The role of anxiety in the development, maintenance, and treatment of childhood aggression

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Abstract

The majority of aggressive children exhibit symptoms of anxiety, yet none of our developmental models of aggression incorporate the role of anxiety, and our treatments ignore this comorbidity. This article outlines a novel theoretical model that specifies three hypotheses about comorbid anxious and aggressive children: (a) unpredictable parenting induces anxiety in children that in turn triggers aggressive behavior; (b) prolonged periods of anxiety deplete children's capacity to inhibit impulses and trigger bouts of aggression, and aggression in turn functions to regulate levels of anxiety; and (c) minor daily stressors give rise to anxiety while cognitive perseveration maintains anxious moods, increasingly disposing children to aggress. Little or no research has directly tested these hypotheses. Extant research and theory consistent with these claims are herein reviewed, and future research designs that can test them specifically are suggested. The clinical implications most relevant to the hypotheses are discussed, and to improve the efficacy of treatments for childhood aggression, it is proposed that anxiety may need to be the primary target of treatment.

A man who is not afraid is not aggressive, a man who has no sense of fear of any kind is really a free, a peaceful man.

Jiddu Krishnamurti

Anger and aggression are often linked together, but contemporary psychologists have largely overlooked the potential importance of an association between aggression and fear. The main premise of this article is that anxiety (a particular variant of fear) may be one of the key emotional underpinnings of childhood aggression and the relative neglect of anxiety in developmental and intervention theories may have led to an incomplete understanding of children's aggressive behavior.

Aggression is the most widely studied of all child behavior problems, and a wide range of treatment and prevention programs have been developed, yet prevalence rates and outcomes remain disturbing. Half of all referrals to children's mental health agencies are for oppositional or aggressive behavior problems (Patterson, Dishion, & Chamberlain, 1993; Stouthamer-Loeber, Loeber, & Thomas, 1992). Left untreated, aggression is highly persistent (Farrington, 1994; Jester et al., 2008; Loeber & Farrington, 2000; Temcheff et al., 2008; Tremblay, Pihl, Vitaro, & Dobkin, 1994) and predicts later delinquency, marital problems, depression, substance abuse, and severe difficulties in peer relations, academic functioning, occupational stability, and employment (Campbell, Spieker, Burchinal, Poe, & The NICHD Early Child

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Care Research Network, 2006; Katja & Pulkkinen, 2000; Loeber, 1988, 1990; Loeber, Burke, Mutchka, & Lahey, 2004; O'Donnel et al., 2006; Stattin & Magnusson, 1989; Stipek & Miles, 2008). Childhood aggression not only is detrimental to the child but also has a significant impact on the well-being of the victims, and the public costs associated with violence are enormous (e.g., to mental health institutions, juvenile justice systems, schools; Krug, Mercy, Dahlberg, & Zwi, 2002). Some progress has been made in identifying treatments that are effective in decreasing childhood aggression. However, despite their popularity, they remain only moderately effective (Connor, 2002; Kazdin, 1987, 2001a, 2002; Weisz, Doss, & Hawley, 2005). One of the main reasons for these modest effects may be that the vast majority of aggressive children exhibit co-occurring, clinically elevated, anxiety symptoms; yet, anxiety is neglected in contemporary developmental theories of aggression, and it is largely ignored in intervention models.

Anxiety is a biologically basic emotion that arises in situations that are ambiguous, potentially threatening and unpredictable (Darwin, 1872; Fridja, 1986; Izard, 1991). A great deal of research has focused on anxiety and its influence on inhibition and withdrawal (e.g., Gray, 1982; Gray & McNaughton, 2000), but almost no studies focus on anxiety's influence on children's aggressive behavior. The main objective of the current paper is to introduce a developmental and clinical model of the role of anxiety in children's aggression and suggest research strategies to test the model. Three new hypotheses emerge from the model:

1. Early unpredictable parenting induces anxiety in children that in turn generates aggressive behavior.

- Prolonged periods of anxiety in real time (from moment to moment) deplete children's capacity to inhibit their impulses and trigger bouts of aggression as a result; aggression in turn functions to regulate, or decrease, initial levels of anxiety.
- Minor daily stressors give rise to anxiety, but cognitive perseveration (worrying and rumination) maintains anxious moods, predisposing children to aggress.

These hypotheses remain largely untested at this point; thus, the two main goals of the current paper are to review extant research that is consistent with the model and to suggest research designs and methodologies that can test it more specifically. Some considerations for clinical practice that follow directly from the proposed model are presented. It is argued that in order to most effectively decrease children's aggressive behavior, treatment programs need to target anxiety rather than focus solely on the aggression itself.

Subtypes of Aggression

My contention that anxiety is critical for understanding aggressive behavior is meant to apply to the majority of aggressive children and youth, but there are categories of aggressive behavior to which it does not apply. Several classification systems have been proposed, but one of the most widely used is the distinction between reactive and proactive aggression. Proactive aggression is instrumental, goal-directed, and often premeditated, whereas reactive aggression is hostile, retaliatory, and "hot-blooded" (Feshbach, 1970; Dodge, 1991; Dodge & Coie, 1987; Hinshaw & Zupan, 1997). A recent meta-analysis by Polman, de Castro, Koops, van Boxtel, and Merk (2007) demonstrated the extent to which these forms of aggression are overlapping. Correlations between measures of reactive and proactive aggression ran as high as r = .87, suggesting that there is no clear distinction and/or many children exhibit both types of aggression.

In this review, I am concerned with modeling the emergence and stabilization of reactive aggression, by far the most prevalent type of aggression (Feshbach, 1970; Dodge, 1991; Dodge & Coie, 1987; Hinshaw & Zupan, 1997; see review by Bubier & Drabnick, 2009). This kind of aggression has been described as defensive in function and is often said to be accompanied by fear, anger, or frustration (Merk, de Castro, Koops, & Matthys, 2010; Scarpa, Haden, & Tanaka, 2010; Vitaro, Brendgan, & Barker, 2006). However, these negative emotions should not be assumed to function in a similar manner. Although reactive aggression may result from frustration alone, I argue that anxiety is a key causal engine in most acts of reactive aggression. Past research and theory on reactive aggression has largely remained at the descriptive or nosological level, at least when it comes to specifying the emotional underpinnings of aggression. My intention is to go further and model the precise mechanisms by which anxiety (the emotion, not the disorder) triggers aggression, how anxious moods heighten the probability of aggression, and the implications for prevention and treatment.

As noted, reactive aggression may be in response to frustration, not anxiety; this subcategory of aggressive behavior will not be addressed in this review. In addition, there is a very small proportion of children who exhibit instrumental or proactive aggression exclusively; these children cold-bloodedly premeditate acts of aggression and consider plans and goals before acting out. These children may be "fledgling psychopaths" (Lynam, 1996), and they too are not addressed by the current modeling.

Comorbidity in Aggressive Children

In order to make the case for the importance of anxiety for aggressive children, we need to establish that many clinically aggressive children experience problematic anxiety in the first place. One way to do so is to examine the clinical literature on children's behavior disorders. Aggressive children are often comorbid for anxiety problems (DSM-IV-TR; Fleitlich-Bilyk & Goodman, 2004; Ford, Goodman, & Meltzer, 2003; Greene et al., 2002; Marmorstein, 2007; Oland & Shaw, 2005; Shields & Cicchetti, 2001; Zoccolillo, 1992). Rates of anxiety disorders in conduct-disordered children range from 22% to 33% in community samples and 60% to 75% in clinic-referred samples (Boylan, Vaillancourt, Boyle, & Szatmari, 2007; Russo & Beidel, 1993; Zoccolillo, 1992). These are probably underestimates given that, generally, adults and peers pay more attention to children's disruptive, violent behavior than to their distress (e.g., Luby, Belden, Sullivan, & Spitznagel, 2007; Stallings & March, 1995), and adults are less likely to recognize internalizing symptoms like depressive affect in children with externalizing problems such as aggression (Achenbach, McConaughy, & Howell, 1987; De Los Reyes & Kazdin, 2005; Kolko & Kazdin, 1993; Muris, Merckelbach, Ollendick, King, & Bogie, 2002; Wu et al., 1999).

In our own research program, with three clinic-referred samples, 75% to 85% of aggressive children and adolescents also showed anxiety problems (Lewis et al., 2008; Granic, Meusel, Woltering, Lamm, & Lewis, 2012; Woltering, Granic, Lamm, & Lewis, 2011). These high rates of co-occurrence made it useful to distinguish children who exhibit "pure" aggressive behavior, with no anxiety symptoms (AGG), from children who show elevated symptoms of both aggression and anxiety (AGG/ANX). Past findings have demonstrated that AGG children compared to AGG/ANX children exhibit distinct parent-child interactions (Granic & Lamey, 2002) and unique brain activation patterns associated with emotion regulation (Stieben et al., 2007; Lamm, Granic, Zelazo, & Lewis, 2011). These subtypes in turn are associated with diverse treatment outcomes (Beauchaine, Gartner & Hagen, 2000; Beauchaine, Webster-Stratton & Reid, 2005; Costin & Chambers, 2007; Kazdin & Whitley, 2006).

Longitudinal studies of anxious and aggressive symptoms and disorders

The most common way that researchers have addressed a possible causal link between anxiety and aggression is through

longitudinal research that attempts to delineate pathways of these problem behaviors. This body of research focuses on comorbidity issues and the extent to which one set of symptoms, or a disorder, may act as risk factors for the other. To summarize this line of research (for a review, see Bubier & Drabnick, 2009), there is some evidence that anxiety disorders (e.g., separation anxiety, social phobia) precede, and may act as risk factors for, the emergence and/or maintenance of aggressive behavior disorders (e.g., conduct disorder, oppositional defiant disorder; Bittner et al., 2007; Ialongo, Edelsohn, Werthamer-Larsson, Crockett, & Kellam, 1994; Last, Perrin, Hersen, & Kazdin, 1996; Vittaro, Brendgen, & Tremblay, 2002). In contrast, there are also several studies that have documented the opposite causal direction (Burke, Loeber, Lahey, & Rathouz, 2005; Lahey, Loeber, Burke, Rathouz, & McBurnett, 2002; McBurnett et al., 1991; Speltz, McClellan, DeKlyen, & Jones, 1999). The problem with many of these studies is that they only test one hypothesized causal direction and causal relations may change with development (Cicchetti & Toth, 1991). However, even if we were to show that anxiety disorders precede aggressive behavior disorders in development or vice versa, the causal story would still be missing. Anxiety and aggression could result from a third factor or from entirely independent factors. Moreover, subclinical anxious feelings may still fuel aggressive tendencies even if there is no diagnosis of anxiety. What we do know is that, by the time children have been referred for treatment for their aggression, the vast majority of them also have serious problems with anxiety (e.g., Lewis et al., 2008; Stieben et al., 2007; Woltering et al., 2011).

Given the body of research that has examined the timing of and relations between anxiety and aggressive behavior disorders and symptomotology, it may seem that I have overstated the paucity of research on the role of anxiety in aggressive behavior. However, none of the studies I reviewed addressed the *mechanisms* by which anxiety and aggression become linked in real time and over development. Instead, the studies reviewed thus far are correlational or descriptive in nature, even when they are longitudinal. By strictly focusing on the relation between clusters of symptoms from one time point to another, researchers make no attempt to explain how anxiety may fuel and maintain aggressive tendencies moment to moment and what the developmental consequences might be. The current review attempts to model these explanatory mechanisms on a fine-grained scale.

In the following section, I lay out three hypotheses that serve as cornerstones to a model of the causal role of anxiety in aggressive behavior problems, both over development and within situations. For each hypothesis, the extant research and relevant theory is reviewed, novel theoretical extensions are offered, and new studies that directly address these claims are proposed for future work.

Hypothesis 1

Early unpredictable parenting induces anxiety in children, which then gives rise to aggressive behavior.

Extant research and relevant theory

Psychoanalytic and attachment approaches. Freud (1926/ 1959) was the first to theorize about the developmental impact of anxiety: when young infants feel anxious, they instinctually reach for their mothers for soothing and comfort. A mother who is temporarily out of reach, or even the expectation that the mother may become unreachable at some point, induces intense feelings of anxiety; so does the possibility of punishment coming from either parent at a slightly later stage. For Freud, it was not the behavior of the parent per se that induces anxiety, it was the anticipation of that behavior and its fundamental unpredictability (Freud, 1926/1964). Klein (1948) picked up on Freud's notions of anxiety and explicitly linked them to aggression. For Klein, anxiety came in two types: one was anxiety about the loss of the mother and the other was fear of retribution. Both sources of anxiety are defended against by means of aggression, which may further induce anxiety in anticipation of retribution. Although these psychoanalytic models are compelling, they have remained outside the arena of empirical testing, with the possible exception of Margaret Mahler's work in the 1970s.

Mahler, Pine, and Bergman (1975) demonstrated how young children, especially around the age of 18 months, peaked in their feelings of anxiety when the mother was perceived as inaccessible. This "crisis" phase heralded a flowering of social intelligence. Along with separation anxiety, Mahler also observed the concomitant onset of temper tantrums and rage attacks. It is interesting that the age at which Mahler documented peaks in anxiety corresponds precisely with well-established normative peaks in physical aggression (just before 2 years; Tremblay et al., 1999). Thus, anxiety and aggression seem to become pronounced emotional experiences in early childhood, and they may become linked by the end of this period.

Attachment theory is thought to be the most successful spinoff of psychoanalytic theory. Thus, it is not surprising that anxiety also plays a central role in the attachment framework; the original two insecure attachment styles were labeled anxious-avoidant and anxious-ambivalent (Ainsworth, Blehan, Waters, & Wall, 1978). Attachment styles are thought to emerge in reference to the sensitivity with which primary caregivers respond to their children (e.g., Bowlby, 1969; Mikulincer & Shaver, 2007; van IJzendoorn, Schuengel, & Bakermans-Kranenburg, 1999). For children who develop insecure attachment styles, early experiences with a mother who is either unavailable or unpredictable give rise to anxiety. Both anxious-insecure attachment styles have been empirically linked to the development of aggressive and/or delinquent behavior (Allen, Porter, McFarland, McElhaney, & Marsh, 2007; Greenberg, Speltz, DeKlyen, & Jones, 2001; Spelz et al., 1999); however, none of these studies actually measured anxiety per se in these children (they were simply classified as one of the two insecure subtypes). Although these attachment studies did not test the link directly, the results suggest that the anxiety that underlies the development of insecure attachment styles may also provide the foundation for subsequent problems with aggression. Thus, from the psychoanalytic perspective as well as a more contemporary attachment theory approach, anxiety may be an important causal engine that emerges in infancy and early childhood and elicits aggressive feelings or actions that may stabilize into aggressive personalities over time.

Behavioral approaches. From the behavioral research on parent-child interactions, there are particular interaction patterns that have been repeatedly implicated in the development of childhood aggression (e.g., Dumas & LaFreniere, 1993; Patterson, 1982; Patterson, Reid, & Dishion, 1992; Snyder & Patterson, 1995) or anxiety (Barrett, Dadds, & Rapee, 1996; Dadds, Barrett, Rapee, & Ryan, 1996; Donenberg & Weisz, 1997). A large body of research has shown that when children use aversive interaction tactics (e.g., coercion, tantrums) to avoid complying with parental demands, and the parent acquiesces repeatedly, the child learns to use aggression more often (for reviews, see Hill & Maughan, 2001; Hinshaw, 2002; Kazdin, 2002; Moffitt, 1993) and the parent becomes more and more permissive to avoid conflict (for a review, see Granic & Patterson, 2006; Patterson, 1986; Snyder, Edwards, McGraw, Kilsgore, & Holton, 1994). When children experience this permissive parenting exclusively, they are likely to develop AGG problems, with no anxiety-related issues (Granic & Patterson, 2006). In contrast, parents of pure AGG children are often overcontrolling and use hostile, retaliatory behavior to quash oppositional behavior (Dumas, LaFreniere, & Serketich, 1995; Sigueland et al., 1996). However, children with parents who combine permissive and hostile/controlling methods tend to raise children who exhibit AGG/ANX tendencies (Granic & Lamey, 2002; Granic & Patterson, 2006). Other researchers have described a related parenting style as inconsistent or indiscriminant parenting (Dumas & LaFreniere, 1993; LaFreniere & Dumas, 1995).

As noted earlier, the distinction between the parent–child interactions of pure AGG versus AGG/ANX children has some correlational support (Granic & Lamey, 2002; Grimbos & Granic, 2009; Sanders, Dadds, Johnston, & Cash, 1992). However, developmental studies that identify the causal pathways that connect these parenting behaviors with AGG/ANX symptoms have yet to be conducted. Furthermore, the switch back and forth from permissive to hostile parenting behaviors makes it impossible for children to predict their parents' actions. Thus, consistent with the psychoanalytic and attachment principles outlined above, unpredictability may capture a key feature of interaction styles that lead to childhood anxiety (Granic & Patterson, 2006). Unpredictable threat is inherently linked to anxiety in humans and other animals (Darwin, 1872; Fridja, 1986; Izard, 1991). Feelings of anxiety may in turn give rise to aggressive behavior.

Although psychoanalytic/attachment and behavioral approaches have suggested a link between unpredictable parenting, anxiety, and aggression, empirical support is either missing or flawed: (a) studies generally use questionnaire methods

to ask participants how consistently they parent (Brody et al., 2001, 2003; Laird, Pettit, Dodge, & Bates, 2003), whereas it is widely acknowledged that observational methods are optimal for measuring parent-child behaviors most relevant to the development of anxiety (e.g., Gonzalez, Moore, Garcia, Thienemann, & Huffman, 2011; Hawes & Dadds, 2006; McLeod, Wood, & Weisz, 2007; Wood, McLeod, Sigman, Wei-Chin, & Chu, 2003) and aggression (e.g., Dumas & LaFreniere, 1993; LaFreniere & Dumas, 1995; Patterson, 1982; Patterson et al.,1992; Snyder & Patterson, 1995); (b) when observations are collected, studies rarely compare two or more observational sessions (e.g., the Strange Situation studies in the attachment literature; van IJzendoorn et al., 1999) so predictability across episodes cannot be measured; (c) most often, global ratings instead of real-time measures are used to quantify observations of parenting. However, global ratings are poor indices of predictability because predictability is inherently a timebased concept; it is important to establish how consistently a parent reacts toward a child from moment to moment, whether those real-time patterns are different across contexts, and whether they remain consistent over development. Thus, assessing predictability requires process-level research designs and measures (e.g., Granic & Hollenstein, 2003, 2006).

Pulling together the theoretical insights from psychoanalytic and attachment approaches and linking them to the behavioral extant research on parent-child relations, I propose a novel developmental model that links anxiety and aggression. We have shown that anxiety and aggression problems co-occur at high rates in children, suggesting some causal link. For those children who are comorbid for anxiety and aggression problems, aggression in real time may emerge in response to triggers of anxiety present since early parent-child interactions. This anxiety may be about anticipating hostile retaliation from the parent when they have been "bad." However, children's anxiety is also about the difficulty of predicting how the parent will react in the first place. Figure 1 represents the current model at the developmental scale. I have reviewed behavioral research showing that "purely" aggressive children seem to have parents that are overly permissive and "purely" anxious children have hostile and overcontrolling parents. I propose that children who are both anxious and aggressive have parents who are sometimes permissive (reinforcing aggression) and sometimes hostile/overcontrolling (triggering anxiety) in response to the same type of (mis)behavior. The day-to-day unpredictability of these parental reactions may not only exacerbate anxiety but also link it with aggressive behaviors reinforced by parental permissiveness. The importance of parental unpredictability in the development of childhood anxiety and aggression has been pointed to by others, yet very little explicit modeling and almost no empirical research directly tests this assumption.

Directions for novel research

Given the diversity of theoretical avenues that have placed emphasis on unpredictability, it is crucial to establish the ex-

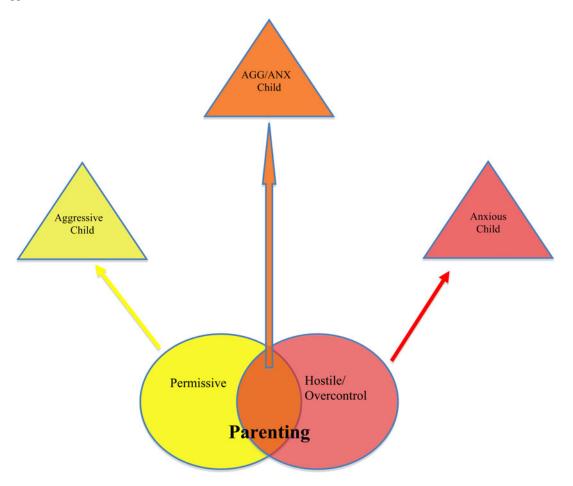


Figure 1. (Color online) Unpredictable oscillations between permissiveness and hostile/overcontrolling parenting leads to the development of co-occurring anxiety and aggression problems.

tent to which unpredictable parenting may contribute to the early onset and amplification of anxiety and subsequent aggression. Observational and longitudinal designs with nonclinical but at-risk children may be particularly powerful to track the emergence of AGG/ANX children's psychopathology. Parents and children can be videotaped in their homes, for example, from as early as 3 years of age, every 6 months for several years, to track the emergence of children's comorbid symptomotology.

Measuring predictability meaningfully requires not only observational methods but also a process-level analytic approach that can tap changes from moment to moment in parent—child interactions. How these moment-to-moment patterns change over the course of development is also critical for understanding the impact of unpredictability. Predictability can be assessed within an interaction episode (e.g., how consistently does a parent communicate her disapproval following noncompliant behaviors during one particular discussion) and it can also be assessed across interaction episodes (e.g., how much does that level of consistency change over the course of 1 year).

Dynamic systems methods have been particularly successful at addressing predictability in child and family systems

(e.g., Dishion, Nelson, Winter, & Bullock, 2004; Granic & Hollenstein, 2003, 2006; Granic, Hollenstein, Dishion, & Patterson, 2003; Hollenstein, Granic, Stoolmiller, & Snyder, 2004; Lewis, Lamey & Douglas, 1999; Lickwarck-Aschoff, Hasselman, Cox, Pepler, & Granic, 2012; Lunkenheimer, Olson, Hollenstein, Sameroff, & Winter, 2011). These methodologies allow researchers to examine several coexisting interaction patterns and explore movement from one to the other in real time (Granic & Hollenstein, 2003, 2006; Lewis et al., 1999). This movement can be quantified and the level of predictability over time can be established empirically through process-level indices.

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A concrete example may prove useful. Figure 2 shows a state space grid (Lewis et al., 1999), a dynamic systems method we have previously used to represent and measure parent–child behavior in real and developmental time (Granic et al., 2003; Granic, O'Hara, Pepler, & Lewis, 2007; Hollenstein et al., 2004). Parent–child interactions that are video recorded every 6 months (for example) over the course of several years, can be subsequently coded for affective behavior. It would be ideal to use a real-time coding system. For instance, nine common affective codes that can capture parent–child interactions are shown in Figure 2; the parent's

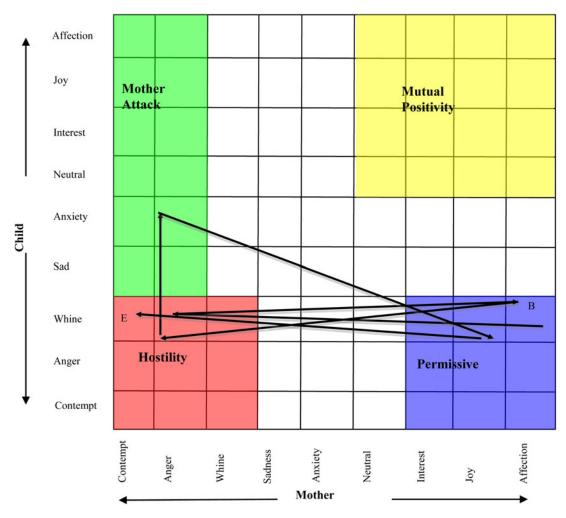


Figure 2. (Color online) State space with the four regions (highlighted) representing dyadic patterns of interest. The trajectory shown is an interaction pattern that is expected to characterize aggressive—anxious (AGG/ANX) children. The "B" cell is where the dyad begins, and the "E" cell is where it ends. Mother is highly unpredictable (e.g., there are many transitions between cells for her) and she moves back and forth from affectionate/joyful to angry/contemptuous in response to the same child behavior (i.e., whining).

behavior is tracked on the *x* axis and the child's behavior on the *y* axis. State space grids can be constructed for all dyadic observations at each assessment wave separately.

With this methodology, the dyad's trajectory (i.e., the sequence of codes) is plotted on a grid of cells. To test my proposed model, four grid regions are of particular interest, representing different styles of parent—child interactions (see Figure 2). The total duration in each region can be computed with the expectation that the two bottom regions (permissiveness and hostility) will be occupied for longer durations for children who eventually develop AGG/ANX problems. In addition, at least three measures of unpredictability can be derived from the grids: entropy, number of transitions, and dispersion.

Because it would be particularly important to establish the predictability of mother's responses to the same child affective behavior (e.g., whining), movement between "mutual hostility" and "permissive" can be quantified for analyses. These indices of predictability can then be combined with

macromeasures of anxiety and aggression (e.g., using the Child Behavior Checklist; Achenbach, 1991) to run statistical models that can differentiate trajectories of change, measured across the longitudinal waves. Mediational analyses can be conducted within this framework such that, for example, maternal unpredictability at Wave 1 predicts anxiety at Waves 2 and 3 and aggression at Waves 4 and 5. Thus, the proposed model of parenting precursors to the development of the AGG/ANX profile can be directly tested. Moreover, observations coded in real time would allow the sequential tracking, on a fine-grained level, of children's expression of anxiety and aggression patterns during episodes characterized by heightened parental unpredictability.

Testing the proposed model in Figure 1 seems like an important first step for establishing the parenting factors that give rise to the development of co-occurring childhood aggression and anxiety. A dynamic systems approach holds particular promise, given that it provides a means by which predictability can be quantified in both real and develop-

mental time. However, the dynamic systems approach in developmental psychology is still primarily a descriptive one, and the proposed method of testing the model does not provide a precise mechanism by which anxiety links up with aggression intraindividually. To establish such a mechanism, we need to study the instances during which anxiety is triggered within an individual and the conditions under which it does or does not lead to aggression in real time.

Hypothesis 2

Prolonged periods of anxiety deplete children's capacity to inhibit impulses and trigger bouts of aggression that in turn function to regulate initial anxiety levels.

Extant research and relevant theory

Given the high prevalence rates of co-occurring anxiety and aggression, why has so little research attempted to understand the precise mechanisms by which feelings of anxiety and the tendency to act out aggressively are linked? One reason may be that the association is simply counterintuitive: If a child is fearful, one might think that he is *less* likely to lash out and aggress. Individuals with antisocial and aggressive personalities sometimes lack fearful inhibition (Gray, 1982) and show low trait anxiety (Cleckley, 1982); anxiety inhibits aggressive behavior in some contexts (e.g., Ferreira, Hansen, Nielsen, Archer, & Minor, 1989; Gray, 1987; Hard & Hansen, 1985). However, in two recent reviews, one on rodents (Neumann, Veenema, & Beiderbeck, 2010) and the other on clinical children (Bubier & Drabnick, 2009), anxiety and aggression were shown to co-occur more often than not. This seems paradoxical: How can anxiety inhibit aggressive impulses while also being associated with higher levels of aggression? It may be difficult to reconcile these contradictory findings unless we examine more closely the real-time causal relations between anxiety and aggression.

The research on emotion and emotion regulation in developmental psychopathology and neuroscience provides some hints as to the processes by which anxiety links with aggression for some children. When children are clinically anxious, they have learned to anticipate threat in neutral circumstances (Pine, 2003). As a result they become overvigilant concerning potential threats, especially in challenging circumstances (Eisenberg, Hofer, & Vaughn, 2007; Eisenberg & Morris, 2003; Salters-Pedneault, Roemer, Tull, Rucker, & Mennin, 2006). They tend to amplify their fears by focusing on stress-induc-

ing stimuli rather than recruiting a flexible repertoire of regulation strategies (e.g., problem solving; Bradley, 2000).

Children who perceive their environment as aversive or threatening will normally tend to withdraw or avoid the source of threat (Amstadter, 2008), but in other cases, aggression emerges by way of one of two routes: one direct (the top arrow, Figure 3) and one indirect, through loss of inhibitory control, or ego depletion. The direct route is consistent with Gray's updated model (1994), which posits a neural subsystem that underpins fight or flight responses and defensive aggression. In a recent empirical review of neuroscientific evidence, Potegal and Stemmler (2010) argued that the medial hypothalamus and periaqueductal gray most likely underlie defensive aggression (see also Adams, 2006; Siegel, 2004; Siegel, Roeling, Gregg, & Kruk, 1999). They further proposed that defensive aggression is fear driven, based on the anatomy of these circuits. Thus, there is some evidence on the neural level that "hot," reactive aggression is specifically triggered by anxiety-driven circuitry.

The notion of a direct route from anxious feelings to aggression is based on animal models (e.g., Potegal & Stemmler, 2010). However, because humans are routinely engaged in some level of inhibitory control (Carlson & Wang, 2007; Fox & Calkins, 2003), these models are no doubt incomplete. I propose that an indirect route from anxiety to aggression works through the loss of inhibitory control, a process akin to Baumeister and colleagues' ego depletion (Baumeister, Bratslavsky, Muraven, & Tice, 1998; Baumeister & Heatherton, 1996; Baumeister, Vohs, & Tice, 2007). Their work shows that the biological substrates of self-control become diminished or used up within minutes (for a review, see Heatherton & Wagner, 2011). It may be that anxiety at first serves to inhibit aggression (Gray, 1987, 1994; Salters-Pedneault, et al., 2006), but over time, it may lead to the disinhibition of aggressive behavior through ego depletion.

Longitudinal and correlational studies that tap global constructs of anxiety and aggression are unlikely to disentangle these dual effects of anxiety on aggression. To address this gap, models need to be developed and commensurate studies need to be conducted that focus on the mechanisms by which anxiety and aggression influence each other in the lived experiences of children. Toward this goal, Figure 3 presents a conceptual model for the real-time link between anxiety and aggression for AGG/ANX children. The most important contribution of this model is the role of ego depletion, which helps to reconcile the seemingly paradoxical effects of anxiety on the tendency to aggress. At first, anxiety may serve to inhibit impulsive acts of reactive aggression. However, sus-

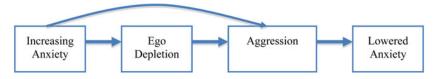


Figure 3. (Color online) Hypothesized real-time model linking anxiety to aggression.

taining inhibitory control while attending to potential threats (e.g., unpredictable parent hostility) may lead to ego depletion, causing AGG/ANX children to eventually "snap" and aggress against perceived sources of threat.

Moreover, consistent with early psychoanalytic thinking (c.f., Greenberg et al., 2001; Spelz et al., 1999), I propose that, for AGG/ANX children, aggression itself is a regulatory response that decreases anxiety because it increases a sense of power and efficacy. Disinhibition in general (letting go of control) may often paradoxically induce feelings of power (Hirsh, Galinsky, & Zhong, 2011; Lewis, 2011). Thus, aggression may become highly useful and rewarding for these children because it works; it makes them feel better.

Directions for novel research

The proposed real-time model (Figure 3) is a novel one and needs to be systematically tested with studies that induce anxiety, measure inhibitory control (and its loss through ego depletion), and track levels of aggression over time. In animal studies (mice in particular), anxiety levels have repeatedly been shown to positively covary with aggression levels (e.g., Bosch, Meddle, Beiderbeck, Douglas, & Neumann, 2005; Nyberg, Vekovischeva, & Sandnabba, 2003). Human studies that experimentally induce increasing levels of anxiety and then allow an opportunity for aggression would be informative in this regard.

For example, AGG/ANX children and a comparison group could be run through an anxiety-induction go/no-go procedure (e.g., Lewis et al., 2008; Stieben et al., 2007; Woltering et al., 2011). As with many of these paradigms, children will need to click a button (i.e., go) for each letter or picture presented onscreen but avoid clicking (i.e., no-go) when an infrequent alternative letter or picture is presented. No-go trials require participants to withhold a prepotent response and thus tap inhibitory control mechanisms (Simpson & Riggs, 2005). Because ego depletion can be conceptualized as the loss of inhibitory control, the errors in the no-go trials can be used as a marker for ego depletion.

To induce anxiety the task can be rigged such that points are gained in an initial block of trials then drop to near zero while the opponent's points rise in an anxiety-induction phase. Because anxiety manifests in sympathetic arousal, heart rate can serve as an index for anxiety levels throughout the task. An opportunity to aggress may then be introduced during the task such that the game stops and participants' accumulated points are displayed, perhaps in contrast with a fictitious opponent. Then participants can be allowed to administer a blast of noise at this fictitious opponent, for example, by using the Taylor Aggression Paradigm (Taylor, 1967). This task allows participants to react to their loss of points by blasting their opponent with a loud noise, varying in intensity from "not painful at all" to "extremely painful."

Thus, with this type of set-up, the extent to which anxiety levels just prior to the opportunity to aggress predict levels of subsequent aggression can be examined. Accelerated error

rates over the course of the task can serve as a measure of ego depletion and should predict increases in the intensity of aggression for the AGG/ANX group but not for a normal control group. Furthermore, one can adjust the design to take away the opportunity to aggress. If aggression functions to regulate anxiety as hypothesized, then heart rate should increase during the emotion-induction block and remain high for AGG/ANX children when they have no opportunity to aggress.

The kind of paradigm that I have just described assumes that errors on the no-go trials are due to increasing anxiety that brings the child closer and closer to becoming depleted. However, differences in these error rates can also be more simply conceptualized as indexing individual differences in trait levels of inhibitory control. The errors themselves do not distinguish between the real-time process of decreasing inhibition and trait levels of disinhibition. It is clear that lower trait levels of inhibition do distinguish aggressive children from their typically developing peers (e.g., Nigg, 2000); to more rigorously test the real-time model, the process (loss of inhibition) and the trait (lack of inhibition) would need to be distinguished by baseline measures tapping inhibitory control. These levels could be taken as moderators or used as a means by which children are grouped and compared.

Study designs can also do more than systematically increase anxiety levels. For example, evidence for the role of anxiety might also include oxytocin administration studies. Oxytocin is known to decrease anxiety (Huber, Veinante, & Stoop, 2005). Within-subject ABA designs that induce anxiety, provide conditions to aggress, then administer oxytocin to decrease anxiety, and again assess aggressive behavior, would provide one possible test of the link between anxiety and aggression.

Hypothesis 3

Minor daily stressors give rise to anxiety while cognitive perseveration maintains anxious moods, both disposing children toward aggression.

Extant research and relevant theory

Anxiety often does not diminish in a few minutes; it can extend into anxious moods that persist for hours and even days. If the previous arguments are valid, then these moods should further predispose AGG/ANX children to aggress against perceived threats. There is no research on the factors that trigger and subsequently maintain aggressive children's anxious moods. The literature on stress, however, is helpful. In particular, minor, everyday stressors seem to play a critical role in the development of psychopathology, perhaps even more than do traumatic events (e.g., Cohn et al., 2009). The impact of daily life stressors on adult psychopathology is undisputed (e.g., Myin-Germeys et al., 2009; Wichers et al., 2010). However, far less is known about how everyday stressors (e.g., not being picked to be on a team, being teased in the playground,

getting a bad grade) affect children's development. If several daily stressors cluster closely in time, children may become locked into attending to ongoing threats, perpetuating their anxious moods over hours. However, most children experience daily stressors without developing AGG/ANX problems. Thus, an additional mediating mechanism is necessary that maintains and amplifies anxious moods: cognitive perseveration (e.g., worrying, ruminating).

Research on adult anxiety disorders and cardiovascular health suggests that cognitive perseveration is a key mechanism by which individuals maintain and amplify anxious moods (Brosschot, 2010; Pieper, Brosschot, van der Leeden, & Thayer, 2010; Verkuil, Brosschot, Gebhardt, & Thayer, in press). AGG/ANX children may not only attend to actual threatening cues in the form of daily life stressors but may also excessively anticipate, worry about, and ruminate over the impact of these threats, even when they are in a safe context. Thus, these children "carry" their anxiety-fueled threat distortions with them into contexts that may seem banal or even safe to others around them.

Consistent with social information processing accounts of childhood aggression (e.g., Dodge, 1980; Dodge, Price, Bachorowski, & Newman, 1990; Dodge & Somberg, 1987), the daily stressors that may be most salient for aggressive children are social in nature. Aggressive children repeatedly have been shown to attribute hostile intentions to peers in ambiguous (Aydin & Markova, 1979; Dodge, 1980), and even positive, prosocial circumstances (Dodge, Murphy, & Buchsbaum, 1984). The emotional underpinnings of these hostile attributions are still unknown, but negative affect has been suggested as one mechanism and anxiety may be that affect (Dodge & Somberg, 1987).

In addition to focusing and perseverating on current or anticipated hostile interactions, much of AGG/ANX children's cognitive perseveration may be allocated toward maintaining inhibitory control (e.g., "That guy is looking at me weird . . . I should just ignore him"). Consistent with the real-time model

presented in Figure 3, it may be that after hours of attempting to inhibit recurrent aggressive impulses, these children finally lose self-control (become ego depleted) and subsequently aggress (e.g., "I can't handle this anymore! I'm going to wipe that grin off him!"). This model suggests that children may act out aggressively later in the day and later in the week. Their capacity to inhibit impulses may wane as they are repeatedly challenged with threatening stimuli during the day or as they experience clusters of daily stressors over the course of a week. Figure 4 summarizes the impact of daily life stressors on aggressive behavior via anxious moods that are maintained by cognitive perseveration.

Directions for novel research

Part of the reason why we know so little about how anxious moods affect children's aggressive behavior has to do with methodological limitations inherent in most child-focused research. To understand how moods influence behavior, detailed information about emotions, their durations, and the context in which they emerge is necessary. Process-level methods are needed, but observational methods are difficult to employ for these purposes because researchers cannot videotape children throughout the whole of their day, across their varied social contexts (e.g., at home, in the playground, in the classroom). Questionnaires pose even greater challenges because asking individuals to report on their moods retrospectively introduces systematic biases due to current emotional states and recall failures. Moreover, research on child psychopathology most often relies on reports from parents and teachers, distancing the phenomena of lived emotional experiences even further from the source.

One way to address these limitations is to apply experiencesampling methods (ESMs). ESMs involve contacting participants several times every day, over the course of several days or weeks, to answer a brief set of questions about their current emotions, thoughts, and context (usually on a smartphone or

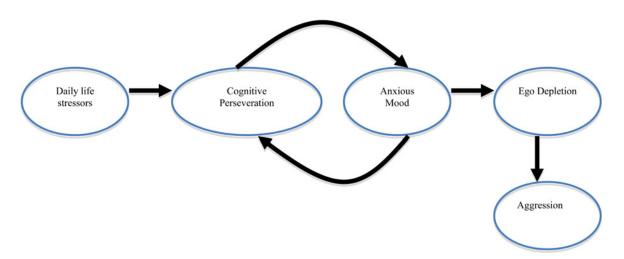


Figure 4. (Color online) The role of daily life stressors and cognitive perseveration on anxious moods and subsequent aggression.

similar portable electronic device; e.g., Larson & Lampman-Petraitis, 1989; Silk et al., 2010). Participants can report what they are feeling that very moment, who they are with, and what they are doing; thus, retrospective biases are eliminated, detailed sampling of individuals' emotional lives is collected, and the data are highly ecologically valid (Myin-Germeys et al., 2009; Silk et al., 2010; Wichers et al., 2010).

ESM designs can be employed to test the relation between daily life stressors, anxiety, and aggression, as well as the mediating role of cognitive perseveration; they can reveal relations between the frequency and intensity of anxiety and subsequent aggressive behavior. Moreover, the relation between the intensity of daily stressors and anxiety levels should be mediated by the degree of cognitive perseveration, and high ratings of cognitive perseveration on several consecutive calls would be expected to implicate ego depletion and thus predict aggression. It is critical that an ESM design has the potential to extend the real-time model illustrated in Figure 3 from the momentary impact of anxious feelings to the more extended influence of anxious moods. Moreover, ESM studies allow for a more ecologically valid assessment of the daily life experiences of AGG/ANX children, experiences often neglected in contemporary research.

Clinical Implications

The three main hypotheses that have been presented as part of a theoretical model linking anxiety and aggression, in real time and over development, lead to some clear implications for prevention and treatment. Despite promising outcomes from randomized controlled studies with aggressive children, there remains enormous variability in treatment outcomes, and processes of change are rarely examined (Brestan & Eyberg, 1998; Eyberg, Nelson & Boggs, 2008; Kazdin, 2001a, 2002, 2007). Based on the current model, I propose that treatment for aggressive children is less effective than it could be because anxiety is the driver of aggression for the majority of children, yet this is completely ignored in existing interventions. Moreover, some of the most strongly supported treatment protocols for aggression place children in contexts that can be viewed as highly anxiety producing. For example, many cognitive-behavioral group interventions for children use role-playing methods that involve asking children to act out scenarios during which they were previously aggressive (e.g., Augimeri, Farrington, Koegl, & Day, 2007; Koegl, Farrington, Augimeri, & Day, 2008; Lochman, 1992; Lochman & Lenhart, 1993). These public performances may cause some children intense feelings of anxiety that may limit the utility of these exercises for reducing future acts of aggression. A more serious effect is that these experiences may completely "turn off" the children and frighten them further, potentially exacerbating aggressive behavior problems or leading to attrition and negative appraisals about the efficacy of treatment programs in general.

There are additional iatrogenic considerations to keep in mind that are specific to anxiety problems. Many standard manuals on the treatment and prevention of anxiety disorders warn that insufficient duration of exposure to fear-inducing stimuli (not providing enough time for the client to get used to, and overcome, particular fears) may actually exacerbate symptoms of anxiety (Lilienfeld, 2007; Shipley & Boudewyns, 1980; Stone & Borkovec, 1975). The danger for children who are anxious but have been referred to treatment for only their aggression may be that they are exposed directly to the fear-inducing contexts that most distress them (e.g., provocations by peers, disapproval by counselors, teachers, or parents), but the therapists are not trained to help the child work through their distress. In other words, for many children in anger and aggression programs, we may be treating the symptoms, completely avoiding the causes and thus making some children worse.

On a more optimistic note, it is likely that many of the most effective treatments for aggressive children in the real world are already implicitly, if not explicitly, targeting children's anxiety. My own clinical research experience with children's mental health agencies in both North America and Europe suggests that the role of anxiety in childhood aggression is very much acknowledged and even addressed by frontline therapists in everyday practice. However, this clinical reality has yet to become explicit in most research programs that examine the efficacy of treatments for aggressive behavior problems. It is interesting that one of the original developers of effective, cognitive-behavioral prevention programs for childhood aggression recently noted that, when he and others are conducting their intervention, they often implement classic anxiety-reduction techniques (e.g., exposure therapy) before they proceed with their main cognitive-behavioral program to target aggression (J. E. Lochman, personal communication, May 2012). This may be more common than expected in community-based practice, and it echoes the need for the systematic testing of the effectiveness of targeting anxiety.

If anxiety is one of the causal generators of reactive aggressive behavior, this does not necessarily suggest that aggression-focused treatments are useless for AGG/ANX children. Rather, targeting anxiety at the beginning of therapy may be all that is needed to maximize the efficacy of these programs. Once children stop focusing exclusively on threats and learn to regulate their anxious moods, it may be that they can more flexibly allocate their attention to relevant aspects of their environment. With this more flexible attention allocation, their implementation of nonaggressive problem-solving strategies (a cognitive-behavioral skill taught in many programs for aggressive children) may be more successful.

The suggestion that anxiety-focused treatment is more effective than aggression-focused treatment for AGG/ANX children needs rigorous testing. However, even if this hypothesis is borne out, it may still be the case that some combination of these strategies will ultimately be most effective. Two types of research designs could be implemented to get a better sense of the best approach with AGG/ANX comorbid children. The first could be a straightforward randomized control

trial (RCT) meant to establish the relative efficacy of anxietyversus aggression-focused treatments on decreasing children's aggression.

The unique strengths of an RCT design are (a) it provides an opportunity to experimentally test causation, (b) it does so in an ecologically valid paradigm outside the laboratory, and (c) its results can be immediately relevant to clinical contexts where they can have their greatest impact. For example, among the most effective treatments for aggressive children are family-based parent management training combined with child-focused cognitive behavioral therapy (for reviews, see Brestan & Eyeberg, 1998; Dumas, 1989; Eyberg et al., 2008; Kazdin, 1987, 2001a, 2002; Tremblay, Pagani-Kurtz, Masse, Vitaro, & Pihl, 1995; Weisz, Doss, & Howley, 2005). AGG/ANX children and their parents could be randomly assigned to one of two interventions: a parent management training/cognitive behavioral therapy program that focuses on either (a) anxiety (Barrett, Duffy, Dadds, & Rapee, 2001; Dadds, Spence, Holland, Barrett, & Laurens, 1997; Flannery-Schroeder & Kendall, 2000; Kendall et al., 1997) or on (b) aggression (Barkley, 2000; Bloomquist & Schnell, 2002; Forgatch & DeGarmo, 1999; Martinez & Forgatch, 2001). It is essential that both anxiety- and aggressionfocused programs would have already been shown to be effective in previous RCT trials and that both are manualized, based on the same principles (e.g., cognitive-behavioral, parenting skills), and delivered for the same duration. If the comorbid children in the anxiety-based treatment protocol show more pronounced improvements in aggression compared with children in the aggression-based treatment group, then this would provide some evidence for the causal influence of anxiety on aggression problems.

The second type of treatment design could be combined with an RCT but the focus would be on examining mechanisms of change that account for improvements in children's anxious and aggressive symptoms. If RCT results turn out as hypothesized, it would remain unclear whether decreases in anxiety specifically caused the changes in aggressive behavior. Moreover, the model that I have presented posits that specific changes in parenting and cognitive perseveration will be associated with reductions in children's anxiety and subsequent aggression. However, change processes associated with treatment cannot be tapped by simply assessing outcome variables.

What is needed is a fine-grained assessment of emotional and behavioral changes in both parents and children, over the course of treatment, to pinpoint the timing of key changes (e.g., beginning, middle or end of treatment), their proximal causes (e.g., changes from unpredictable to predictable parenting in the home, reductions in cognitive perseveration), and the temporal sequence of those changes (e.g., decreases in anxiety should precede decreases in aggressive behavior). Toward these goals, the same ESM procedures that were described earlier could be combined with an RCT to examine precise relations between changes in anxiety and aggression. Observations of parent—child interactions over the course of

therapy (e.g., every 2 weeks) can also be used to identify parenting and emotional change processes associated with decreases in aggression (Lichwarck-Aschcoff et al., 2012).

In sum, if the proposed model of AGG/ANX children's development is correct, then one of the most important implications is its clinical relevance. Conducting an RCT comparing two well-validated treatments (anxiety and aggression focused) and identifying the precise mechanisms by which problem behaviors are reduced has the potential to advance both theory and practice in the field.

Scope of the Model

I have reviewed information most pertinent to a model that emphasizes how anxiety influences reactive aggression in childhood, including research on parent-child relationships, comorbidity rates, neural processes of emotion regulation, and treatment implications. My main aim was not to flesh out a comprehensive model of childhood aggression (for this type of integrative theoretical model, see Granic & Patterson, 2006) but to highlight often neglected internalizing mechanisms that may play a central role in the etiology and treatment of children's behavior problems. There are additional factors that influence the development of child psychopathology: Child temperament plays a central role in vulnerability to behavioral and anxiety problems (Calkins & Fox, 2002; Caspi, Henry, McGee, Moffitt, & Silva, 1995; Frick & Morris, 2004; Muris & Ollendick, 2005; Nigg, Goldsmith, & Sachek, 2004) and future research could integrate temperament data when testing the proposed model. There are additional ways to conceptualize comorbidity in children. Comorbidity for ADHD is high among aggressive children (Hinshaw, 1987, 1994). Diagnoses of depression may also co-occur frequently with those of children's anxiety, especially because the discriminant validity of distinctions between childhood anxiety and depression is highly controversial (e.g., Cole, Truglio, & Peeke, 1997; Kazdin, 1987; Patterson, Greising, Hyland, & Burger, 1997; Laurent & Ettelson, 2001; Turner & Barrett, 2003). However, my current purpose was not to focus on nosological issues or diagnostic criteria. Instead, my focus was on how anxiety, a basic emotion with well-established behavioral concomitants and a distinct psychophysiological signature (Izard, 1991; Fridja, 1986), impacts aggressive behavior. Genetic makeup is surely relevant (Caspi et al., 2003; Copeland et al., 2011; Dodge & Pettit, 2003; Raine, 2002) and there is little doubt that designs could be developed that incorporate Gene × Environment interactions. This would help determine for whom the model is most relevant, based on whether these emotional mechanisms differ for different genotypes.

Summary

I have reported that about 75% of aggressive children are also comorbid for anxiety, yet none of our most advanced developmental models of aggression incorporate the role of anxi-

ety, and our treatment programs largely ignore this comorbidity. Theoretical insights and extant research were brought to bear on a novel model of the potential impact of anxiety on reactive aggressive behavior. Specific suggestions were made about new research programs that could systematically test the hypotheses set out by this model. I suggested a variety of potentially innovative approaches, proposing that the link between anxiety and aggression can be examined in observational, experimental, and diary designs, at time scales ranging from seconds to years, and in the context of clinical and intervention trials, to establish causal specificity and real-world applicability.

If the hypotheses laid out in this article are supported by a new set of studies that directly test the effects of anxiety on aggression, then clinical practice could incorporate this knowledge to improve care for aggressive children. Supervisors and managers may need to train front-line clinicians to identify anxiety symptoms early and to target these symptoms effectively. Moreover, it is important to note that about 25% of children are anticipated not to have problems with anxiety; these are the children for whom an anxiety-focused treatment may not be appropriate. By highlighting these subtype differences in clinical practice, treatments may be tailored to diverse children and families. If we know more about these subtypes of aggressive children, and we focus on identifying the most relevant treatment strategies, programs can be distilled to their essential components, leading to stronger, more beneficial and cost-effective interventions.

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