

Emotion Regulation

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We begin this chapter reviewing literature characterizing emotion dysregulation as a core feature of children's and adolescents'¹ externalizing problems and disruptive behavior disorders,² emphasizing critical issues including involuntary reactivity versus voluntary control, the importance of anger, the role of effortful control, and biological correlates. Next, we consider the distinction between the reactive and proactive functions of aggression, the role and biological markers of emotion dysregulation in reactive aggression in particular, and the "unemotional" nature of proactive aggression. The third section of the chapter focuses on multiple pathways to disruptive behavior disorders and the importance of emotion dysregulation to each of these pathways. We conclude with a discussion of implications for prevention and intervention.

Defining Emotion Regulation

Emotion regulation encompasses the processes used to maintain or modify the valence or intensity of emotion (Cole, Martin, & Dennis, 2004; Eisenberg & Spinrad, 2004; Frick & Morris, 2004). The construct involves the modulation of internal experience and physiological states as well as overt expressive behaviors (Eisenberg & Spinrad, 2004). Furthermore, definitions emphasize adaptive responding, whether that response entails suppression, enhancement, or change in emotional experience and expression (Frick & Morris, 2004).

Another demarcation, based in the literature on temperament, distinguishes between involuntary reactivity, or initial autonomic responses to emotion-evoking events, and voluntary control of reactivity (Rothbart & Bates, 2006). These voluntary and involuntary components may interact, so that children who are both highly reactive and unskilled in control may struggle the most with emotion regulation (Derryberry & Rothbart, 1997; Eisenberg et al., 2000).

¹ Hereafter, references to "children" encompass both children and adolescents, unless a purely adolescent sample is being described.

² To narrow and focus the literature review, specific disruptive behavior disorders covered include Oppositional Defiant Disorder (ODD) and Conduct Disorder (CD) but exclude Attention Deficit Hyperactivity Disorder.

Emotion Regulation and Disruptive Behavior Disorders

Difficulties with emotion regulation have been identified as a core feature of most if not all forms of child psychopathology (Beauchaine, 2015; Cole, Michel, & Teti, 1994). Moreover, a vast literature characterizes emotion dysregulation as essential to both general externalizing psychopathology and specific disruptive behavior disorders such as ODD and CD. Within this literature, there is a focus on the challenges that externalizing children face when attempting to regulate anger, frustration, and hostility in particular (e.g., Casey & Schlosser, 1994; Eisenberg et al., 2005; Keltner, Moffitt, & Stouthamer-Loeber, 1995; Rothbart, Ahadi, & Hershey, 1994).

Much of this literature assesses emotion dysregulation as a unitary construct without regard to the distinctions between involuntary reactivity and voluntary control. It is easiest to make this distinction when studying infants, who do not yet self-regulate. From infancy, children display individual differences in their emotional responses to environmental stimuli (Cole et al., 2004), which may be considered a relatively pure index of trait-like reactivity at very young ages. Individual differences in this reactivity predict later externalizing problems, with young children who are susceptible to anger being more likely than other children to develop externalizing behaviors and disorders later in childhood (Arsenio, Cooperman, & Lover, 2000; Lengua & Kovacs, 2005; Rothbart et al., 1994).

Although involuntary reactivity develops early in life, voluntary control appears later (Derryberry & Rothbart, 1997). Thus, children's initial regulatory experiences are dyadic, and the quality of these experiences serves as a precursor to children's eventual regulatory skill (Feldman, Greenbaum, & Yirmiya, 1999; Field, 1994). Moreover, as children begin to self-regulate, the caregiver as a coregulator, model, and coach remains essential and predicts children's growing emotion regulation capability (Calkins & Johnson, 1998; Denham, 1993; Morris et al., 2011). When children do not receive strong regulatory assistance from caregivers in their earliest years, particularly around anger, their risk for externalizing problems increases (Cole, Teti, & Zahn-Waxler, 2003; Gilliom, Shaw, Beck, Schonberg, & Lukon, 2002).

Once children progress beyond the infancy period, however, observed expression of emotion becomes difficult to categorize as either involuntary reactivity or voluntary control (Beauchaine, 2015; Cole et al., 2004). Investigations have converged to suggest that children with externalizing problems display more negative emotion than controls in frustrating situations (Cole, Zahn-Waxler, & Smith, 1994; Gilliom et al., 2002; Rubin, Burgess, Dwyer, & Hastings, 2003). Whether these externalizing children are more emotionally reactive than their peers, less able to control their negative emotions, or a combination of the two is hard to determine.

An important advance in attempts to separate these components of children's emotion regulation can be seen in investigations of effortful control, which develops throughout maturation. Effortful control refers to a child's efficiency at modulating attention, inhibiting behavioral responses, and activating alternative behavioral responses, particularly in the context of emotion-evoking situations (Rothbart & Bates, 2006). Children's effortful control capabilities have been inversely linked to their externalizing behaviors across numerous studies (Duncombe, Havighurst, Holland, & Frankling, 2013; Eisenberg et al., 1996; Eisenberg et al., 2001; Gilliom et al., 2002; Rothbart et al., 1994; Valiente et al., 2003), including longitudinal prospective investigations (Eisenberg et al., 2000; Henry, Caspi, Moffitt, & Silva, 1996). This association may be particularly strong for children who exhibit high levels of negative emotionality or reactivity (Valiente et al., 2003). Moreover, separate components of effortful control, such as attentional control and inhibitory behavioral control, have demonstrated unique negative associations with externalizing behaviors (Eisenberg et al., 2001, 2005, 2009). Strong inhibitory control in particular may buffer children who

tend to experience negative emotions from engaging in externalizing behaviors; in one study of preadolescent boys, a link from increased anger and decreased fearfulness to alcohol use initiation was found only for boys without strong inhibitory control (Pardini, Lochman, & Well, 2004). As a caveat, effortful control is merely one component of the broader construct of executive functioning, which also relates to conduct problems (e.g., Lynam, 1996; Nigg, 2000). Therefore, it is hard to know whether externalizing children's difficulties with effortful control are specific to the management of emotions or are a sign of broader executive functioning problems.

Another valuable development in the assessment and understanding of emotional control is our growing knowledge of the role of vagal tone, the fundamental element of the parasympathetic nervous system. The most common index of vagal tone, respiratory sinus arrhythmia (RSA), measures heart rate variability by assessing the ebb and flow of heart rate during respiration (Murray-Close, 2013). RSA has been proposed as a psychophysiological marker of emotion regulation (Beauchaine, 2001), an idea supported by a growing empirical foundation. On the one hand, higher resting levels of RSA and less RSA withdrawal in response to emotion-evoking events are associated with more adaptive functioning and emotional control capacity (El-Shiekh, Hinnant, & Erath, 2011; Gordis, Feres, Olezeski, Rabkin, & Trickett, 2010; Porges, Doussard-Roosevelt, Portales, & Greenspan, 1996) and, longitudinally, improvements in emotion regulation have been linked to improvements in RSA (Vasilev, Crowell, Beauchaine, Mead, & Gatzke-Kopp, 2009). On the other, both low resting RSA (Beauchaine, 2001; Beauchaine, Gatzke-Kopp, & Mead, 2007; Beauchaine, 2015; de Wied, van Boxtel, Matthys, & Meeus, 2012; Hastings et al., 2008) and greater RSA withdrawal in anger-inducing situations (Gatzke-Kopp, Greenberg, & Bierman, 2015) have been linked to externalizing symptoms (See Chapter 9 of this book for a review of neurobiological factors related to disruptive behaviors).

The neural circuitry of both effortful control and RSA have been mapped to the prefrontal cortex (PFC), which has been associated with explicit emotion regulation processes (Etkin, Buchel, & Gross, 2015) such as reappraising and cognitively controlling emotions (Johnstone & Walter, 2014). Specifically, children's effortful control appears to operate through orbitofrontal and dorsolateral prefrontal inhibition of striatal activity and reactivity (Davidson, 2002; Heatherton, 2011), and positive correlations have been demonstrated between RSA and medial PFC activity in functional magnetic resonance imaging (fMRI) studies (Beauchaine & Thayer, 2015; Lane et al., 2009). Children with disruptive behavior disorders evidence less functional connectivity in striatal-anterior cingulate connections than comparison children (e.g., Shannon, Sauder, Beauchaine, & Gatzke-Kopp, 2009). More broadly, the typical reductions in PFC volume that begin in preadolescence as a result of gray matter pruning are not seen in children with conduct disorder (De Brito et al., 2009), and this maturational lag may well be implicated in the deficits that these children exhibit.

Hypothalamic-pituitary-adrenal (HPA) indices of emotion dysregulation in externalizing children have been uncovered as well. In particular, cortisol levels have been proposed to index emotion regulation, with both decreased baseline cortisol levels and increased cortisol reactivity to stress suggesting dysregulation (de Veld, Riksen-Walraven, & deWeerth, 2012; Zeman, Cassano, Perry-Parrish, & Stegall, 2006). In fact, reduced baseline cortisol levels have been linked to externalizing behavior including ODD and conduct problems (Alink et al., 2008; McBurnett, Lahey, Rathouz, & Loeber, 2000; Stoppelbein, Greening, Luebke, Fite, & Becker, 2014). This relation may be mediated by behavioral control (Shoal, Giancola, & Kirillova, 2003) and moderated by testosterone, with more pronounced effects for adolescents with higher testosterone levels (Platje et al., 2015; Popma et al., 2007). Furthermore, elevated cortisol response has also been associated with externalizing behavior (McBurnett et al., 2005), and this response is moderated by emotional control (Poon, Turpyn, Hansen,

Jacangelo, & Chaplin, 2016), with stronger relations emerging for children who struggle with emotional control.

This discussion of biological markers does not imply, however, that the struggles children with disruptive behavior problems face with emotional control are trait-like or present from birth. Rather, theory suggests that, although individual differences in reactivity to emotion-eliciting stimuli may be present from infancy (Arsenio et al., 2000; Lengua & Kovacs, 2005; Rothbart et al., 1994), voluntary control of that reactivity develops later and is less heritable and largely socialized (Beauchaine, 2015). In fact, parental emotion coaching shows positive effects on youth anger regulation and externalizing problems well into adolescence (Shortt, Stoolmiller, Smith-Shine, Mark Eddy, & Sheeber, 2010). Moreover, Beauchaine argues that temperamental traits such as emotional reactivity are not sufficient to lead to disruptive behaviors disorders. Instead, he emphasizes that externalizing disorders result from the coupling of these temperamental qualities with emotional control deficits conferred through socialization processes including poor parenting and early life stressors such as poverty (Hanson, Hair, et al., 2013) and neglect (Hanson, Adluru, et al., 2013). In particular, Beauchaine (2015) argues that emotion dysregulation is learned through repetitive cycles in which aggressive children escape from negative affective exchanges with family members and peers by escalating anger and hostility until the interactions terminate, resulting in negative reinforcement of the escalating behavior (Patterson, DeBaryshe, & Ramsey, 1989; Snyder, Edwards, McGraw, Kilgore, & Holton, 1994; Snyder & Patterson, 1995; Snyder, Schrepferman, & St. Peters, 1997). Moreover, recent work suggests that externalizing children are reinforced not only by escape from others' negative emotions and behaviors, but also by escape from their own aversive physiological state (Beauchaine & Zalewski, 2016; Skowron et al., 2011).

Emotion Regulation and Reactive versus Proactive Aggression

The literature reviewed above paints a clear picture of emotion dysregulation as a core feature of externalizing problems in general and disorders such as ODD and CD in particular. However, externalizing psychopathology is comprised of a wide variety of behaviors, only some of which emerge from dysregulated emotion. As a key example, we turn now to the distinction between reactive and proactive aggression. Reactive aggression is defensive, retaliatory, and in response to real or perceived provocation. In contrast, proactive aggression is initiated to reach a goal, whether that goal involves material or territorial gain or social dominance (Hubbard, McAuliffe, Morrow, & Romano, 2010). Based on these theoretical definitions, emotion dysregulation may be an important mechanism driving the reactive aggression of externalizing children, whereas it may play little role in their proactive aggression.

Reactive Aggression In fact, a growing body of literature supports the contention that emotion dysregulation is central to children's displays of reactive aggression but unrelated to their displays of proactive aggression. Several studies have related negative emotionality and expression to reactive aggression specifically, both concurrently and prospectively (Evans et al., 2016; Shields & Cicchetti, 1998; Vitaro, Barker, Boivin, Brendgen, & Tremblay, 2006). In particular, children's reactive aggression has been linked to their experience and expression of anger (Dane & Marini, 2014; Hubbard et al., 2002; Marsee & Frick, 2007; Orobio de Castro, Merk, Kooops, Veerman, & Bosch, 2005). Moreover, the link between children's anger and reactive aggression has been supported both longitudinally (Calvete & Orue, 2012) and cross-culturally (e.g., in China; Fung, Gerstein, Chan, & Engebretson, 2015).

Beyond negative emotional experience and expression, voluntary emotion regulation has been consistently linked to reactive aggression more strongly than proactive aggression, a finding supported in a meta-analysis of 11 studies (Card & Little, 2006), which further

demonstrated that this relation increased with age. Some studies suggest that emotion dysregulation is related only to reactive but not proactive aggression (Nas, Orobio de Castro, & Koops, 2005; Xu & Zhang, 2008), while other studies indicate a stronger link for reactive than proactive aggression, although relations between emotion dysregulation and both subtypes of aggression are found (Fite et al., 2016; Marsee & Frick, 2007). In one important study, the relation over time between children's anger and reactive aggression was moderated by their ability to regulate emotion, such that the longitudinal association was weaker for children with stronger emotion regulation skills (Calvete & Orue, 2012). Studies examining effortful control specifically as an index of emotion regulation have also revealed unique links to reactive but not to proactive aggression in middle childhood (Rathert, Fite, Gaertner, & Vitulano, 2011) and adolescence (Dane & Marini, 2014), with the latter study suggesting that the association is specific to the overt, but not the relational, form of aggression. In addition, effortful control has been shown to moderate the relation between anger and reactive aggression (Xu, Farver, & Zhang, 2009), such that the relation is significant at low and moderate but not at high levels of effortful control.

The connection between emotion dysregulation and reactive aggression has also been evidenced in work on psychophysiology, the HPA axis, and neural circuitry. In the first study to investigate the psychophysiological correlates of reactive and proactive aggression, children's sympathetic nervous system (SNS) reactivity in response to a laboratory-based peer provocation predicted teacher-rated reactive but not proactive aggression (Hubbard et al., 2002). More recent work suggests that children with low resting heart rate variability or vagal tone are more likely to engage in reactive aggression in particular (Scarpa, Haden, & Tanaka, 2010; Xu, Raine, Yu, & Krieg, 2014), a finding implicating RSA in the regulatory deficits of this subtype of aggression. The most current work on the psychophysiology of reactive aggression examined the interaction of children's sympathetic reactivity and parasympathetic RSA in-the-moment as they were given the opportunity to engage in reactive aggression against a provocative virtual peer; RSA moderated the in-the-moment relation between SNS reactivity and reactive aggression, with children displaying both elevated SNS reactivity and blunted RSA being particularly likely to respond with reactive aggression when provoked (Moore et al., 2016).

Turning to HPA correlates, a study by Lopez-Duran, Olson, Hajal, Felt, and Vazquez (2009) suggests that a heightened cortisol response to stress is linked to reactive aggression in particular. Consistent with this finding, van Goozen, Matthys, Cohen-Kettenis, Gispen-de Wied, Wiegant, and van Engeland (1998) found that children high on both externalizing and anxious symptoms displayed an elevated cortisol response to stress, whereas children high on externalizing but low on anxious symptoms displayed a blunted response; anxiety and depression have both been linked to reactive but not proactive aggression (Dodge, Lochman, Harnish, Bates, & Pettit, 1997; McAuliffe, Hubbard, Rubin, Morrow, & Dearing, 2007; Morrow, Hubbard, McAuliffe, Rubin, & Dearing, 2006; Raine et al., 2006; Vitaro et al., 2002). Thus, the link between elevated HPA axis responding may be implicated when children aggress in response to provocation but not when they aggress for instrumental or social gain.

In terms of neural circuitry, the amygdala, which may be involved in eliciting, monitoring, and stopping emotional arousal, serves both as a target of emotion regulation and as a regulatory influence (Thompson, Lewis, & Calkins, 2008; Zeman et al., 2006). The amygdala is thought to be associated with reactive aggression in particular through elevated threat responding (Crowe & Blair, 2008). In fact, Herpertz et al. (2005) demonstrated that boys with both CD and comorbid internalizing problems were most likely to display enhanced amygdala activity in response to emotional images, and this symptom pattern is most closely linked to the reactive subtype of aggression.

Before proceeding, a brief comment on hostile attributional bias seems warranted. As reviewed by Orobio de Castro and van Dijk in this volume (see Chapter 15), reactive but not proactive aggression is linked to children's tendency to attribute hostile intent to others when ambiguously provoked. While this social cognitive process can be theoretically separated from emotional reactivity, hostile attributional biases undoubtedly play an essential role in triggering reactive aggression in children by leading them to perceive situations as anger-inducing more often than their peers.

Proactive Aggression While emotion dysregulation may be an important mechanism underlying reactive aggression, the display of proactive aggression appears considerably more unemotional. In fact, proactive aggression is closely linked to the construct of callous-unemotional (CU) traits reviewed by Frick and Wall in Chapter 3 of this volume (and see also Marsee & Frick, 2007; Thornton, Frick, Crapanzano, & Terranova, 2013).

The theory that proactive aggression is unemotional in nature is borne out by psychophysiological work. The low resting heart rate that characterizes aggressive children has been linked to proactive aggression in particular (Raine, Fung, Portnoy, Choy, & Spring, 2014). Furthermore, in the first study to assess the SNS reactivity of children as they were given the opportunity to engage in unprovoked aggression for instrumental gain, Moore et al. (2016) found that children's in-the-moment skin conductance was inversely related to the level of proactive aggression they displayed toward a virtual peer. Theorists suggest that this blunted physiology may be a marker of temperamental fearlessness (Pardini, 2006) or the tendency to sensation-seek to increase arousal to normal thresholds (Beauchaine et al., 2007). Relatedly, children high in proactive aggression have been found to display elevated resting RSA, suggesting a strong capacity to regulate arousal (Scarpa et al., 2010).

In fact, in a 2006 meta-analysis, although a small positive zero-order correlation emerged between emotion dysregulation and proactive aggression, this association disappeared when reactive aggression was taken into account (Card & Little, 2006). More recent individual studies are equivocal on the relation between emotion regulation and proactive aggression taking reactive aggression into account. Two studies suggest a positive relation (Ostrov, Murray-Close, Godleski, & Hart, 2013; Rathert et al., 2011). These authors theorized that children skillful in regulating emotion may be more adept at carrying out the purposeful, goal-oriented behaviors that characterize proactive aggression. In contrast, another study revealed a modest negative relation (Calvete & Orue, 2012). However, this study differed from the majority of those reported above in that the sample was adolescent, emotion regulation was assessed via self-report responses to hypothetical vignettes, and proactive aggression was measured with a combination of self and peer report.

Of note, children who display proactive aggression or CU traits tend to be unable to identify others' sad or fearful facial expressions accurately (Blair, Colledge, Murray, & Mitchell, 2001; Blair & Coles, 2000; Dadds et al., 2006; Marsh & Blair, 2008) and to be less responsive to negative emotional images (Herpertz et al., 2005). This deficit may be critical to the characterization of proactive aggression as unemotional; it may be easier for children to remain emotionally unaroused and aggress for instrumental or social gain if they are not aware of the negative emotional reactions of their victims. In fact, when viewing videos of others being harmed, children's level of CU traits is inversely related to activity in the posterior insula, a region that plays a key role in empathy (Michalska, Zeffiro, & Decety, 2016). Furthermore, children with CD display hyporesponsiveness in the amygdala when processing fearful facial expressions (Stadler, Poustka, & Sterzer, 2010), and these authors speculate that this deficit may be linked to displays of "unemotional aggression" in particular. In support of this idea, this reduced amygdala response has been shown to be specific to externalizing children with CU traits (Hwang et al., 2016) and to mediate the relation between CU traits and proactive

aggression (Lozier, Cardinale, Van Meter, & Marsh, 2014). More generally, theorists have speculated that the enhanced amygdala response associated with reactive aggression and the diminished amygdala response linked to proactive aggression may indicate differential pathways to externalizing disorders (Crowe & Blair, 2008; Frick & White, 2008).

Emotion Regulation and Pathways to Disruptive Behavior Disorders

The literature reviewed above may lead readers to conclude that two distinct groups of aggressive children exist, with one engaging in primarily reactive aggression and the other engaging in predominantly proactive aggression. In fact, researchers originally hypothesized that such well-defined groups would emerge (Dodge, 1991). However, the correlation between reactive and proactive aggression is consistently high across studies (Card & Little, 2006; Polman, Orobio de Castro, Koops, van Boxtel, & Merk, 2007), suggesting that many aggressive children engage in both subtypes of aggressive behavior. To some degree, then, the subtypes of aggression may be more accurately conceptualized as continuous measures of the extent to which children display each subtype, rather than as categories into which children are placed.

In fact, when the SNS arousal of children diagnosed with disruptive behavior disorders was assessed in both a baseline condition and a peer provocation, findings suggested that externalizing children demonstrated both lower baseline arousal and greater reactivity to the peer provocation than controls (van Goozen, Matthys, Cohen-Kettenis, Buitelaar, & van Engeland, 2000). Although these authors did not assess the reactive and proactive functions of aggression, their results suggest that aggressive children's blunted baseline SNS arousal may put them at risk of displaying proactive aggression when faced with the opportunity to aggress for instrumental gain, but that their sympathetic arousal in response to peer provocation may also increase the chance that they will display reactive aggression. In fact, if aggressive children's SNS profiles are characterized by both of these patterns, then it follows that many aggressive children may aggress for both reactive and proactive reasons, albeit in different contexts, as the consistently high correlation between the subtypes of aggression across studies suggests.

However, two recent rigorous investigations suggest that some aggressive children may display reactive aggression only, while others display both reactive and proactive aggression. In a study by Smeets et al. (2017), self-report data on reactive and proactive aggression from a large sample of adolescents were analyzed using latent class analysis; two latent classes of aggressive adolescents emerged, one that engaged primarily in reactive aggression and a second that displayed both reactive and proactive aggression. Similarly, in a recent study of adolescents in community, at-risk residential, and detained samples, cluster analyses of reactive and proactive aggression revealed three groups, with the first low on aggression overall, the second elevated on reactive aggression only, and the third elevated on both reactive and proactive aggression. With a few exceptions, these findings were replicated across the three samples, across boys and girls, and across physical and relational aggression (Marsee et al., 2014). Further analyses suggested that these three groups differed in severity, with the combined group displaying higher levels of emotion dysregulation, CU traits, and delinquency than the reactive-only group, which, in turn, exhibited higher levels of these constructs than the low-aggression group (Marsee et al., 2014). Both studies converged to suggest that few if any children display proactive aggression only.

These findings may have important implications for our understanding of multiple pathways to disruptive behavior disorders such as CD. This work suggests a first and less severe pathway characterized primarily by reactive aggression and a second and more severe one marked by both reactive and proactive aggression. It may well be that some externalizing children aggress predominantly when provoked while other disruptive children aggress both

when provoked and to achieve instrumental or social gain. Importantly, both of these pathways toward disruptive behavior disorders are typified by emotion dysregulation.

Of note, a recent fMRI study supports the notion that children with and without CU traits evidence emotion dysregulation when provoked. Compared to a control sample, all adolescents with disruptive behavior disorders displayed reduced amygdala–ventromedial PFC connectivity when provoked, regardless of whether they had CU traits, and this reduction predicted both their tendency to retaliate during a laboratory task and parent ratings of reactive aggression. These results suggest that all youth with disruptive behavior disorders may be at risk for reactive aggression and propose one neural mechanism behind this risk (White et al., 2016).

This theory parallels in many ways Frick’s hypothesis of two pathways toward CD, with one marked by anger dysregulation and the other by CU traits (see Frick, 2012; Frick & Morris, 2004; Frick & White, 2008; Pardini & Frick, 2013; and Chapter 3 of this volume for elegant reviews of this theory and empirical work supporting it). Notably, both of these models are supported by literature suggesting that the divergent pathways are denoted by differential familial precursors, cognitive mechanisms, and outcomes, although a review of these findings is beyond the scope of the current chapter (see Hubbard et al., 2010; Frick & White, 2008 for reviews).

However, the models diverge in that we emphasize that aggressive children with CU traits also struggle with emotion regulation, perhaps particularly when provoked, and in fact, that their regulatory deficits may be more serious than those of children who do not evidence CU traits or proactive aggression. Thus, we deviate from Frick’s thinking by emphasizing that both pathways toward disruptive behavior disorders are characterized by emotion dysregulation, making this construct particularly critical to our understanding of the full continuum of externalizing problems.

Clearly, careful longitudinal work utilizing rigorous measurement approaches is needed to determine the extent to which the constructs of reactive-versus-proactive aggression and CU-traits-versus-anger dysregulation overlap, as well as the distinctness of the pathways characterized by one or more of these constructs. In our view, this effort represents a critically important direction for future research on emotion regulation and externalizing behaviors.

Implications for Prevention and Intervention

Because of the important role that emotion dysregulation plays in the development of children’s disruptive behavior disorders, it is critical to target emotion regulation skills in prevention and intervention programs for externalizing disorders. Current best practice recommendations suggest that interventions for disruptive behavior are more effective when they occur earlier in childhood (Eyberg, Nelson, & Boggs, 2008). For this reason, one possible and infrequently considered target may be the quality of the parent–child relationship. In fact, an intervention program termed Attachment and Biobehavioral Catch-Up which aims to improve attachment quality has shown effects on toddlers’ negative affect (Lind, Bernard, Ross, & Dozier, 2014) and cortisol regulation (Bernard, Dozier, Bick, & Gordon, 2015), although it is too soon to know whether these effects will translate into lower levels of disruptive behavior disorders. More generally, intervention programs directed at improving general parenting skills have shown effects on children’s emotion dysregulation and reactive aggression (e.g., Barker et al., 2010; Scott & O’Connor, 2012). However, as emotion regulation is thought to be highly socialized (Beauchaine, 2015), it may be wise to target parental coaching of emotion regulation more directly (Gottman, Katz, & Hooven, 1996). Of note, one series of studies has demonstrated the effectiveness of the “Tuning In” program, with

increases in parental emotion coaching predicting reductions in externalizing behaviors for toddlers (Lauw, Havighurst, Wilson, Harley, & Northam, 2014), children (Havighurst, Wilson, Harley, & Prior, 2009), and adolescents (Havighurst, Kehoe, & Harley, 2015). However, these studies did not directly assess emotion regulation, leaving open the question of whether emotion coaching lowered externalizing behaviors through the mechanism of increased emotion regulation.

Of course, efforts to increase children's emotion regulation skills and decrease their aggressive behaviors should also target the children themselves. In fact, when a child component was added to the Tuning In program targeting the identification and regulation of emotions, similar reductions in behavior problems resulted (Havighurst, Duncombe, et al., 2015). Interestingly, Lewis et al. (2008) combined parent management training with child cognitive-behavior therapy to reduce children's behavior problems. Their results revealed that those children who benefitted from the intervention displayed ventral prefrontal activation reduction at the peak of the N2 (an event-related potential marker of inhibitory control) that was similar to comparison children, while children whose behavior problems were not changed did not show similar reductions. These findings suggest that programs do not have to directly target emotion regulation skills to produce measurable effects on biological markers of emotion regulation. Finally, no discussion of programs aimed at teaching children emotion regulation skills in the service of decreasing externalizing behaviors would be complete without mention of the Coping Power program, reviewed thoroughly in Chapter 27 of this volume by Boxmeyer and colleagues. This program targets the emotion regulation deficits of aggressive children, along with cognitive and social-problem-solving skills, to produce reductions in externalizing behaviors at postintervention (Lochman et al., 2009), one-year follow-up (Lochman & Wells, 2003), and three-year follow-up (Lochman, Wells, Qu, & Chen, 2013).

In addition, the body of work on reactive and proactive aggression reviewed above suggests that it may be fruitful to target children's reactive and proactive aggression separately in intervention efforts. Treatment for reactive aggression could focus on anger dysregulation and hostile attributional biases, while efforts to reduce proactive aggression could emphasize reading others' distress cues, empathy-building, and balancing instrumental and social goals. Although numerous calls for separate treatment packages have been made (Dodge, 1991; Phillips & Lochman, 2003; Vitaro & Brendgen, 2005), little progress has followed, perhaps owing to concern that many children need treatment for both subtypes of aggression, given their high correlation, as well as findings suggesting that these children may display the most severe symptoms. However, research is needed to determine whether these treatments could be delivered more effectively in separate modules.

Conclusions

As we conclude, it seems appropriate to note the remarkable progress made over the past decades in our understanding of the role of emotion dysregulation in children's disruptive behavior disorders, and celebrate those researchers who have advanced our field to this point. Twenty years ago, it would have been impossible to imagine that we could know all that we do now about the nature of emotion regulation, its biological markers, and links to children's externalizing behavior in general and reactive versus proactive aggression in particular. Much work remains as we continue to move forward in our understanding of the multiple pathways toward disruptive behavior disorders and complementary treatment approaches for different subtypes of aggressive behavior. We feel certain that our field is up to the task and look forward to the exciting advances that are sure to come in the years ahead.

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