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Co-occurrence of ODD and Anxiety: Shared Risk Processes and Evidence for a Dual-Pathway Model

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Abstract

Although oppositional defiant disorder (ODD) and anxiety disorders (ADs) often co-occur, the literature is mixed regarding the effects of such co-occurrence. For example, there is evidence that AD symptoms may mitigate ODD symptoms (buffer hypothesis) or exacerbate ODD symptoms (multiple problem hypothesis). A dual-pathway model incorporates previous research and addresses both hypotheses. We describe several possible etiological or risk processes that may underlie each of these ODD–AD pathways, including child temperament, aggression, limbic system processes, executive functioning abilities, and social information–processing biases, and suggest an integrated model. We conclude with implications for the model and directions for future research involving co-occurring ODD and ADs.

Keywords

anxiety; assessment; comorbidity; intervention; ODD; risk factors

Children with oppositional defiant disorder (ODD), which refers to a recurrent pattern of negativistic, hostile, and defiant behavior toward authority figures lasting at least six months (American Psychiatric Association [APA], 1994), are a heterogeneous group inasmuch as any combination of four of the specified eight symptoms are required for diagnosis. It is also specified that the criteria for conduct disorder (CD) cannot be met to obtain the diagnosis of ODD. The prevalence of ODD is approximately 10–12% (lifetime) and 2.7% (three-month) in community samples and up to 25–30% in clinical samples (Costello, Mustillo, Erkanli, Keeler, & Angold, 2003; Loeber, Burke, Lahey, Winters, & Zera, 2000; Nock, Kazdin, Hiripi, & Kessler, 2007). Similarly, children with anxiety problems comprise a heterogeneous group who display excessive worries, fears, and anxiety. *DSM-IV* (APA, 1994) specifies that all of the anxiety disorders (ADs) of adulthood can be observed in childhood (i.e., generalized anxiety disorder [GAD], specific phobia [SP], social phobia [SOC], panic disorder [PD], agoraphobia, obsessive-compulsive disorder, and posttraumatic stress disorder), as well as one disorder whose onset must occur during childhood (separation anxiety disorder [SAD]). ADs are also among the most common mental health problems in youth, with prevalence rates approximating 12–15% (lifetime) and 2.4% (three-

month) in community samples and 30–40% in clinical samples (Costello et al., 2003; Kessler et al., 2005; Ollendick, Jarrett, Grills-Taquechel, Hovey, & Wolff, 2008).

Several epidemiological investigations have explored the comorbidity of ODD and ADs. However, some of these studies did not separate youth with ODD from youth with CD or examine individual ADs, which limits our knowledge of specific ODD–AD relations (Bubier & Drabick, 2009). For example, in a meta-analytic review of 21 community-based epidemiological studies, Angold, Costello, and Erkanli (1999) reported that the rate of ADs among youth with either ODD or CD was about 40%; however, this report did not parse out relations with specific ADs or disentangle these relations with ODD and CD, respectively. Fortunately, in the Virginia Twin Study of Adolescent Behavioral Development, Simonoff et al. (1997) reported three-month prevalence rates of ODD, CD, and the various ADs in 2,762 twins between eight and 16 years of age. Rates of any AD among children with ODD specifically approached 40%; however, the rates were highest for overanxious disorder (the *DSM-III-R* precursor to GAD), SP, and SOC (about 5% each). More recently, Nock et al. (2007) examined comorbid rates of ADs and ODD in the National Comorbidity Survey Replication study. They reported that about 60% of those with lifetime ODD also met criteria for lifetime ADs. The most common comorbid ADs were SOC (31.4%), SP (24.7%), GAD (15.5%), SAD (12.5%), and PD (10.9%). Although these lifetime rates were higher than the three-month prevalence rates reported in the Simonoff et al. study, the highest rates were observed for the same ADs in these studies.

Examination of prevalence rates in clinical samples is also important given their applicability to clinical practice (Caron & Rutter, 1991). However, as with the epidemiological studies, there are only a few investigations of the comorbidity of ADs in clinic-referred children with ODD. In an early study, Greene et al., (2002) reported that approximately 40% of 643 youth (mean age 10.7 years) with ODD also met criteria for an AD. Unfortunately, this report did not include the prevalence of specific ADs. In a specialty clinic for ODD youth between seven and 14 years of age (mean age 10.3 years), T. H. Ollendick and R. W. Greene (in prep.) have found that 53 of the 120 (44%) youth with ODD met criteria for an AD: 28 GAD, 26 SOC, 22 SP, and 18 SAD (numbers exceed 53 because the average child with comorbid anxiety had two or more ADs). Among treatment-seeking anxious youth in the Child/Adolescent Multimodal Study with principal diagnoses of GAD, SOC, or SAD ($N = 488$), the prevalence rate of ODD was 9.43% (Kendall et al., 2010). Taken together, these findings suggest the rates of comorbid ADs among youth with ODD are approximately 40%, whereas rates of ODD among youth with ADs are considerably lower (about 10%), although these conclusions are based on limited research. Nonetheless, these rates are very similar to those found in community-based samples and affirm that ODD–AD comorbidity is greater than what would be expected by chance.

Although the findings are not always consistent, most studies show that when ODD is accompanied by ADs, the clinical presentation is more severe and includes additional academic, social, and familial complications (Drabick, Gadow, & Loney, 2008; Greene et al., 2002). Moreover, ODD when accompanied by an AD is prospectively associated with additional psychological conditions and negative sequelae, including the development of CD, major depressive disorder, substance use and abuse, and affiliation with deviant peers (Drabick et al., 2008; Fergusson & Horwood, 1999; Merikangas & Avenevoli, 2000). Recent reviews of the literature confirm the negative sequelae associated with comorbidity of these disorders (Bubier & Drabick, 2009; Ollendick, Jarrett, Wolff, & Scarpa, 2009).

COMORBIDITY EXPLANATIONS AND DUAL PATHWAYS

There are a variety of potential explanations for the co-occurrence of ODD and ADs, including the possibilities that (a) one disorder may precede the other or (b) shared risk factors or etiological processes account for their co-occurrence. For example, some evidence suggests that ODD precedes ADs among clinic samples of preschool children followed into later childhood (Lavigne et al., 2001; Speltz, McClellan, DeKlyen, & Jones, 1999) and in clinical samples of children followed into adolescence (Burke, Loeber, Lahey, & Rathouz, 2005). Similar findings have been obtained for community samples of youth reassessed 14 years later in late adolescence and early adulthood (Roza, Hofstra, van der Ende, & Verhulst, 2003). Other research, however, suggests that ADs (e.g., SAD) precede ODD, although these findings are limited to clinic samples of youth (e.g., Foley, Pickles, Maes, Silberg, & Eaves, 2004; Last, Perrin, Hersen, & Kazdin, 1996). Moreover, ODD and ADs may arise from shared risk factors; that is, ODD and ADs are associated with overlapping and unique risk factors, and their comorbidity may stem from factors that are shared by ODD and ADs (Angold et al., 1999; Bubier & Drabick, 2009; Caron & Rutter, 1991). Potential shared risk factors include child temperament, social information processing biases, parent-child processes, or neighborhood violence exposure, among others (Bubier & Drabick, 2009; Muris & Ollendick, 2005).

This mixed support for various explanations may be attributable, in part, to heterogeneity in the measurement of ODD and ADs. For example, as seen in our review of the literature, studies often examine a combination of externalizing diagnoses (e.g., attention-deficit/hyperactivity disorder [ADHD], ODD, and CD) and ADs (e.g., GAD, SOC, SP, and SAD) when testing longitudinal trajectories, which makes it difficult to disentangle the complex relations among symptoms and diagnoses. Moreover, the developmental period considered may affect the applicability of each explanation. For example, given ages of onset, SAD may be more likely to precede ODD, but ODD may be more likely to precede GAD (Bubier & Drabick, 2009; Costello et al., 2003). One conclusion we can draw, however, is that mixed support for the developmental pathways between ODD and ADs suggests that a variety of explanations for their co-occurrence are possible.

Further complicating this issue is evidence that at least two patterns may characterize the relations between ODD and ADs: one in which anxiety mitigates or lessens ODD behaviors (i.e., buffering hypothesis) and a second in which anxiety exacerbates or increases ODD behaviors (i.e., multiple problem hypothesis; Garai, Forehand, Colletti, & Rakow, 2009; Hofmann, Richey, Kashdan, & McKnight, 2009; Lansford et al., 2008; Ollendick, Seligman, & Butcher, 1999; Walker et al., 1991). Unfortunately, few studies have tested specific relations among ODD and ADs in the context of these hypotheses. However, research that has considered conduct problems more broadly (e.g., CD, ODD) suggests that these hypotheses may be applicable to co-occurring ODD and ADs. For example, in support of the buffering hypothesis, using data from the National Comorbidity Survey-Revised, Hofmann et al. (2009) reported that among adults with disruptive behavior disorders (ODD, CD, and/or ADHD), co-occurring ADs were associated with lower odds for substance use problems compared to individuals without co-occurring ADs. Similarly, in childhood, youth with co-occurring anxiety and conduct problems (CD or ODD or both) evidenced lower levels of impairment (e.g., fewer police contacts, lower aggression ratings) than youth with conduct problems only (Walker et al., 1991).

However, several studies in adolescence suggest that anxiety may exacerbate conduct problems or fail to show the hypothesized buffering effect. One prospective study suggested that both conduct problems (ODD or CD or both) and comorbid conduct problem-internalizing (anxiety/depressive) disorders were associated with co-occurring substance use

disorders, with steep increases in substance use during adolescence and decreases in substance use by early adulthood (Lansford et al., 2008). Thus, the buffering effect of ADs may be evident in childhood and again in adulthood but not during adolescence. Similarly, co-occurring anxiety may not attenuate or alter levels of risk-taking behaviors among youth with elevated levels of parent-reported aggression (including ODD behaviors; Garai et al., 2009) or the age of onset, frequency, or severity of delinquent behaviors among youth meeting diagnostic criteria for CD (Ollendick et al., 1999). Thus, applying this evidence for the multiple problem hypothesis specifically to ODD, ADs may contribute to high-risk behaviors among youth with ODD, although the developmental period likely affects the strength of these relations.

POSSIBLE ETIOLOGICAL PROCESSES AND RISK FACTORS

In the following section, we review potential risk factors that may be shared by ODD and ADs and may differentially confer risk for the proposed dual ODD–AD pathways (i.e., the buffering hypothesis or the multiple problem hypothesis). This section concludes with a model that links these processes to the dual ODD–AD pathway model.

Temperament

Temperament can be defined as “biologically rooted individual differences in behavior tendencies that are present early in life and are relatively stable across various kinds of situations and over the course of time” (Bates, 1987, p. 1101). Buss and Plomin (1984) developed a three-dimensional model of temperament and referred to it as the emotionality–activity–sociability model. Emotionality refers to psychological instability and a proneness to experience heightened levels of emotions such as fear, sadness, frustration, and anger. The second dimension, activity, refers to characteristics such as tempo, vigor, and endurance, whereas the third dimension, sociability, refers to tendencies to affiliate and to be socially responsive to others.

The temperamental dimension of emotionality is particularly important for our review. It consists of various lower-order traits, of which fear/sadness and anger/frustration are most important (Buss & Plomin, 1984). The specific relations between lower-order traits of emotionality and subsequent behavior problems are nicely illustrated in a longitudinal study of young children by Rydell, Berlin, and Bohlin (2003). These researchers demonstrated that fear/sadness predicted internalizing problems (i.e., anxiety/depression), whereas anger/frustration predicted externalizing problems (i.e., aggression, noncompliance) in later childhood as determined by parent ratings. Similar findings were obtained by Blair (2002), who studied low birth weight, premature infants over a two-year period. Temperamental fear/sadness at 12 months predicted internalizing symptoms at age 3, whereas anger/frustration at 12 months was associated with externalizing symptoms at age 3, again via parent ratings. Thus, emotionality serves to predispose children to various forms of childhood problems; however, the specific lower-order traits of this reactive temperament factor play an important role in the types of problems evidenced by the child.

These relations notwithstanding, the contribution of temperament to the etiology of child psychopathology is not simply a reactive process. According to the reformulated temperament theory of Rothbart (Putnam, Ellis, & Rothbart, 2002; Rothbart & Bates, 1998), temperament consists not only of reactive dimensions such as emotionality, but also a self-regulative factor, labeled effortful control. Effortful control can be defined as “the ability to inhibit a dominant response to perform a subdominant response” (Rothbart & Bates, 1998). Effortful control shows strong similarities to what is frequently referred to as “executive functions” (Murray & Kochanska, 2002). It is generally assumed that the capacity for effortful control processes is inborn (Poggi Davis, Bruce, & Gunnar, 2002), relatively stable

from toddlerhood into early school years (Kochanska & Knaack, 2003; Kochanska, Murray, & Coy, 1997), and develops further as a result of brain maturation and interaction with the environment (Kochanska, Murray, & Harlan, 2000; Posner & Rothbart, 2000).

Contemporary temperament researchers assume that vulnerability to psychopathology is characterized by a combination of high levels of emotionality and low levels of effortful control (Calkins & Fox, 2002; Lonigan & Phillips, 2001; Muris & Ollendick, 2005). More specifically, a stressful life event may elicit negative emotions in children, particularly those who are characterized by high levels of emotionality. However, only children with low levels of effortful control tend to experience difficulties in dealing adequately with these negative emotional states and react with fearful or avoidant behavior, depression, and aggression, dependent upon the lower-order traits of emotionality that characterize the child. In contrast, children with high levels of effortful control are capable of regulating these negative emotions by employing more strategic, flexible, and effective coping strategies (Lengua & Long, 2002; Salmon & Pereira, 2002). This view on the role of temperament in the pathogenesis of child psychopathology offers a plausible explanation for the high prevalence of ODD and ADs among children (Muris & Ollendick, 2005). Moreover, this view suggests that ADs may mitigate ODD among youth with low anger/frustration, high fear, and/or high effortful control, but ADs may exacerbate ODD among youth with high anger/frustration, low fear, and/or low effortful control.

Consistent with these possibilities, Oldehinkel, Hartman, De Winter, Veenstra, and Ormel (2004) reported that, compared to control children, children with internalizing (i.e., anxiety/depression) problems displayed higher levels of fear and children with externalizing problems (i.e., aggression, noncompliance) exhibited higher levels of frustration. Both groups displayed low levels of effortful control. Children with co-occurring parent-reported internalizing and externalizing problems were characterized by strong emotional reactivity (i.e., high levels of both fear and anger/frustration), in combination with low levels of effortful control. Muris, De Jong, and Engelen (2004) found similar results: high levels of emotionality and low levels of effortful control were accompanied by higher levels of anxious–depressive (internalizing) and aggressive–delinquent (externalizing) symptoms. Further, a significant interaction between emotionality and effortful control on psychological problems was noted, revealing that the combination of high levels of emotionality and low levels of effortful control were associated with the highest levels of psychopathology and the comorbidity of internalizing and externalizing disorders in particular.

The findings associated with the temperamental features of emotionality and effortful control are compromised by their reliance on parental ratings of internalizing and externalizing behaviors, rather than psychiatric disorders such as ADs and ODD. Still, inasmuch as internalizing and externalizing problems are predictive of ADs and ODD, respectively, the findings are highly suggestive (Muris & Ollendick, 2005) that temperament may set the stage for dual ODD–AD pathways.

Limbic and Prefrontal Processes

Dysfunction in the limbic system and prefrontal cortex (PFC) also has been linked to both ODD and ADs. The amygdala has been implicated in stimulus-reinforcement learning, as well as recognition and processing of the emotional significance of sensory and social stimuli (e.g., sadness, fear; Blair, 2004, 2007; Crowe & Blair, 2008; Davidson, 2002; Guyer et al., 2008; Thomas et al., 2001). The PFC has been subdivided into a variety of areas that are relevant to emotion processing, and thus directly to ODD and ADs. These include the dorsolateral (associated with planning and higher-order cognitions); ventrolateral (associated with stimulus-response learning, generally activated when response modulation is required); and ventromedial (associated with anticipation of both positive and negative affective

consequences, subsumed within the orbitofrontal area) regions of the PFC (Adolphs, 2001; Blair, 2004, 2007; Davidson, 2002; Guyer et al., 2008; Monk et al., 2006).

Broadly, there are extensive reciprocal connections between the amygdala and PFC. Some of these connections (e.g., amygdala–ventrolateral PFC) form a circuit that detects personally and affectively salient stimuli and permits modulation of the amygdala’s response to environmental threats. Connectivity between the amygdala and ventromedial PFC has been implicated in processing of fearful expressions and extinction, and is important for guiding appropriate behavioral selection. Additional reciprocal connections with the dorsolateral PFC support selection, implementation, and monitoring of cognitive responses to threat and other socially salient stimuli, and thus further integration of emotion and cognition (Davidson, 2002; Guyer et al., 2008). Given these functions, disruptions in the limbic-PFC system may underlie the hypervigilance, attention to threat-related stimuli, and autonomic responses often observed among youth with ADs (Davidson, 2002; Guyer et al., 2008; Monk et al., 2006), and/or problems in representing reinforcement expectancies, which are critically important for inhibiting the aggressive and oppositional responses observed among youth with ODD (Blair, 2004, 2007; Crowe & Blair, 2008). However, multiple patterns of associations among limbic and PFC processes with ODD and ADs have been reported; thus, it is unlikely that there are specific patterns of limbic and PFC functioning associated with each ODD–AD subtype or pathway.

Several explanations suggest that deficits in limbic and/or PFC functioning may be associated specifically with ODD. First, underactive amygdala functioning could lead to difficulties in processing negative expressions or learning stimulus–incentive associations, which may contribute to ODD behaviors (Adolphs, 2001; Blair, 2004, 2007; Davidson, 2002). A second line of reasoning involves reactive aggression, which is modulated by the amygdala and ventromedial PFC (Blair, 2004, 2007) and is a frequent correlate of ODD behaviors (Bubier & Drabick, 2009). The ventromedial PFC is hypothesized to increase or decrease the probability of engaging in reactive aggression based on contextual cues (Blair, 2004). For example, if someone bumps into a child but an adult is nearby, the ventromedial PFC would be responsible for decreasing the probability that he or she would engage in reactive aggression (Bubier & Drabick, 2009). However, with an overactive amygdala or underactive ventromedial PFC, the child may be more likely to engage in reactive aggression. Additional support for PFC deficits among youth with ODD stems from reports of structural and functional abnormalities in the PFC among individuals with a history of aggression (Raine, 2002), and from findings that individuals with ODD exhibit deficits in executive functioning, including attention, concentration, planning, and inhibition of prepotent responses (Drabick, Gadow, & Loney, 2007; Seguin, Boulerice, Harden, Tremblay, & Pihl, 1999).

Several lines of evidence also suggest that the pathophysiology of ADs may involve dysfunction in limbic- PFC functioning. For example, relative to comparison participants, individuals with ADs (i.e., GAD, SOC, SAD) exhibit greater amygdala activation in response to angry and fearful faces, as well as greater orienting to and subsequent attentional bias away from angry faces (Monk et al., 2006; Thomas et al., 2001). In addition, youth with a primary diagnosis of GAD evidence greater right ventrolateral PFC activation in response to faces displaying negative emotions than comparison participants (Monk et al., 2006). When anticipating evaluation from peers, youth with ADs (GAD, SOC, SAD) evidence greater amygdala, as well as greater amygdala–ventrolateral PFC, activation than comparison adolescents (Guyer et al., 2008). Given that ventrolateral PFC activation is negatively associated with GAD symptom severity, Monk et al. (2006) suggested that ventrolateral PFC activation may serve a compensatory role in regulating abnormal functioning in another region (e.g., subcortical structures). Consistent with this possibility,

both amygdala–ventrolateral PFC co-activation (Guyer et al., 2008) and the magnitude of amygdala change from fearful to neutral faces (Thomas et al., 2001) are associated with anxiety symptom severity. These findings corroborate the possibility that the ventral PFC plays an important role in modulating the amygdala's response among youth with ADs. Taken together, findings suggest that heightened amygdala and/or attenuated PFC activity may lead youth with ADs (e.g., GAD, SOC, SAD) to misperceive neutral or ambiguous stimuli as threatening (Guyer et al., 2008). Thus, different patterns of limbic-PFC dysfunction are evident among youth with ODD or ADs, and models of comorbidity should consider these child-specific processes with contextual factors to provide a more complete framework for the dual ODD–AD pathway model.

Peer-Child Processes

The co-occurrence of ODD and ADs may, in part, be explained by social information processing models, which enumerate the components of children's cognitions that directly contribute to social behavior. One of the primary cognitive processes examined in relation to anxiety among children is hypervigilance to threat (Beck & Emery, 1985; Ingram & Kendall, 1987). Anxious children show an attentional bias to emotionally threatening stimuli and tend to make threatening interpretations when presented with ambiguous information and to attribute hostile intent to others' actions (Guyer et al., 2008; Monk et al., 2006; Puliafico & Kendall, 2006). Aggressive children, many of whom also meet diagnostic criteria for ODD, also exhibit information processing biases, such as encoding ambiguous cues as hostile, neglecting to encode nonhostile cues, making hostile attributions of intent, selecting dominance- and revenge-related goals, generating more maladaptive responses, exhibiting more confidence in their ability to enact such responses, and expecting that their aggressive behavior will result in a desirable outcome (Crick & Dodge, 1994; Crick & Ladd, 1990; Dodge & Frame, 1982; Dodge & Newman, 1981). Consistent with this broader literature involving aggression, preschool-aged boys with ODD have been found to generate more aggressive solutions and encode social information less accurately (Coy, Speltz, DeKlyen, & Jones, 2001). Thus, anxiety and aggression are associated with comparable biases across multiple stages of information processing, including hypervigilance for threat-related cues, negative interpretations of social situations, and hostile attributions (e.g., Reid, Salmon, & Lovibond, 2006); moreover, anxiety and ODD are associated with misinterpretations of social situations. These findings suggest that social information processing biases may underlie co-occurring ODD and ADs.

Similar biases are observed among reactively aggressive and anxious children as well. For instance, reactively aggressive children tend to interpret peers' behaviors as more hostile and to react with aggression more frequently when faced with ambiguous provocations compared to proactively aggressive children (Dodge & Coie, 1987). These emotionally reactive responses can be contrasted with evidence that proactively aggressive individuals do not necessarily exhibit poor performance on tests of frontal lobe functioning (Blair, 2004). Age-appropriate frontal lobe functioning may facilitate proactive aggression, as planning, understanding emotional processes, and regulating emotions could facilitate engaging in instrumental aggression without being detected by authority figures and recruiting others to conspire with them in aggressive acts (Deater-Deckard, 2001). In this way, consideration of the distinction between the social information processes specific to proactive and reactive aggression may be useful for distinguishing among ODD–AD subgroups.

Integrated Developmental Model

The evidence presented thus far, drawn from the broader conduct problem (ODD, CD, aggression) and internalizing (anxiety, depression) literatures, can be linked to provide

support for a dual ODD–AD pathway model (Table 1). Among youth in the first co-occurring ODD–AD group, we hypothesize that anxiety exacerbates ODD (i.e., multiple problem hypothesis; Garai et al., 2009; Lansford et al., 2008; Ollendick et al., 1999). Youth in this subgroup are expected to exhibit increased responsiveness in neural regions associated with threat responses (e.g., amygdala) and/or decreased responsiveness in the ventromedial and/or ventrolateral PFC. These difficulties would be expected to contribute to impaired ability of the ventromedial and/or ventrolateral PFC to regulate the limbic system's functioning and to relay information to the dorsolateral PFC, which could lead to problems in self-monitoring, emotion control, and behavioral selection. Given these neurobiological underpinnings, we hypothesize that this subgroup also will exhibit higher levels of negative emotionality, reactive aggression, and emotion dysregulation; difficulties with social–cognitive attributions; low effortful control; and poor executive functioning (e.g., planning, response inhibition). We also hypothesize that this subgroup would exhibit lower levels of interpersonal competence. Specifically, children who are emotionally dysregulated are more likely to exhibit anger, frustration, and irritability to requests and demands. Negative interactions with parents and peers can maintain and exacerbate emotionally dysregulated behaviors, as these negative interactions limit both children's exposure to appropriate emotion regulation strategies, as well as opportunities to model and engage in adaptive behaviors (Patterson, 1982; Patterson & Capaldi, 1990; Scaramella & Leve, 2004).

Among youth in the second co-occurring ODD–AD group, anxiety may mitigate ODD (i.e., buffering hypothesis; Hofmann et al., 2009; Walker et al., 1991). The expectation would be that this group will exhibit overactive limbic system functioning in response to angry or sad faces and higher levels of negative emotionality (e.g., fear/sadness). However, we hypothesize that age-appropriate PFC functioning and/or effortful control will serve to modulate the overactivity of the limbic system and the expression of the youth's negative emotionality. That is, this subgroup would evidence sufficient amygdala–PFC (ventrolateral, ventromedial) co-activation to modulate the limbic system activity, and reciprocal limbic system–PFC connections would facilitate more frequent selections of appropriate responses and self-monitoring, thereby contributing to lower levels of ODD behaviors than would be expected among youth in the other (multiple problem) subgroup. However, given hypothesized age-appropriate PFC functioning among youth with ODD, we also expect that youth in this subgroup would exhibit lower levels of reactive, but higher levels of proactive, aggression (Blair, 2004, 2007; Crowe & Blair, 2008).

IMPLICATIONS AND CONCLUSIONS

The within-category heterogeneity and high levels of comorbidity among psychological disorders greatly affect our understanding of the relations among potential risk processes and psychological disorders and contribute to the lack of clear evidence that diagnostic categories are discrete syndromes (Beauchaine, 2003; Drabick, 2009; Maser et al., 2009). Efforts to identify more homogeneous subgroups within and potentially across diagnostic categories may be fruitful. A dual ODD–AD pathway model, in which anxiety either mitigates (buffer hypothesis) or exacerbates (multiple problem hypothesis) ODD, may provide a useful framework for future research aimed at examining co-occurring ODD and ADs. Of course, the idea of considering correlates or risk processes associated with specific diagnostic syndromes is not new; indeed, such external validators (e.g., family history, risk factors, co-occurring symptoms, treatment response, diagnostic stability, course) have long been evaluated and often have been incorporated into *DSM* (Feighner et al., 1972; Kendler, 1990; Maser et al., 2009; Robins & Guze, 1970). Although the dual-pathway model addresses some of the heterogeneity associated with ODD and ADs, future research will be necessary to determine whether the proposed external validators (e.g., temperament, prefrontal and limbic processes, social information processing) for the ODD–AD subgroups

in fact are useful for differentiating these ODD–AD subtypes. Moreover, future research could determine whether additional external validators will support the construct validity of these subtypes (e.g., course, treatment response). Little research to date consistently has controlled for multiple comorbid conditions or used a developmental perspective for examining comorbidity. Thus, research that considers co-occurring ODD and ADs also should evaluate and potentially control for the effects of co-occurring ADHD and depression, test the specificity of this dualpathway model for different types of ADs (e.g., GAD vs. SOC), and examine whether the applicability of various comorbidity explanations differs based on the developmental period under consideration, type of AD, or additional co-occurring conditions. Such information could inform intervention efforts and subsequent versions of the *DSM*. For example, if future research supports the utility of these distinct ODD–AD subgroups, this information could be included in the *DSM* ODD and AD categories as subtypes of ODD or AD or both; alternatively, the co-occurring symptoms could be included as specifiers or dimensions to highlight potentially different correlates, courses, and/or treatment responses associated with co-occurring ODD and AD, consistent with efforts to include dimensions within the *DSM* framework (Drabick, 2009; Maser et al., 2009).

In addition to testing the utility of these candidate processes as external validators for the dual ODD–AD pathways, there are other potential directions for future research. We focused on correlates that may be best conceptualized as biologic and psychological factors (Kendler, 1990). However, given that individuals and their contexts have transactional and reciprocal effects on each other, it is clear that these candidate processes both influence and are influenced by contextual factors (Rutter & Sroufe, 2000). Future research that considers contextual factors and child $\times$ context interactions could be useful for establishing further construct validity of these potential ODD–AD subtypes, as well as whether these variables could be included as “associated features” in the *DSM*. In addition, the specificity of these external validators should be examined. For example, temperamental features are associated with a range of psychological disorders (Muris & Ollendick, 2005), but when combined with other risk processes (e.g., limbic system functioning), certain temperamental features may be more specific to ODD or AD. The applicability of these risk processes to other disorders that often co-occur with ODD (e.g., ADHD, CD) or ADs (e.g., mood disorders), as well as specificity to types of ADs, should be evaluated in future research as well.

To accomplish these goals, future research that concurrently examines the buffer hypothesis and the multiple problem hypothesis for co-occurring ODD and AD would be useful. Such research should assess for and incorporate potential co-occurring conditions and risk processes that may underlie, maintain, exacerbate, or buffer ODD and AD symptoms. Furthermore, many of these variables may serve as moderators of treatment and thus should be incorporated into prevention and intervention research. Developmentally sensitive inclusion of these variables is critical, given that some processes (e.g., prefrontal processes) are differentially amenable to intervention throughout childhood and adolescence and thus may have differential utility in moderating intervention outcomes. In sum, identification of relevant factors could lead to recognition of meaningful ODD–AD subgroups with distinct external validators, including course, treatment response, family history, concurrent symptoms, and biopsychosocial risk processes. Such knowledge can not only inform future versions of the nosological system, but also improve understanding of these challenging conditions and their co-occurrence, and thereby improve the lives of the individuals and families affected by them.

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Table 1

Two-subgroup model among youth with co-occurring ODD and anxiety disorders

Candidate process	Subgroup 1: Anxiety exacerbates ODD (Multiple problem hypothesis)	Subgroup 2: Anxiety mitigates ODD (Buffer hypothesis)
Negative emotionality	High anger/frustration Low fear	Low anger/frustration Moderate to high fear
Effortful control	Low levels	Age-appropriate levels
Aggression function	Reactive	Proactive
Limbic system	Overactive	Overactive but attenuated by PFC
Executive functioning	Poor	Age-appropriate
Social information processing	Poor	Age-appropriate; biases attenuated by PFC

Note: PFC = prefrontal cortex.