteristics at multiple levels (e.g., genetic or neurophysiological) intersect with and transact with multiple contextual influences, such as parent and family characteristics, peer relationships, and community conditions. This interaction contributes to increasingly differentiated and determined developmental trajectories, characterized by variation in manifestation and functional impact. Nonetheless, instability and uncertainty remain more often the case for any given individual. While we are beginning to unravel the different contributors and their relationships, the field still needs to trace, in a sophisticated way, child developmental patterns and influences in a way that recognizes the intersection of long-term and immediate contextual influences with ongoing individual development. This should become a necessary perspective, not just a complex theoretical formulation that is put aside for simpler scientific studies. The past 15 years has provided the ability to probe more fully the neurophysiological and neuropsychological processes in DBD that were long considered explanatory. As a result, we have begun to tie together those processes to genetic characteristics and processes.

This volume represents one of the first attempts to apply this developmental-ecological approach to viewing DBDs within a brain functioning framework. One implication drawn from this volume is the importance of a shared framework that guides work from the most molecular level to the most molar. Another is the importance of articulating the connection of work deeply focused within one topic or method to other work within this model. While this may require more complex organization of research and more close reconciliation with work in diverse areas, a

shared larger frame is also mostly likely to efficiently build knowledge that can guide intervention, determine etiology, and provide sounder practices. Thus, while each chapter focused on one question/subtopic each is marked by being a summary of complex findings and by dependence on knowledge in other areas of research on DBD for best understanding. In addition, to summarizing and noting key topics, therefore, each chapter identified needed research as a basis for connecting work across areas in a bio-psycho-social model.

More Sophisticated Identification of Disruptive Behavior Disorders

A prerequisite for establishing a sound scientific understanding of the etiology and interventions for DBDs must have accurate specification of the phenotype as one element of its foundation. Therefore, one important area for continued work is specifying our understanding of the signs and symptoms that constitute DBD. What are the necessary and sufficient behaviors and personal experiences to form the various types of DBD? What differentiates DBD from typical developmental struggles and challenges and from less than optimal but not pathological functioning? Are there specific biological or neuropsychological markers? What differentiates DBD from other conditions that share at least some of the signs and symptoms of DBDs? And, as we progress to identify empirically sub-groupings or different types of DBD, how should these be differentiated from each other?

It is evident from the accumulated studies, and as noted in several of the chapters in this volume, the current diagnostic categories of ODD and conduct disorder do not correspond well to specific behavioral patterns seen in DBDs and fall substantially short in terms of their developmental sensitivity (see Wakschlag, Leventhal, Thomas, & Pine, 2005; Wakschlag, Tolan, & Leventhal, 2010 for specific discussion of these limitations). Carter et al. (2013) "deconstruct" the key clinical features of child behavior that are designated to be indicative of a DBD; they suggest that four dimensions constitute the framework for DBDs: (1) Reliance on aggression; (2) Noncompliance; (3) Ease of angering; and, (4) Limited concern for others. While they are cautious to note that this may not be the only way to organize the DBD behavioral phenotype, this does consider multiple dimensions that are both consistent within current diagnostic systems and empirically differentiate DBD children from nonclinical populations. The chapter, and related papers, provide the link to an empirical basis for the suggested dimensions, how these dimensions differentiate DBDs from other disorders, theoretical and some empirical relation of such manifestations to developmental atypicality, and how each relates potentially to differences in the neurodevelopmental features of DBDs. This work provides a model for clinical descriptive work that ties identified variations in presentation back to their neurodevelopmental underpinnings in order to differentiate the developmental patterns of manifestations of DBDs, and suggest the bases for the different clinical courses over childhood. Perhaps the most promising suggestion for reframing the construct of DBD, as highlighted by Carter et al.'s chapter, is that this

dimensional approach and its empirical yield may help differentiate treatment needs among those with DBD.

In their chapter, Frick, Blair, and Castellanos describe an approach to DBDs that is not focused on the topography of DBD behavioral symptoms. Instead, they describe their particular efforts and a larger literature that is part of a long-time interest in understanding the motivation for aggression as a way of differentiating DBD subgroups. Frick and colleagues focused on whether youth with DBD can be characterized by low concern for others and limited emotional arousal during aggression (labeled callous-unemotional) (see Frick & White, 2008 for a review). They outline a systematic approach for tracing the suspected childhood precursors of adult psychopathy, utilizing risk studies, intervention effect variations, neurodevelopmental studies, and clinical experimental studies, to argue that callous-unemotional aggression constitutes a distinct form of DBD from that motivated by impulsive reaction or desire to remove perceived threats. The interest is tied to a vibrant strain of research on child aggression showing that motivation for aggression can be differentiated between that which is reactive or impulsive from that which is instrumental or planned with intention to hurt the other (Dodge et al., 2006). The latter often is accompanied by lower concern for or recognition of the potential harm to others. In addition, Frick et al. (2013) relate these findings to the current dominant perspective of differentiating those with life-course persistent patterns of aggression from in whom aggression is limited to adolescence (Moffitt, 1993). Tests of this theory have yielded mixed results, or at least more complex sets of patterns that will require more than a timing of onset distinction. The approach suggested by Frick et al. may provide an additional discriminating feature that can explain the not insubstantial patterns of early onset DBDs that stop of their own accord and later starters who graduate to serious aggression with persistent involvement (Dishion & McMahon, 1998; Frick & White, 2008). They suggest callous-unemotional DBDs are more likely to be chronic.

One of the remaining challenges for this track of work is how *seriousness* of aggression, defined as potential for physical harm to a victim, and *level of* aggression, defined as the sheer amount or frequency of aggressive acts, relates to likelihood of callous-unemotional motivation. As with the distinction between reactive and proactive aggression, there is a relation between such motivation and the seriousness of the aggression. The link and the boundary between motivational difference and action difference may be important in testing the robustness of this distinction but also its utility for risk identification beyond merely measuring the seriousness and frequency of aggression. At least potentially, one should not label traits as callous-unemotional merely by the seriousness of acts or the extent of such behavior.

Among the impressive set of findings are that those individuals with callous-unemotional traits respond less favorably to parenting intervention programs than do those without such features. This may explain why even though parenting programs have been the most consistent success in treatment studies, and often have the largest effect sizes for DBD interventions, a substantial portion of individuals do not respond to the intervention. Disentangling the motivational difference from the seriousness of the behavior problems will be important in building on these findings

particularly in terms of directing changes in who is targeted for each particular intervention or how modifications in the current interventions will be needed for specific subpopulations.

Given the state of the current work in DBDs, it is not surprising that two chapters in this volume which have a descriptive focus come to different formulations for the number and nature of the key dimensions to be considered the essential elements of DBDs. This suggests that we have only seen the earliest efforts at probing alternative models of the features necessary to define a DBD. From this work, it appears that potentially rich area for research will be the reconciliation of the findings of each of these seemingly different approaches. For example, how might callous-unemotional behavior look in preschoolers and is it manifest as a developmental immaturity or as personal motivation differences already present in preschoolers? And, how might motivation for aggression enrich any multidimensional formulation of DBD sub-groupings? There seems to be considerable similarity in "callous unemotional" and "low concern for others and between easily angered/aroused." And, both seem quite distinct from "low emotionality." With increasing ability to conduct sensitive neuropsychological challenges and track within-brain communication, it may be that motivation and behavioral presentation links will be evident by differences in brain function. One can only imagine the possibilities of exploring. It may well be that some combination of the focus on clinical presentation of Carter et al. and that of motivation for behavior of Frick et al. will be integrated into a multilevel formulation of brain functioning, behavioral characteristics, and motivational features.

Developing More Sophisticated Models of Development of DBD

The need for more specific and elaborate models of what constitutes DBD and differentiates subtypes builds on and will feed back to studies of patterns of occurrence and relation of patterns to risk factors. In addition, these studies can provide focus for developmental longitudinal and laboratory studies to help explicate the major correlates and ultimately the causal influences on DBDs, in each of the various forms likely to be identified. Within the overall framework of an ecology of influences on development and of the brain processes involved in major symptoms, there is need to understand the relative contribution of the avenues of influence that Tolan, Rutter, and Dodge identified in their chapter. In addition, there is need to understand how peer relations, social engagement in general, and neighborhood and community features can help explain the likelihood of DBD, its manifestation, and likely course (Horney, Tolan, & Weisburd, in press).

Substantive and methodological scientific challenges remain that are essential for advancing brain-child development-DBD connections. To address these, investigators must reconcile complex models of genetic contribution that Marceau and Neiderhiser described in their chapter, framing development as contextual but informed by and informing a genetic perspective tied through brain functioning

(including regulation as outlined by Susman and Pollak in this volume). Marceau and Neiderhiser (2013) provide some mapping to guide such pursuits. They suggest that by using a genetic framework, one can build sampling and measurement models that address questions about shared behavioral patterns as well as the genetic influences on them. By plan fully differentiating what are thought to be passive, additive, interactive, and multi-genetic influences and then articulating the specific relations between the genetics and developmental expression at different points in the developmental pathway, our explanatory studies can start to trace and describe rich correlates and ultimately differentiate the causal factors for DBD. Despite the optimistic tone, Marceau and Neiderhiser note precautions about the vexing problems of confounding, corresponding, and correlative relations between identified gene variations and other potential influences. As they note, and in a discussion amplified by Susman and Pollak (2013), even though important advances in technology and conceptualization have been made with respect to measuring the neurobehavioral substrates thought to underlie DBD, there are still severe limitation in the sensitivity of measurement and specificity of focus. Thus, while a small but apparently robust set of candidate genetic variants associated with risk for DBD have been identified, these are likely to be only part of a group of genes, gene–gene and gene–environment interactions with varying influence from direct to conditional and indirect influence on risk of DBD. Similarly, as noted by Susman and Pollak, while neurophysiological and neuropsychological investigations have begun to relate hormonal regulation with attention, arousal, and memory in DBDs, these areas of potential importance have limited specificity with respect to DBD. For example, animal models of neurobiological systems of regulation and environmental impact have suggested that deprivation relates to differences in brain structure in the cerebellum among previously institutionalized children which they associated to lower executive functioning in a manner consistent with the findings of Pollak and colleagues (Bauer, Hanson, Pierson, Davidson, & Pollak, 2009). Pollak (2008) suggested that this may result from deprivation of necessary maternal care. Susman and Pollak (2013) identify several other findings of structural and functional deficits in relation to trauma, aggression, or other disruptive behavior features. Analogous findings are summarized for the endocrine system, particularly the HPA axis, for a role in behavioral regulation. These exciting initial pieces of potential explanation call for more specific and focused study not only of the hypothesized processes that relate these to other pieces of evidence for neurobiological influences.

Further, there is a need for coordinated studies that examine the extent of environmental challenge, including deprivation, or harm, relate to such brain changes as a requisite for DBDs. As Susman and Pollak suggest, there is evidence that even seemingly minor traumatic experiences can attenuate basal cortisol levels but it is not clear whether this relates to risk for DBD in the same manner as more serious traumatic or chronic experiences of unmet developmental need (deprivation, mistreatment). In other words, as their chapter points out, there is a pressing need to determine if the effects on brain structure and function and related hormonal shifts are dependent on a qualitatively detrimental experience (a traumatic event or chronic unmet need). Apparently, DBD risk increases when an individual's needs are met

less well. Programs of research that can map, within a genetic framework, the relation of brain processes and physiology and of regulatory processes and behavior neurodevelopmentally are critical not only for identifying formulations of typical developmental advancement but also for understanding how variation relates to psychopathology such as DBD. As DBD seems to be characterized by self-regulatory limitations in management of affect and planning of action, the evident interplay between neurocognitive and neurophysiological factors, as related by Susman and Pollak, offers insights that can advance our understanding of DBDs.

The suggestions of Marceau and Neiderhiser (2013) and the complementary approaches offered by Susman and Pollak (2013) do not suggest that studies should be reductionistic nor is it like that such work will lead to a simplified understanding of DBDs. In the discussion of gender and DBD by Loeber et al. (2013), there are multiple levels of explanations for differences to be considered. Most models are summative, meaning the transactional interplay of multiple influences account, in toto for the likelihood of DBD, the course once it is manifest, and the features that will be evident over time. In addition, the influence process is one of confluence, while there are direct effects that can be traced to specific factors; there are also interactions and indirect influences. For example, child irritability may promote DBD but also may elicit and be affected by harsh and inconsistent parenting which, in turn, also increases the risk for disruptive behavior problems. The overall risk models suggested by Marceau and Neiderhiser are clear with respect to family influences found in the chapter by Tolan, Rutter, and Dodge (2013). But, we have been less than adept at disentangling the avenues of influences and which "level" of influence from direct genetic influence to childrearing conditions are truly causal and in learning how complexly these factors are related. As these various influences are more finely measured, with a particular eye toward differentiating interdependence from misattribution of influence due to research design limitations or confounding, it seems likely that family influences will continue to be central to understanding all forms of DBD, and that family-focused interventions will remain at the forefront of approaches for treatment and prevention. Further elaborations are needed for the specific influences and transaction related to these influences in relation to subgroup patterns of DBDs and the specific clinical manifestations as well as how the typical and atypical developmental course unfolds. Tolan et al. (2013) illustrate the need to appreciate the interdependence of biological, psychological, and sociological processes in our studies and challenge to overtly incorporating the study of these processes in future studies. This may be an alternative to a search for "the root cause" or "the true influence."

Toward Intervention Guiding Neurodevelopmental-Ecological Understanding

Although this symposium focused on describing and formulating potential developmental models for DBDs, informed by recent advances in genetic, neurophysiological, and neuropsychological measurement and findings, it also had the goal of

pointing the field toward more advanced approaches for intervention. Ultimately, there is an essential interest in what can be done to prevent, treat, and ameliorate DBDs. While there has been substantial advances in the development of, and proving efficacy for several interventions for DBDs, the understanding of effects, like the understanding of causes of DBD, remains general and with considerable additional work needed (Dishion & McMahon, 1998). Of particular importance is the need to identify what differentiates clinical needs, the interventions themselves, and the likely responses. One of the most promising implications of the work of Frick and colleagues on callous-unemotional traits is that it may help identify which subgroup is less likely to benefit from family and parenting interventions (Frick & White, 2008). Subsequently, this may also point toward alternatives or elaborations of current intervention that can meet the unique needs of this subgroup. Similarly, there is limited understanding of what makes parenting interventions effective (Dishion & McMahon, 1998). While there have been mediational studies suggesting that parenting improvement along lines theorized to mitigate risk is critical (see Forgatch, Patterson, DeGarmo, & Beldavs, 2009), but, to date, even in the most carefully designed and executed studies, the results do not explain fully what is causing the effects related to intervention exposure. For example, the extensively and well-studied FastTrack intervention to prevent conduct disorder shows variation in effects over the course of development (Conduct Problems Prevention Research Group, 1992, 2007). This points to a need for a re-conceptualization of intervention studies within a developmental ecology framework, with attention to genetics and measurement of the neuropsychological and neurophysiological treatment effects, along with the reduction of symptoms. For example, how can understanding of the variations in clinical presentation and neurodevelopmental indicators of functioning be incorporated into intervention studies? Or, how might assignment in randomized trials take such diverse information into account? Might there be methods to focus on genetic or neurophysiological factors in order to examine how intervention effects vary? Such strategies will likely help expedite the refinement and elaboration of our current, basic (one size fits all) interventions. This will not only help inform intervention design but provide experimental evidence that can efficiently inform the further developmental understanding of disorder. For example, even within a family focused intervention, emphasizing basic parenting skills such as consistency of discipline, close monitoring, and warmth and involvement, some aspects of the intervention are meant to alter parenting on risk, some are meant to help manage the youth and parent risk (i.e., reduce problem precipitating tendencies), and some are meant to educate (i.e., increase understanding of how youth with DBD react to parenting). Perhaps, this can lead to a more concerted effort to test various pathways of causality and therapeutic change, simultaneously, through emerging analytic models for mediation (see MacKinnon, Fairchild, & Fritz, 2007 for an overview of emerging applications). Such an approach is but one step that can represent an important shift in perspective toward a model of multilevel individual within context developmental understanding being extended to intervention effects. With such an approach and its promise of more precise and differentiated identification, clearer connection of causes, precipitants, and exacerbators of risk, better characterization of likely variation in presentation and developmental course of meaningful types of

DBD, and important relating of processes from genetic to person-context systems, it is likely that important and substantial progress will be made toward developing more effective and usable interventions. Similarly, this can promote more emphasis on early intervention and prevention so that the current state of major health, economic, and social costs can be reduced. In the end, it is our hope this exchange and the report from that helps move us toward that important goal.

From the outset, this symposium and this volume were designed to ask more questions than it answers. But, do not be fooled, there is a considerable amount of solid data on the identification, causality, prevention, and treatment of DBDs. Similarly, our understanding of the genetics, neuropsychology, and neurophysiology of DBDs is expanding quite rapidly. Where the current work has attempted to take a leap forward is by integrating seeming disparate work in a single set of conversations and subsequent volume. In so doing, we have attempted to provide a new consensus that will break down traditional barriers and open the way for novel, integrative approaches to studies of the development and treatment of DBDs. In so doing, we have also attempted to shine some light on new strategies and a new agenda for the next phase of etiologic and treatment studies for DBDs. The individual contributions converge in support of this goal, with the interaction during the symposium and this resulting volume suggesting important directions for future work from within a shared framework.

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