

Emerging Research and Theory in the Etiology of Oppositional Defiant Disorder: Current Concerns and Future Directions

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Abstract

A large amount of research has been done on Disruptive Behavior Disorders in general and on Oppositional Defiant Disorder in particular. Although research has examined many facets of Oppositional Defiant Disorder, many questions remain. Further, inconsistencies in terminology and methodological concerns across research studies have made it difficult to think consistently about Oppositional Defiant Disorder. As a result, before examining research concerning the etiology of Oppositional Defiant Disorder, concerns in identifying cases of this disorder are discussed. Risk factors for and potential courses of Oppositional Defiant Disorder are examined in the context of possible varying etiologies. Finally, theories about the etiology of and future directions for research related to Oppositional Defiant Disorder and other behavioral problems are examined.

Keywords: Disruptive Behavior Disorders (DBD), Oppositional Defiant Disorder (ODD), Behavioral Problems.

A plethora of research exists regarding numerous facets of Disruptive Behavior Disorders (DBDs) in general and Oppositional Defiant Disorder (ODD) in particular. This research has examined topics such as the definitions and epidemiology of these disorders, the most likely comorbid or co-occurring disorders, risk factors and potential courses for these disorders, potential etiologies for these disorders, as well as underlying theories and interventions. Although a great abundance of research has been done on each of these topics, many questions remain unanswered. The purpose of this review is to summarize emerging research and theory regarding the etiology of ODD. Before examining the etiology of a disorder, the disorder must be defined and identified reliably and validly. Terminology and methods of assessment for ODD and related behavioral problems vary across research studies, however. Thus, the etiology of ODD is discussed in the context of these terminological and methodological limitations. Once these limitations are addressed, risk factors and theories related to the etiology of ODD are examined. For the purposes of this manuscript, the terms ODD, Conduct Disorder (CD), Attention Deficit/Hyperactivity Disorder (ADHD), and antisocial behavior refer to behaviors that fall under specific diagnostic categories, whereas the term *behavioral problems* refers to the broad spectrum of behaviors that are involved in the development of DBDs in general.

Terminological and Methodological Considerations

Despite an increase in research on DBDs, the construct of ODD has remained relatively unchanged (Rey & Walter, 1999). Based on the criteria suggested by the American Psychiatric Association (2000), ODD is a diagnosis that is defined by a pattern of negative, hostile, and defiant behaviors occurring over at least a six month period of time. In particular, to meet the specific diagnostic criteria for ODD, an individual must demonstrate clinically significant impairment in his or her functioning and must meet at least four of the suggested disruptive symptoms. These symptoms include losing one's temper, arguing with adults, refusing to comply with adults' requests, annoying others, blaming others for one's own mistakes, being annoyed easily by others, being angry, and being spiteful and vindictive (APA, 2000). Further, it also is suggested that the noted disruptive behaviors must occur more frequently than would be expected for an individual of a comparable age and developmental level (APA, 2000). Thus, although the diagnostic criteria are relatively specific, there is still some relative

subjectivity in determining the normative nature of the behaviors that may be observed in any one individual.

Given this subjectivity, ODD, as a diagnostic category, is not consistently agreed upon by researchers and clinicians (Rey & Walter, 1999). Some researchers and clinicians question whether or not ODD is a true disorder, if it can be distinguished from normative developmental patterns of behavior, or if it is merely a mild form of CD (Rey & Walter, 1999; Rowe, Maughan, Costello, & Angold, 2005). For example, distinguishing between normative developmental milestones and clinically significant disruptive behaviors may be particularly difficult during the earlier years of childhood (Keenan & Wakschlag, 2002), as behaviors such as temper tantrums, noncompliance, and aggression appear to be expected during these early years (Keenan, Shaw, Delliquadri, Giovannelli, & Walsh, 1998). A survey of children ranging in age from 2- to 5-years found, however, that ODD is the most common condition diagnosed by far (16.8%), is twice as prevalent in boys (relative to girls), and peaks at approximately the age of 3-years (Rey & Walter, 1999). These results are concerning, however, in that normative behaviors for the toddler and early childhood years potentially may be mislabeled as symptoms of ODD.

Thus, the distinction between normative and problematic development must be made carefully because some disruptive behaviors (e.g., oppositionality) occur normatively during the toddler, childhood, and teenage years (e.g., Campbell, 1990, 1995). Even with such a developmental context for interpreting disruptive behaviors, most researchers and clinicians accept that a persistent and pervasive pattern of defiant and noncompliant behavior represents a disorder. In particular, if disruptive behaviors continue past the expected normative developmental timeframe or if they promote a worsening of or an interference with the development of more age-appropriate skills (e.g., social skills), researchers may be more likely to consider them to be a part of an emerging diagnosable disorder (Campbell, Shaw, & Gilliom, 2000). In such cases, a diagnosis of ODD would be appropriate.

Clouding the situation even further, the ODD label is used to describe a wide range of individuals with a wide range of problems, suggesting that ODD applies to a heterogeneous group of individuals (Kempes, Matthys, de Vries, & van Engeland, 2005; Rey & Walter, 1999). Although ODD generally is diagnosed beginning in the early to middle grade school years and develops by early adolescence, its onset is variable (Christophersen & Finney, 1999). For instance, some individuals begin showing symptoms of ODD in the infancy and toddler years. These symptoms then persist throughout childhood and become more severe over time. In contrast, other individuals show symptoms of ODD that do not increase in severity or persist over time (Rey & Walter, 1999). Thus, different courses for the presentation of disruptive symptoms are encompassed in the diagnosis of ODD.

Further, current research may not address adequately the development of ODD in girls, as many research studies do not analyze data on girls separately (Carlson, Tamm, & Hogan, 1999) or do not examine girls at all. Gender differences in the diagnosis of ODD may be important, however. For example, research that has examined gender differences (Sanson & Prior, 1999) suggested that boys are influenced more by temperamental factors whereas girls are influenced more by familial factors. Thus, although the same diagnostic criteria are used to diagnose boys and girls at this time (i.e., APA, 2000), boys and girls may be susceptible to different etiological factors and take different paths to the same diagnosis. Overall, the heterogeneity that is present across groups described by the ODD label may explain some of the variations in findings noted in the empirical literature (Rey & Walter, 1999) and may cloud future research that is conducted on this disorder.

Although individuals diagnosed with ODD may exhibit disruptive behaviors of varying severity levels (APA, 2000), the relationship between ODD and CD remains unclear, especially when more severe levels of ODD are displayed. Certainly, some researchers have suggested that a proportion of children who are diagnosed with ODD will eventually receive a diagnosis of CD (Loeber & Hay, 1997). Based on

the diagnostic criteria provided by the American Psychiatric Association (2000), CD is characterized as a pattern of disruptive behaviors in which an individual violates the basic rights of other individuals as well as major age-appropriate societal norms or rules. In particular, the individual receiving a CD diagnosis exhibits aggressive behavior toward other individuals and animals, destruction of property, deceitfulness or theft, and major violations of rules (APA, 2000). Further, if an individual meets diagnostic criteria for both ODD and CD, a diagnosis of ODD is then subsumed under the diagnosis of CD (APA, 2000). Unfortunately, research studies have had difficulty separating the study of these disorders, as most studies group these two disorders together or refer to broader terms such as aggressive, defiant, or externalizing behaviors (Christophersen & Finney, 1999; Rey & Walter, 1999; Rowe et al., 2005). These differences in terminology across studies contribute to problems in distinguishing and interpreting evidence related to ODD and other behavioral problems (Rey & Walter, 1999).

Similarly, the comorbidity of ODD with other psychological disorders in general and with other DBDs in particular presents yet another concern. In community samples, children with ODD (i.e., after waiving exclusion criteria) are 15 times more likely to also be diagnosed with Major Depression, 14 times more likely to be diagnosed with CD, and 4 times more likely to be diagnosed with ADHD (Rey & Walter, 1999). Comorbid diagnoses also may vary with the age of the individual. For example, in referred adolescents, the association between ODD and Major Depression is weak, whereas the relation between ODD and ADHD is strong (Rey & Walter, 1999). ODD also is comorbid with learning and communication disorders (Rey & Walter, 1999), suggesting that children and adolescents with this combination of diagnoses will struggle in their academic settings. Given these high comorbidity rates, it is unfortunate that the vast majority of studies examined in a review by Carlson and colleagues (1999) did not specifically assess for ADHD and that only a few studies examined groups of individuals with only ODD or CD. Certainly, these findings suggested that distinguishing ODD from other disorders should be the first step in clarifying the research on this disorder.

Further, given that the rates of comorbidity between ODD and other disorders also may relate to the etiology and/or course of ODD (e.g., impulsivity interacting with oppositionality), it is especially important to account for other disorders when examining ODD. In fact, Carlson and colleagues (1999) suggested that ODD alone does not result in dysfunction unless accompanied by ADHD. This conclusion, however, is clearly tentative given the little existing data and small sample sizes noted. On the other hand, it may be misleading to refer to overlapping disorders as comorbid when they share a common etiology, such as different diagnoses resulting from atypical brain development (Kaplan, Dewey, Crawford, & Wilson, 2001). Although research has supported distinct diagnostic categories for the DBDs, a great deal of overlap among externalizing disorders does exist (Frick & Kimonis, 2005). Given these findings, it is apparent that more work must be done in distinguishing ODD from comorbid disorders, including other DBDs. At the very least, research studies must begin to distinguish those children and adolescents who would receive an ODD diagnosis alone from those who would receive an ODD diagnosis in conjunction with other identifiable diagnoses.

Overall, each of these concerns can be tied together with an examination of caseness (Lahey, Miller, Gordon, & Riley, 1999). Research examining various operational definitions of ODD found that various definitions produce different prevalence rates, test-retest reliabilities, and associations with risk factors (Frick & Kimonis, 2005; Lahey et al., 1999). Similarly, different studies using different methods of assessment found different prevalence rates as well (Frick & Kimonis, 2005). Even variation within the same method of assessment (e.g., using different checklists) has yielded varying prevalence rates (Lahey et al., 1999). As a result of findings such as these, Lahey and colleagues (1999) suggested that prevalence rates and other epidemiological findings are method-specific, making the selection of assessment methods a particularly crucial component of designing a research study meant to examine ODD. Thus, prevalence rates will vary depending on how parameters such as pervasiveness, severity, persistence, and impairment are defined (Christophersen & Finney, 1999; Rey & Walter, 1999). For example, recent studies examined

by Rey and Walter (1999) reported 3-month prevalence rates of 2.8% to 3.4% for ODD, whereas other research noted prevalence rates for ODD as high as 8.7% (Christophersen & Finney, 1999). Given these findings, epidemiological research of ODD appears to be incomplete for the reasons noted above.

Overall, researchers and clinicians examining ODD must know clearly what they are measuring and how they are measuring it before clear advances can be made. McMahon and Frick (2005) provided recent insight into and suggestions for addressing these measurement difficulties. Although the diagnosis of ODD is well accepted at this point, its use varies as it is often subsumed under broader terminological categories and measured differently across research studies. Further, comorbid disorders, which are often unmeasured, may influence the etiology and development of ODD. These terminological and methodological inconsistencies regarding ODD continue to impede a clear understanding of the various possible presentations and etiologies of ODD (Rey & Walter, 1999) and may, in part, help to explain the mixed pattern of results on DBDs found in the current research (Carlson et al., 1999; Rey & Walter, 1999). Thus, researchers must strive to enhance the clinical utility, sensitivity, reliability, and validity of measures assessing conduct problems (McMahon & Frick, 2005) and other disruptive behaviors. Despite these shortcomings, the diagnosis of ODD has proven to be useful. Overall, ODD is one of the more common diagnoses given to children and adolescents referred to mental health services and represents a significant portion of children and adolescents who are receiving mental health services (Christophersen & Finney, 1999; Rey & Walter, 1999). With the context of these limitations in defining and measuring cases of ODD, risk factors in the etiology of ODD are now discussed.

Risk Factors in the Etiology of ODD

Overall, numerous factors, including familial, genetic, biological, environmental, and individual factors, have been associated with behavioral problems in children and adolescents and may play a role in the etiology of ODD (Christophersen & Finney, 1999). Following is a review of recent literature regarding the variety of risk factors associated with ODD and related behavioral problems.

Demographic Risk Factors. Sex differences between boys and girls often have been noted for behavioral problems, with boys tending to have a higher number and a greater severity of behavioral problems relative to girls (Alvarez & Ollendick, 2003; Lahey et al., 2000). For example, boys tend to exhibit more mother-reported physical aggression and fewer prosocial behaviors (Romano, Tremblay, Boulerice, & Swisher, 2005). Such findings may be due in part to societal or stereotypical expectations regarding appropriate behaviors for boys versus girls. For example, parents may reinforce antisocial behaviors differentially in boys versus girls, suggesting that the initial developmental pathways to behavioral problems may be the same between the sexes but that these pathways then are influenced directly by parental responses (Alvarez & Ollendick, 2003; Keenan & Shaw, 1997). Further, boys may be at greater risk for neurodevelopmental problems, peer problems, and ADHD (Messer, Goodman, Rowe, Meltzer, & Maughan, 2006), possibly resulting in susceptibility to disruptive behaviors. In contrast, girls may be easier to socialize (Keenan & Shaw, 1997) and may be less likely to attribute hostile intentions to the behavior of others (Frick et al., 2003).

Differences in prevalence rates between the sexes appear to lessen by adolescence, however. In general, using a nationally representative sample of Canadian children, it was shown that the majority of children use occasional instances of physical aggression in their early years but then use very few (if any) instances of physical aggression by pre-adolescence (Côté, Vaillancourt, LeBlanc, Nagin, & Tremblay, 2006). With regard to sex differences and developmental changes over time, however, it may be that case that young girls have higher levels of empathy and guilt but that these characteristics decrease with age (Alvarez & Ollendick, 2003; Keenan, Loeber, & Green, 1999). For example, girls may have better communication skills than boys of the same age and, therefore, have more positive interactions with others early on (Alvarez & Ollendick, 2003; Keenan & Shaw, 1997). At some point in development, these patterns may change or reverse, with boys catching up in their social skills or finding a different manner

in which to related to others and girls lessening their use of empathy. Although there may be an interaction between sex and age, age alone also has been related to the development of behavioral problems. For example, the relationship between age and the development of behavior problems has led to age-related diagnostic subtypes of CD, with individuals in the childhood-onset subtype experiencing more chronic behavioral problems and those in the adolescent-onset subtype having more proximal risk factors (Alvarez & Ollendick, 2003). Such patterns may be applicable to ODD as well.

Low socioeconomic status (SES) also has been identified as a demographic risk factor for behavioral problems (McGee & Williams, 1999). Research found that low SES is a risk factor for almost 60% of families of children with behavioral problems in contrast to 23.8% of families of children without behavioral problems (Alvarez & Ollendick, 2003; Loeber, Green, Keenan, & Lahey, 1995). Some research suggested that there is a direct relationship between low SES and aggression, whereas other research indicated that low SES is related to other risk factors that are associated with behavioral problems (Alvarez & Ollendick, 2003; Lahey et al., 1995). For example, children from low SES backgrounds may be exposed to high rates of community and domestic violence, poor peer influences, negative parental adjustment, and low levels of social support (Alvarez & Ollendick, 2003; Brennan, Hammen, Katz, & Le Brocque, 2002; Wakschlag & Keenan, 2001). In addition, low SES has been associated with poor parenting factors, such as coercive and inconsistent parenting as well as poor parental monitoring. Each of these correlates have, in turn, been related to problematic disruptive behaviors (e.g., chronic adolescent offending; Capaldi & Patterson, 1994).

Given the large intra-group differences across individuals from a low SES background, the importance of examining other risk factors in the development of behavioral problems has been emphasized (Alvarez & Ollendick, 2003). For example, ethnicity also has been related to behavioral problems; however, research suggested that ethnicity may act as a risk factor only to the degree to which it is related to other risk factors (Alvarez & Ollendick, 2003; McGee & Williams, 1999). Further, neighborhood factors related to both low SES and ethnicity may be important. For example, living in poverty or in impoverished areas has been associated historically with increased rates of physical aggression in children (Reiss & Roth, 1993; Romano et al., 2005). These findings may be qualified by the fact that poor families live in neighborhoods that have higher concentrations of other poor families, all of whom may have aggressive children (Tremblay et al., 1996). Thus, demographic risk factors may act in tandem, rather than individually, to promote ODD-like behaviors.

Familial and Other Environmental Risk Factors. Research reviewed by Carlson and colleagues (1999) suggested that groups of children and adolescents with ODD tend to have an impaired family environment relative to control groups (e.g., Frick et al., 1992; McGee & Williams, 1999). For example, levels of impairment in paternal-rated family dysfunction and child-rated affectionless control of groups with ODD equal that of groups with CD. In contrast, levels of impairment in maternal supervision, maternal inconsistent discipline, and parent ratings of affectionless control in groups with ODD fall between that of groups with CD and control groups (Carlson et al., 1999). Further, parenting stress, low behavioral responsiveness, and use of harsh discipline also have been related to elevated levels of disruptive behavior disorder symptoms (Wakschlag & Keenan, 2001). Overall, research on family environment variables demonstrated consistently that there is a relationship between negative family environment, such as low cohesion and high conflict, and behavioral problems in children (Alvarez & Ollendick, 2003; Carlson et al., 1999; McGee & Williams, 1999).

Further, parental psychopathology has been linked to ODD and other DBDs in children and adolescents. In general, it was demonstrated that aggressive and antisocial tendencies run in families (Capaldi, Conger, Hops, & Thornberry, 2003). Fathers who had children with ODD or CD exhibit higher rates of substance use than did control fathers (Carlson et al., 1999). Although one study found equal levels of psychopathology in fathers of children with ODD and CD, another study found that fathers who

have children with CD show more antisocial personality characteristics than fathers who have children with ODD (Carlson et al., 1999; Frick et al., 1992; Schachar & Wachsmuth, 1990). Other forms of parental psychopathology also have been related to the disruptive behaviors of children and adolescents, however. In addition, Romano and colleagues (2005) found that a higher level of maternal depression is a significant predictor of higher levels of physical aggression and lower levels of prosocial behavior in children. Thus, parental psychopathology may be an important predictor of ODD in children and adolescents. This relationship may be one that is direct; however, parental psychopathology also may be related to the manner in which mothers and fathers parent their children and adolescents who are diagnosed with ODD.

Consistently, research on parenting variables demonstrated that parents of children with behavioral problems may be less consistent in their parenting, show more negative expectations, be less caring, and show higher levels of stress than parents of children without such problems (Carlson et al., 1999; Frick et al., 1992; Wakschlag & Keenan, 2001). Further, negative parenting styles (e.g., uninvolved, rejecting, harsh) have been related to behavioral problems in children (Alvarez & Ollendick, 2003; Carlson et al., 1999; McGee & Williams, 1999). For example, children with problematic behaviors have a history of higher rates of negative interaction and of lower rates of harmonious interaction (Gardner, 1987). Although negative maternal control predicts children's noncompliance or aggression, difficult children also may elicit more inconsistent behavior from their caregivers (Shaw, Keenan, & Vondra, 1994). Thus, interactions between parents and children with behavioral problems may be characterized generally by more disapproval and negative affect, less positive expressiveness, and more parental control. Such characteristics lead both children and parents to have fewer positive cognitions and expectations about their interactions (Carlson et al., 1999).

In addition, parents of children with ODD have been shown to exhibit less effective problem-solving skills, particularly when dealing with conflict (Rey & Walter, 1999). For example, these parents criticize and blame their children during conflictual interactions (Rey & Walter, 1999) and engage in coercive patterns of exchange (Patterson, 1982). Current research in this area, however, has not provided definitive evidence about the etiology of ODD because the direction of causality is unable to be determined (Rey & Walter, 1999). For example, negative parental behavior may evoke oppositional reactions from children and oppositional behaviors from children may evoke negative parental behaviors, or both. Some researchers have suggested that the exchange between parents and their children is bidirectional, with parents placing a demand on their children, their children failing to comply, parents then withdrawing from their demand, and the children then engaging in a positive or neutral behavior because the demand was withdrawn (Patterson, 1982; Patterson, Reid, & Dishion, 1992). Thus, ODD could be children's response to an overcontrolling environment and parenting that is inconsistent and/or harsh (Rey & Walter, 1999).

Parental separation, divorce, and marital discord may be other environmental correlates in the development of behavioral problems (McGee & Williams, 1999; Slutske, Cronk, & Nabors-Oberg, 2003). A recent meta-analysis by Amato (2001) suggested that parental divorce and behavioral problems in children share only a modest relationship. Some research suggested that this association is due merely to preexisting maternal characteristics, whereas other studies controlling for genetic factors found a direct association (Slutske et al., 2003). Thus, parental divorce may play an active role in the development of behavioral problems. Given the mixed results, however, no conclusive statements about the relationship between divorce and behavioral problems can be made (Carlson et al., 1999). Research, however, has shown a stronger relationship between behavioral problems and marital distress (Carlson et al., 1999).

A few other select variables have been related to the development of behavioral problems as well. For example, prenatal exposure to maternal smoking has been implicated in the development of behavioral problems in children (Slutske et al., 2003; Wakschlag & Keenan, 2001). Although prenatal

exposure to nicotine may predict later behavioral problems, this exposure has been associated with several other risk factors as well, such as low SES, experiencing prenatal exposure to other substances, receiving less maternal nurturance, experiencing parental divorce or separation, sexual abuse, and exposure to criminality (McGee & Williams, 1999; Slutske et al., 2003). Peer influences also may play a role in the development and maintenance of behavioral problems; however, this relationship may be reciprocal in nature (Alvarez & Ollendick, 2003; Rey & Walter, 1999). Similarly, peer rejection has been associated with later behavioral problems, even after accounting for previous levels of aggression (Alvarez & Ollendick, 2003).

Dispositional Risk Factors. A number of dispositional factors, many of which are reviewed by Alvarez and Ollendick (2003), have been associated with behavioral problems. One such factor is difficult temperament, including lability, restlessness, negativism, and short attention span (Sanson & Prior, 1999; Wakschlag & Keenan, 2001). This factor is first evident during infancy and has been identified frequently across studies as a precursor to behavioral problems (Alvarez & Ollendick, 2003; Rey & Walter, 1999; Sanson & Prior, 1999; Werner & Smith, 1977). For example, mothers' ratings of difficult infant temperament, as well as ratings of mother-child interactions and mothers' ratings of aggression when their children were 3-years of age, predicted significantly children's conduct problems when they were 9-years of age (Campbell & Ewing, 1990). One possible pathway through which difficult temperament may lead to behavioral problems is that children with such temperaments may be harder to discipline and interact with and may be more likely to evoke negative parenting behaviors (Alvarez & Ollendick, 2003). Keenan and Shaw (2003) also suggested that toddlers with difficult temperaments may be either underaroused or overaroused and receive subsequent parenting that does not promote the most optimal outcome. In general, ODD may represent the extreme end of certain temperamental characteristics (Rey & Walter, 1999).

Another dispositional factor is a reward-dominance behavioral style (Alvarez & Ollendick, 2003; Crowell et al., 2006; Kempes et al., 2005). Research on this factor suggested that two independent subsystems of the brain are involved. These subsystems are the Behavioral Inhibition System (BIS), which inhibits behavior in the context of novel stimuli, innate fear stimuli, and signals of nonreward and punishment, and the Behavioral Activation System (BAS), which activates behavior in those contexts (Alvarez & Ollendick, 2003; Gray, 1970). One possible pathway relating this factor to disruptive behaviors is that antisocial individuals may have an unbalanced system in which the BAS dominates behavior over the BIS (Alvarez & Ollendick, 2003). In these cases, behavior is determined more by rewards instead of avoidance of punishment, resulting in patterns of behavior with less regard for social norms and consequences (Alvarez & Ollendick, 2003). As part of this possible pathway, some children continue to engage in increasingly deviant activities despite the negative consequences of their behaviors and inhibit their actions to avoid consequences less and less over time (Alvarez & Ollendick, 2003).

Children who display a reward-dominant behavioral style also tend to display callous and unemotional (CU) traits (Alvarez & Ollendick, 2003; O'Brien & Frick, 1996). CU traits are a temperamental-like set of traits related to psychopathy that consist of a lack of empathy and helpfulness, selfishness, decreased guilt, a lower need for social interaction and approval, and diminished emotional expression (Alvarez & Ollendick, 2003; Barry et al., 2000; Frick, Bodin, & Barry, 2000). Early onset of CU traits may predict the development of behavioral problems, particularly those behavioral problems that are influenced less by environmental factors, persist longer, and are more severe (Alvarez & Ollendick, 2003; Frick, 1998). In fact, those children who exhibit conduct problems and CU traits in combination demonstrate higher levels of conduct problems, delinquency, and contact with police (over a four year study period) relative to those without CU traits. Those with conduct problems only also exhibit higher rates of such problems relative to children categorized as not having conduct problems (Frick, Stickle, Dandreaux, Farrell, & Kimonis, 2005). In addition to the role of biological processes (i.e., lower behavioral inhibition consistent with a reward-dominant behavioral pattern), interactions among parents

and children both sharing CU traits may lead to behavioral problems in children (Alvarez & Ollendick, 2003; Frick, 1998). For example, parents may be hindered by their own psychopathic features when they are attempting to provide appropriate social modeling, responsiveness, and discipline. Thus, it is unlikely that a child with existing behavioral problems would receive the parenting necessary to prevent behavioral problems (Alvarez & Ollendick, 2003).

Decreased cortisol, a hormone produced by the adrenal cortex during times of stress, is another dispositional factor associated with behavioral problems in children (Alvarez & Ollendick, 2003; Loeber et al., 2000; McBurnett, Lahey, Rathouz, & Loeber, 2000). Research found that boys and girls with behavioral problems have low salivary cortisol levels at resting state and in response to stress, with low levels also being associated with aggressive symptoms (Alvarez & Ollendick, 2003; Loeber et al., 2000). Demonstrating the influence of both biology and environment, cortisol levels may suggest that the BAS and BIS systems interact in the manifestation of behavioral problems and may be reduced by environmental stressors during prenatal and childhood development (Alvarez & Ollendick, 2003; Loeber et al., 2000). Although this factor may be associated with behavioral problems, it has been a poor predictor unless considered in the context of other interacting factors (Alvarez & Ollendick, 2003).

Neuropsychological reviews also have demonstrated deficits in children with behavioral problems. Some form of frontal lobe dysfunction may be involved in children who have difficulty regulating their aggressive behavior (Pihl, Vant, & Assaad, 2003). For example, those categorized as stable aggressives (i.e., those who consistently behave aggressively) score lower on verbal and executive functioning (EF) relative to those categorized as nonaggressives and occasional aggressives (Pihl et al., 2003). EF involves an individual's regulation of goal-directed behavior and measures how one solves a problem rather than one's acquired knowledge (Hogan, 1999). Thus, the disinhibited behavior of children with behavioral problems may be the result of poor EF rather than a lack of general ability (Hogan, 1999). In fact, performance of EF tasks has been related to subsequent aggression ratings; however, this finding has not been found in research that controls for ADHD symptoms in children with CD (Hogan, 1999). It appeared that EF deficits may be found in children and adolescents with ADHD or ADHD and ODD/CD but not in those with only ODD or CD (Clark, Prior, & Kinsella, 2002; Oosterlan, Scheres, & Sergeant, 2005).

Intellectual deficits and poor academic achievement often have been associated with the development of behavioral problems as well. In general, children and adolescents with behavioral problems have slightly lower levels of intellectual functioning than the general population, especially in verbal abilities, even after controlling for SES (Alvarez & Ollendick, 2003; Speltz, DeKlyen, Calderon, Greenberg, & Fisher, 1999). A number of possible pathways explaining the relationship between intellectual deficits and behavioral problems have been suggested. For example, intellectual deficits may impact negatively children's range of responses to perceived threats; verbal intellectual deficits may impair self-regulation strategies important for delaying gratification, controlling affective reactions, and anticipating consequences; intellectual deficits may prevent learning in one context from generalizing to other contexts; and intellectual deficits may impede positive interactions with others (e.g., poor communication skills resulting in increased frustration and negativity in both parent and child; Alvarez & Ollendick, 2003). This relationship has been most evident in children who show an earlier onset of behavioral problems in the absence of antisocial traits (Alvarez & Ollendick, 2003).

Research studying specific groups of children with behavioral problems has provided more detailed information about the relationship of intellectual functioning and behavioral problems. A review by Hogan (1999) found that, when children with only CD are examined, intellectual deficits are not shown. In contrast, children with both CD and ADHD often show intellectual deficits. Studies reviewed by Hogan (1999; e.g., Anderson, Williams, McGee, & Silva, 1989; Campbell, Pierce, March, Ewing, & Szumowski, 1994; Chandler & Moran, 1990; Frick, O'Brien, Wootton, & McBurnett, 1994; Goodman,

Simonoff, & Stevenson, 1995; Moffitt, 1990; Nagin, Farrington, & Moffitt, 1995; Noam, Paget, Valiant, Borst, & Bartok, 1994; Sonuga-Barke, Lamparelli, Stevenson, Thompson, & Henry, 1994; White et al., 1994) indicated that research on children in preschool did not show a link between intellectual deficits and behavioral problems, research during middle childhood is mixed with some studies suggesting a link and others not, and most research during adolescence suggests that intellectual deficits are associated with behavioral problems. Other longitudinal studies reviewed by Hogan (1999; e.g., Goodman, 1995; Schonfeld, Shaffer, O'Connor, & Portnoy, 1988; White, Moffitt, & Silva, 1989) support these findings.

Behaviors associated with this relationship have included truancy, staying out late, and stealing, suggesting that intellectual deficits may be detectable only after years of schooling or a lack thereof. Also, some research suggested specifically that children with behavioral problems have deficits in verbal intellectual deficits, whereas other research suggested overall intellectual deficits (i.e., lower performance and lower verbal intellectual functioning). Research involving path analyses reviewed by Hogan (1999; e.g., Ferguson, Horwood, & Lynskey, 1993; Goodman et al., 1995; Loeber et al., 1995; Moffitt, 1990) suggested that, although intellectual deficits may be a cause for behavioral problems, behavioral problems are not a cause of intellectual deficits. Of importance, studies that have controlled for ADHD have not found this link (Fergusson, Horwood, & Lynskey, 1993; Loeber et al., 1995; Moffitt, 1990; Speltz et al., 1999).

In addition to possible intellectual deficits, children with behavioral problems often achieve academically below what would be expected for their intellectual functioning, indicating that intellectual deficits alone do not account adequately for academic underachievement in these children (Alvarez & Ollendick, 2003). Suggestive of high comorbidity rates, 11 to 61 percent of children with behavioral problems have a learning disability (Alvarez & Ollendick, 2003; Hinshaw, 1992). One pathway that may account for these findings may be that ADHD, not ODD or CD, in early childhood leads to academic problems in early to middle adolescence (Clark et al., 2002). It also may be that, in those children who have a later onset of behavioral problems, academic underachievement may lead to behavioral problems without the presence of ADHD (Alvarez & Ollendick, 2003). As mentioned with other factors, the role of intellectual functioning and achievement factors must be considered in the broader interactional context of other factors (Alvarez & Ollendick, 2003; Clark et al., 2002).

Social cognition may play a role in the development of behavioral problems as well. Social information is processed in a linear order, including encoding a social stimulus, storing interpretations of the stimulus, accessing behavioral and affective responses, evaluating the response in terms of expectations, norms, and anticipated consequences, and enacting the response (Alvarez & Ollendick, 2003; Kempes et al., 2005). Errors in this sequence may lead to the development of behavioral problems (Kempes et al., 2005). For example, children with behavioral problems, especially those with aggressive symptoms, tend to perceive a higher amount of threat in their environment than other children, even in ambiguous or benign situations (Alvarez & Ollendick, 2003; Crick & Dodge, 1996; Kempes et al., 2005). Due to this distortion, these children may maintain or increase their behavioral problems. Also, children with behavioral problems may minimize harmful aspects and overemphasize positive aspects of their behavior (Alvarez & Ollendick, 2003; Crick & Dodge, 1996), suggestive of a reward-dominant behavioral pattern. Again, considering the role of other factors is important, as children without problem-solving skills or frustration tolerance may not be able to access social scripts in an adequate manner (Alvarez & Ollendick, 2003).

Research reviewed by Hogan (1999) provided additional information on the role of cognition in the development of behavioral problems. For childhood-onset CD, two cognitive patterns have been identified. Early-onset cases often have attentional problems and typically show cognitive difficulties from an early age (i.e., they have intellectual deficits, experience academic trouble at school onset and onward, have difficulty developing age-appropriate social knowledge, demonstrate biased and

maladaptive social information processing, and usually show EF deficits and trouble in self-regulation; Hogan, 1999). These cognitive deficits are associated with several other risk factors, including genetic, developmental, familial, and environmental variables (Hogan, 1999). Thus, children who demonstrate CD and ADHD behavioral patterns experience their cognitive difficulties in the larger context of multiple risk factors (Hogan, 1999).

Data reviewed by Hogan (1999) described a second, smaller group of early-onset CD children. These children did not show the numerous cognitive deficits of the children with CD/ADHD, have normal intellectual functioning at least into early adolescence, and are less prone to errors in social cognition. They may be less likely, however, to develop the social and emotional skills necessary for adequate social interactions because they place greater value on outcomes of aggressive acts and minimize aggression's negative consequences (Hogan, 1999; Kempes et al., 2005), suggestive of a reward-dominant or CU behavioral pattern. Increased sensitivity to rewards and trouble delaying gratification may lead to the development of disinhibited and interpersonally maladaptive behavior patterns. Children with pure CD may experience a cumulative intellectual deficit in adolescence rather than in childhood secondary to their behavioral and social problems at school (Hogan, 1999).

Previous Behavioral Patterns. It often has been said that the best predictor of future behavior is past behavior, and this may be the case with ODD (Crowell et al., 2006). Even when identified and treated early, early diagnosis of ODD is often, but not always, predictive of more serious DBDs and psychiatric diagnoses later in development (Christophersen & Finney, 1999). Conversely, children who later developed DBDs often show noncompliant, hostile behaviors earlier in their development (Rey & Walter, 1999). In fact, if behavior problems are stable from preschool to school age, they are more likely to continue into adolescence (Ewing & Campbell, 1995) and, perhaps, adulthood (Moffitt, 1993). Overall, the link between early aggression and later development of behavioral problems has been well established (Alvarez & Ollendick, 2003; Sanson & Prior, 1999).

In fact, physical aggression has been considered one of the more powerful predictors of behavioral problems. Although the frequency and severity of aggression tend to decrease with age in the general population, it increases with age in children who develop behavioral problems (Alvarez & Ollendick, 2003; Kempes et al., 2005). Interestingly, children who display proactive aggression (i.e., unprovoked, instrumental, goal-oriented aggressive behaviors) may develop later antisocial, delinquent behavior, whereas children who display reactive aggression (i.e., maladaptive responses to perceived threat that are related to deficits in affective regulation, social cognition, and impulsivity) typically did not develop as severe behavioral problems (Alvarez & Ollendick, 2003; Kempes et al., 2005).

Genetic, Shared Familial, and Nonshared Individual-Specific Risk Factors. Many factors associated with the development of behavioral problems have been discussed thus far. These factors may be influenced by genetics, by the environment, or by both. Further, these factors may interact with each other, as biology is determined by genetics but may be changed by environmental influences. To tease apart these differences, Slutske and colleagues (2003) reviewed three behavioral-genetic investigations of ODD (i.e., as defined by the DSM) with a combined sample size of 4,056 twin pairs. Across the three studies (Burt, Krueger, McGue, & Iacono, 2001; Cronk et al., 2002; Eaves et al., 1997), the weighted mean heritability estimate was 60 percent, and the weighted mean shared family environmental factors estimate was 7 percent. These estimates suggested that shared familial and genetic factors account for approximately 67 percent of the variation in ODD risk. Slutske and colleagues (2003) assumed that the ODD diagnosis has a reliability of approximately .80 and estimated that approximately 13 percent of the variation in the risk for ODD symptoms could be explained by nonshared individual-specific environmental factors.

Although these results are roughly consistent with findings for CD, it was not determined whether or to what extent the same genetic and environmental risk factors are involved in the development of the two disorders (Slutske et al., 2003). To make this determination, Slutske and colleagues (2003) examined results from two twin studies (Burt et al., 2001; Eaves et al., 2001). These results suggested that there is a modest overlap in genetic and nonshared individual-specific environmental risk factors for ODD and CD and a substantial overlap in shared family environmental risk factors (i.e., within-twin correlations of .50 and .36 for boys and girls, respectively, and cross-twin correlations of .35 and .28). These findings suggested that ODD and CD are distinct etiologically at the genetic and nonshared individual-specific environmental level but not at the shared family environment level. These findings also suggested the possibility of distinct etiological pathways that are malleable to environmental influences.

Existing Theories of ODD

Knowledge of the various risk factors of behavioral problems does not account for the mechanisms by which risk factors lead to the development of behavioral problems in general and ODD in particular. Thus, several theories, reviewed by Carlson and colleagues (1999), have been proposed to account for the possible pathways to behavioral problems.

Coercion theory, first described by Patterson (1982), has been the longstanding theory relating parenting styles to behavioral problems in children. This theory, based in operant conditioning, gives credit to individual differences but emphasizes family interactions as the main factor in the development of behavioral problems in children. For example, negative reinforcement shapes the behavior of both parents and children in ways that reduce the negative behaviors of the other (Patterson, 1982). When a parent stops a child's tantrum by providing a treat, a child's tantrums tend to increase both because the child has been reinforced positively by the treat and the parent has been reinforced negatively by ending the tantrum. According to this theory, this reciprocal interaction leads to increasingly negative patterns of interactions in families of children with behavioral problems. In explaining differences between families of children with behavioral problems and families of nonclinical children, the theory suggested that some parents tend to start and continue negative interactional patterns and provide more reinforcement, even if inadvertent, for children's negative behaviors. Though this theory accounted for a number of studies reviewed, Carlson and colleagues (1999) stated that it does not detail adequately the mechanisms that account for the occurrence of coercive processes in some families but not in others.

Greenberg, Speltz, and DeKlyen (1993) applied attachment theory (which was posited initially by Bowlby) to account for the development of behavioral problems in children. In this theory, children form concepts of the self and of other individuals to guide their expectations for handling negative emotions. Negative parenting behaviors are thought to lead to the development of an insecure attachment. Children learn to expect negative feelings in close relationships and do not learn how to deal with negative feelings. These negative expectations provide a working model that maintains children's negative behavior. Simultaneously, parents have their own expectations, which can facilitate either positive or negative responding in the face of behavioral problems. In research reviewed by Carlson and colleagues (1999), it was found that children with DBDs from community samples are more likely to show insecure attachments than controls. They determined, however, that insecure attachment is not a necessary or sufficient cause for behavioral problems. In interaction with other risk factors, however, insecure attachment may be related to behavioral problems. Thus, even though attachment theory may account for some pathways to behavioral problems, it did not appear capable of accounting for the wide range of findings associated with behavioral problems (Carlson et al., 1999).

Another theory regarding the etiology of behavioral problems is the theory of genotype-environment effects discussed by Scarr (1992). This theory suggested that genotypes guide experiences. For example, individuals create their own environments that are correlated uniquely with their genetically based individual differences. Passive, evocative, and active genotype-environment correlations may

occur. Passive effects are those associated with biological parents providing both the genes and the home environment to children. Evocative effects involve the responses of other individuals that are related to certain characteristics of children. Active effects result when individuals' choices about what environments to live in are related to their genetically determined characteristics.

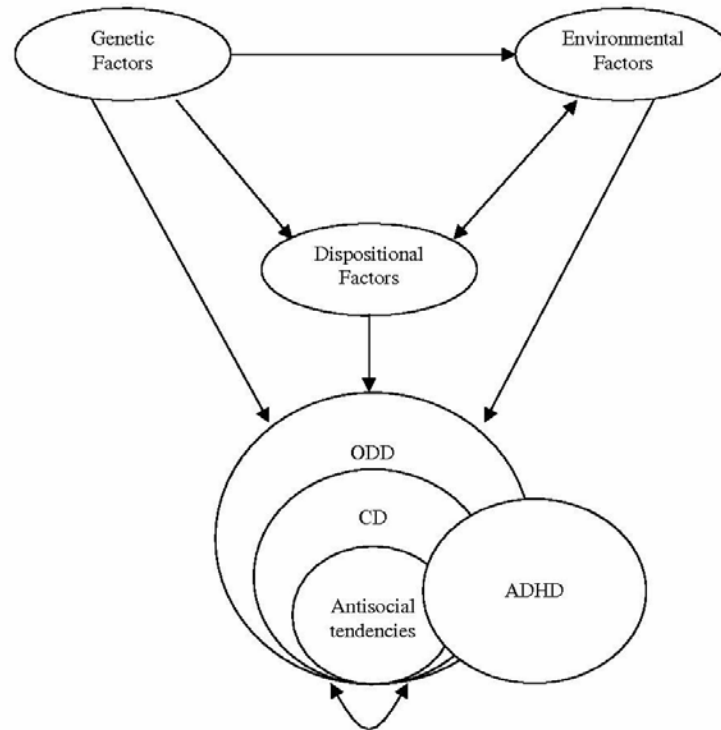
Carlson and colleagues (1999) found this theory to be a feasible framework for integrating the existing findings on the development of behavioral problems. For example, they stated that the relationships among parental, familial, and child characteristics in the development of behavioral problems may be accounted for by passive, evocative, and active effects. Further, they stated that this theory is not inconsistent with other theories but considers a broader range of influences and integrates the role of genetic-biological factors in the etiology of behavioral problems. This theory views both environment and genetics as causative but suggests that genetic variations drive environmental ones. For example, Carlson and colleagues (1999) indicated that fathers of children with ODD or CD experience behavioral problems themselves, demonstrating a genetic component. Further, representing a passive effect, parents of children with CD tend to exhibit negative parenting behaviors. Representing evocative effects, children may evoke negative patterns of interaction. This idea is supported by research showing that confederate boys with CD evoke more negative responses from nonclinical mothers. Further evidence reviewed by Carlson and colleagues (1999) supporting this theory showed that ineffective maternal parenting is unrelated to the severity of behavioral problems for children who display CU traits, suggesting a pure genetic-biological etiology. In contrast, it was found that, for children without these traits, negative parenting is associated with behavioral problems.

Overall, Alvarez and Ollendick (2003) suggested the use of a developmental framework to address the broad findings associated with behavioral problems. For example, behavioral problems may present in a variety of ways across a wide range of contexts over time. Social and parental expectations change across developmental stages as children's communication, self-regulation, social skills, and coping skills mature. Most of the time, behavioral problems are short in duration, minor in severity, and co-occur with developmental stages and changes in familial and social expectations. Thus, risk factors and causal pathways in the development of behavioral problems are viewed within a transactional context involving developing children within an environment. A disorder results from interrelated maladaptive social, emotional, and/or cognitive competencies that hinder adjustment to the environment at a specific stage of development. This framework described by Alvarez and Ollendick (2003) recognizes both individual and environmental factors in the development of behavioral problems.

Multiple Pathways in the Development of Behavioral Problems: A Good IDEA

Risk factors may play a role in the development of behavioral problems in general and ODD in particular through a wide number of pathways. Although the previously discussed theories provide important utility in examining these pathways, they either do not account for and integrate the broad findings, do not emphasize the ongoing development of children, do not give credit to the numerous ways in which risk factors and pathways may interact with each other, do not account for how children's biology is influenced by genetics and the environment (Crowell et al., 2006), and/or do not state explicitly how specific DBDs may develop. Thus, a multiple pathway Interactional-Developmental-Etiological Approach (IDEA) model to account for the development of behavioral problems is suggested here (Figure 1).

Figure 1. Multiple Pathway IDEA Model.



Genetic factors include the extent to which variables, such as parents' psychopathology, parenting behaviors, and children's dispositions, are influenced by genetics. These factors lead to environmental and dispositional factors. Environmental factors include variables, such as SES, parenting, marital relationship, and peer influences. These factors influence and are influenced by dispositional factors. Dispositional factors include variables, such as age, gender, temperament, reward-dominance, CU traits, cortisol levels, intellectual functioning, EF, and social cognition. Genetic, environmental, and dispositional factors lead to the development of behavioral problems, and, depending on the number of and type of factors involved, the development of specific behavioral disorders.

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First, the wide number of possible pathways to behavioral problems must be considered. These pathways include genetic factors (e.g., the extent to which parents' psychopathology, parenting behaviors, and children's dispositions are influenced by genetics), dispositional factors (e.g., age, gender, temperament, reward-dominance, CU traits, cortisol levels, IQ, EF, social cognition), and environmental factors (e.g., SES, parenting, marital relationship, peer influences). It should be noted that many of these factors may play a protective role at one side of their continuum or may present as a risk factor at the other side of their continuum. For example, positive parenting may protect against the further development of an underlying predisposition to behavioral problems, whereas negative parenting may exacerbate such a predisposition. Thus, the IDEA model accounts for adaptive and maladaptive development.

Second, the interactional role of these factors must be considered on two levels. At the first level, broad genetic and environmental factors may interact with each other in producing dispositional factors, particularly those factors that have a biological component. For example, children may be predisposed genetically to a reward-dominant behavioral pattern due to a dominating BAS (e.g., Alvarez & Ollendick, 2003), but this predisposition may not manifest if not nurtured by environmental factors. Specifically, repeated interactions where children learn to attend to positive consequences and ignore negative consequences may increase the capacity at which the already overactive BAS is functioning. Conversely, repeated interactions where children learn to attend more to negative consequences may suppress the predisposition for an overactive BAS and/or increase functioning in the BIS. Parents who are able to provide the necessary positive experiences for their children also may be able to prevent behavioral problems, even in the face of genetic and biological predispositions. Research cited previously examining cortisol levels also demonstrated how genetic and environmental influences may interact with each other (e.g., Alvarez & Ollendick, 2003; Loeber, Green, Lahey, Frick, & McBurnett, 2000; McBurnett et al., 2000).

At the second level, each of specific variables within each broad factor may interact with each other to produce the final manifestation of a behavioral problem. For example, the presence of negative parenting alone may produce a certain likelihood that a behavioral problem will develop, and the presence of difficult temperamental traits alone may do so as well. The combined presence of both of these factors, however, may pose an additive risk in the development of behavioral problems or potentially may produce an interactive effect that is greater than the sum of its parts. The exact nature of the relationships among all these variables is yet to be understood clearly, however.

Third, the importance of the developmental framework argued for by Alvarez and Ollendick (2003) is emphasized. Pathways to behavioral problems may become activated at various stages of development. For example, children who are developing through their preschool years and adolescents who are developing through their teenage years will manifest a certain degree of adaptive behavioral "problems" in striving for their own autonomy. This adaptive level of behavioral problems may be exacerbated to clinical levels in the presence of other risk factors. Thus, age is a critical factor to consider (e.g., Christophersen & Finney, 1999). Sex also is a critical factor, as research demonstrated that males and females may develop differently and may present differing pathways to behavioral problems based on their development (e.g., Carlson et al., 1999; Sanson & Prior, 1999). For example, research cited

previously suggested that males and females are reinforced differentially in behaviors associated with behavioral problems and that certain risk factors are more influential in girls than boys.

Children develop cognitively and socially over time as well. Interruptions or deficits in cognitive and social development may account for the development of behavioral problems. For example, repeated negative social interactions that occur secondary to cognitive deficits (e.g., poor problem-solving, high levels of perceived threat, etc.) may prevent the modeling of appropriate behaviors, result in the reinforcement of aggressive responding and in the perception of more threat, and other outcomes associated with behavioral problems (e.g., Alvarez & Ollendick, 2003; Crick & Dodge, 1996; Kempes et al., 2005). Again, however, these cognitive and social deficits may be countered through positive parenting and other protective factors.

Finally, the IDEA model attempts to integrate the wide range of findings associated with behavioral problems and attempts to do so with consideration for how the previously discussed factors interact in the etiology of specific DBDs. The research reviewed by Slutske and colleagues (2003), who indicated that there are distinct genetic and nonshared individual-specific environmental etiologies for ODD and CD as well as a common shared family environmental etiology, is particularly relevant here. For example, environmental risk factors may or may not be present in conjunction with genetic and dispositional risk factors. Environmental risk factors may play a greater role in the development of pure ODD, whereas genetic and dispositional factors may play a greater role in the development of pure CD, although all factors may play a role in the development of ODD and CD.

For example, inconsistent parenting has been associated with the development of ODD (Carlson et al., 1999; Frick et al., 1992; Wakschlag & Keenan, 2001). Other risk factors, in addition to inconsistent parenting, may be needed for the development of CD, however. Low frustration tolerance in children may lead to the development of ODD as well, but only when combined with parenting that is not equipped to deal with children's low frustration tolerance. Conversely, children who developed CD demonstrated more biological characteristics, such as a reward-dominant behavioral pattern and CU traits (e.g., Frick et al., 2005). Staunchly positive parenting may be able to counteract these risk factors for CD; however, parents of such children often have their own antisocial characteristics that only exacerbate the children's behavioral problem (e.g., Capaldi et al., 2003). Thus, it may be the case that the activation of multiple pathways leads to the development of greater behavioral problems.

ADHD also must be considered within the context of this model. Inattention and impulsivity have been associated often with other cognitive, intellectual, and EF deficits (Clark et al., 2002; Hogan, 1999; Oosterlan et al., 2005; Pihl et al., 2003) and potentially may provide an independent pathway to ODD and CD in conjunction with other risk factors. For example, inattentive and impulsive children may present unique challenges to parents. Negative interactions occurring as a result of children's inattention and impulsivity may lead to further behavioral problems. Further, impulsivity has been related often to difficulties in EF, such as goal-oriented behaviors and the ability to delay gratification (Hogan, 1999). Parents who have environmental and individual resources to provide the positive experiences necessary for the healthy development of children with ADHD may be able to prevent the development of ODD and CD. It becomes increasingly difficult, however, to counter the potentially cumulative or interactive effect of multiple risk factors. Thus, the IDEA model may account for the high rates of comorbidity found among ODD, CD, and ADHD as well as other disorders, as multiple behavioral problems may be expected when multiple pathways to behavioral problems are activated.

The IDEA model also may account for the developmental sequence of ODD and CD. For example, 80 percent of boys with CD had a previous diagnosis of ODD, but this continuity is not bidirectional as over two thirds of children with ODD do not go on to develop CD (Alvarez & Ollendick, 2003; Rey & Walter, 1999). Thus, some pathways may be activated in children with ODD who later

experience the activation of other pathways that lead to a diagnosis of CD. For example, children who receive inconsistent parenting and have low frustration tolerance may present initially with ODD. During development, however, these children may be exposed to another pathway, such as negative peer influences or exposure to criminality, and develop increasingly severe behavioral problems as a result.

Additionally, children who are given a diagnosis of ODD may not develop further behavioral problems due to the absence of other risk factors and/or presence of protective factors. Further emphasizing development, some children who would present with pure CD (e.g., CU, reward-dominant children) are given a diagnosis of ODD merely because they have not reached the developmental milestones necessary to manifest the more complicated behaviors associated with CD (e.g., theft, destruction of property). If these children continue down this pathway without the presence or introduction of protective factors, their behavioral problems may evoke the activation of other pathways that compound their behavioral problems and could lead to the development of antisocial characteristics in adulthood. This hypothesis accounts for the large amount of research suggesting that previous behavioral problems are predictive of future behavioral problems (Alvarez & Ollendick, 2003; Crowell et al., 2006; Sanson & Prior, 1999).

Overall, the multiple pathway IDEA model considers the interactional and developmental processes that occur in the manifestation of behavioral problems. Further, it integrates the broad range of findings associated with behavioral problems and suggests how specific disorders may develop depending on the presence of specific and multiple genetic, environmental, and dispositional pathways to behavioral problems. Finally, it may account for the high comorbidity among DBDs and the developmental sequence of behavioral problems.

Future Considerations

Although the intent of this review was to examine ODD specifically, terminological and methodological inconsistencies in the research, overlapping risk factors, and the possible developmental sequence found in DBDs forces the inclusion of other behavioral problems. Several things must be accomplished to further facilitate understanding of behavioral problems in general and ODD in particular.

First, reliable and valid definitions of ODD, DBDs, antisocial characteristics, externalizing problems, or any other term used to describe the broad spectrum of behavioral problems must be developed and accepted by the research community. In fact, current versions of the DSM and the ICD, though consistent in some respects, classify ODD differently (i.e., the ICD explicitly conceptualizes ODD as part of the same dimension of behavior as CD; Rey & Walter, 1999; Rowe et al., 2005). Second, methodological inconsistencies must be resolved (e.g., McMahon & Frick, 2005). It is expected that an accepted terminology would prove helpful in this area, but more must be done. For example, given that other disorders may be comorbid with ODD and that this comorbidity may relate to the etiology and/or course of ODD, the importance of completing a comprehensive assessment is emphasized. Additionally, given the numerous possible etiologies of behavioral problems, groups of children with behavior disorders, even those with the same diagnosis, may be quite heterogeneous (Kempes et al., 2005; Rey & Walter, 1999). Such heterogeneity could mislead potentially researchers in their attempts to find specific correlates and causes that may be found in homogeneous groups.

Third, the factors related to behavioral problems must be elucidated more clearly. For example, the exact nature of the relationships among the factors discussed here (e.g., additive, interactive) must be examined. Further, several other factors not discussed here may play an important role in the development of behavioral problems. Also, the role of not only risk factors but also protective factors must be examined. Fourth, inconsistencies across the current research must be integrated. Several theories attempting to do so were presented, with the multiple pathway IDEA model being posited.

Finally, given that one of the goals of research is to inform clinical practice, it is important to note that research is done predominantly on groups, whereas practice is done predominantly on individuals. Findings suggested by groups may not apply to the exact etiology of behavioral problems in any one individual, particularly given the numerous pathways in the development of behavioral problems. Thus, it will be important to understand the unique pathways involved in the development of behavioral problems in individuals seeking treatment, possibly using integrative models such as the one proposed here.

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