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FUTURE DIRECTIONS

Future Directions in Emotion Dysregulation and Youth Psychopathology

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This article reviews central nervous system substrates and autonomic correlates of emotion dysregulation and offers several suggestions for future research. Studies conducted in the last two decades indicate that effective emotion regulation requires efficient top-down, cortically mediated regulation of bottom-up, subcortically mediated individual differences in trait impulsivity and trait anxiety. Without making critical distinctions between highly heritable individual differences in trait impulsivity and trait anxiety, versus less heritable and more socialized deficiencies in emotion regulation, progress in understanding the development of psychopathology among children and adolescents will be hampered. Future research can also be improved by measuring emotion dysregulation across multiple level of analysis, specifying physiological mechanisms through which operant reinforcement shapes emotional lability, improving the internal and external validity of psychophysiological measures, integrating emotion dysregulation into factor analytic and behavioral genetic models of psychopathology, identifying molecular genetic risk for emotion dysregulation, and expanding neuroimaging research on emotion dysregulation among children and adolescents.

Interest in the roles of emotion regulation and dysregulation in normative and psychopathological development has burgeoned in the last two decades (see, e.g., Adrian, Zeman, & Veits, 2011). This upsurge in interest began in part with publication of a *Monograph of the Society for Research in Child Development* in which top developmentalists and child psychopathologists considered behavioral and biological bases of emotion regulation, and their relations to psychological adjustment (Fox, 1994a). The monograph, which represents some of the most advanced thinking of the time, is a compendium of papers that were presented initially at a 1991 conference on the development of emotion regulation. Papers in the monograph were highly influential in shaping my own thinking as a graduate student, and paved the way for me to write my first review article, which considered the importance of emotion dysregulation in the development of both internalizing and externalizing disorders (Beauchaine, 2001).

Although it is now generally accepted that problems with emotion regulation confer vulnerability to a wide range of psychopathological outcomes (Cole, Hall, & Hajal, 2013), no such consensus existed in the early 1990s. At that time, behavioral and cognitive paradigms still dominated psychology, and the view that emotional states were subjective, unquantifiable, and unamenable to scientific investigation prevailed (see Beauchaine & Zalewski, in press). Papers in the 1994 monograph helped to change this view by demonstrating how emotional states can be inferred, verified, and quantified by measuring appropriate biological systems via hormonal assays (Stansbury & Gunnar, 1994), electroencephalography (Dawson, 1994; Fox, 1994b), and electrocardiography (Porges, Doussard-Roosevelt, & Maiti, 1994). As a result of such research, and of functional magnetic resonance imaging (fMRI) studies being conducted on the neural bases of emotion among adults (e.g., Davidson, 2000), interest in the constructs of

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emotion regulation and dysregulation resurged and are central to contemporary models of psychopathology. Indeed, emotions are fundamental to the positive and negative valence systems of the Research Domain Criteria (National Institute of Mental Health, 2014).

EMOTION REGULATION VERSUS EMOTION DYSREGULATION

Emotion regulation can be defined as the set of processes through which emotional experience and expression are shaped, whether volitionally or automatically, in the service of adaptive behavior (Thompson, 1990). Such shaping of emotion may occur through various mechanisms, including attentional, cognitive, social, and behavioral (Aldao, Nolen-Hoeksema, & Schweizer, 2010; Cole et al., 2013). According to the functionalist perspective, regulated emotions serve to initiate, sustain, alter, or terminate relations between a person and his or her environment on issues of importance to the person (Campos, Mumme, Kermoian, & Campos, 1994). Because processes that subserve emotion—particularly central nervous system processes—are unobservable under most circumstances, effective emotion regulation is sometimes inferred from positive behavioral functioning in the face of environmental challenge. Although this may make sense conceptually, it is problematic scientifically because emotion regulation is quantified by the absence of problem behavior rather than being measured directly (see Cole, Martin, & Dennis, 2004). More preferably, inferences about emotion regulation should be "gleaned from meticulous observations of behavior, including examinations of emotion-behavior-emotion sequences, convergence of data from multiple levels of measurement (behavioral, self-report, and physiological), and strategic manipulations of situational context" (Cole et al., 2013, p. 344).

Given inherent difficulties with measuring emotion regulation (Cole et al., 2004), particularly in naturalistic settings, and given that our research addresses the development of psychopathology, we focus on emotion dysregulation, which can often be measured more directly. In contrast to the adaptive nature of emotion regulation, emotion dysregulation can be described as a pattern of emotional experience and/or expression that interferes with appropriate goal-directed behavior (see Beauchaine & Gatzke-Kopp, 2012). In most forms of psychopathology, whether internalizing or externalizing, one or more negative emotions (sadness, panic, rage, anxiety) is experienced either too intensely or too enduringly to be adaptive (Beauchaine, Gatzke-Kopp, & Mead, 2007). Emotion dysregulation is therefore easier to quantify via direct behavioral observation and/or self-report. Of importance, emotion dysregulation is a broad risk factor for psychopathology, with neural substrates that interact with dysfunction in other neurobiological systems to produce diverse forms of maladjustment (see Beauchaine, 2001), a point I elaborate next.

TRAIT ANXIETY, TRAIT IMPULSIVITY, AND VULNERABILITY TO PSYCHOPATHOLOGY

As I and others have reviewed elsewhere, emotion dysregulation is observed in almost all forms of psychopathology. Internalizing disorders are characterized by dysregulated anxiety, panic, and/or dysphoria, whereas externalizing disorders are characterized by dysregulated anger or other approach-related affect (Beauchaine, 2001; Beauchaine & Thayer, in press). These emotions have neural substrates in bottom-up, subcortical brain regions that mature very early in life. Trait anxiety is subserved by the septohippocampal system, including serotonergic (5HT) projections of the raphe nuclei and noradrenergic projections of the locus ceruleus (Gray & McNaughton, 2000), whereas trait impulsivity is subserved by the mesolimbic dopamine (DA) system, including projections from the ventral tegmental area to the nucleus accumbens and ventral striatum (see Neuhaus & Beauchaine, 2013; Zisner & Beauchaine, in press-a). Individual differences in activity and reactivity of these systems give rise to ordinary variation in temperament and personality, and at the extremes, vulnerability to internalizing and externalizing psychopathology, respectively (Beauchaine, 2001; Beauchaine & Thayer, in press; Depue, 2012; Gray, 1987; Kagan, 2013; Valle Krieger & Stringaris, in press).[1]

Both trait anxiety and trait impulsivity are highly heritable (e.g., Lamb et al., 2010; Nikolas & Burt, 2010). In childhood, trait anxiety confers vulnerability to social withdrawal and early-onset anxiety disorders (e.g., Schwartz, Snidman, & Kagan, 1999), whereas trait impulsivity confers vulnerability to attention-deficit/hyperactivity disorder (ADHD) and early-onset conduct problems (e.g., Beauchaine & McNulty, 2013). In structural models of symptoms among both children and adults, these predispositions emerge as latent internalizing and externalizing factors (e.g., Achenbach & Edelbrock, 1991; Krueger, 1999), which confer vulnerability to a wide range of anxiety disorders (e.g., Tambs et al., 2009) and externalizing spectrum disorders (Krueger et al., 2002),

[1]Readers will note that both Kagan (2013) and Gray (Gray & McNaughton, 2000) used the term *behavioral inhibition* to refer to temperamental anxiety, as derived from septohippocampal activity and reactivity. Given that the focus of this article is on psychopathology rather than ordinary variation temperament, I use the term *trait anxiety* throughout. Of importance, ordinary variation in behavioral inhibition is unlikely to eventuate in psychopathology. A similar argument can be made for the construct of trait impulsivity, where ordinary variation is better conceptualized as temperamental exuberance than vulnerability to psychopathology (e.g., Degnan et al., 2011).

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respectively. Of importance, even though trait anxiety and trait impulsivity are highly heritable, the specific form(s) of internalizing (e.g., panic, generalized anxiety, posttraumatic stress disorder) and externalizing (e.g., ADHD, conduct disorder, substance dependence) behavior that are exhibited by individuals who score high on these traits are determined by interactions between neurobiological vulnerabilities and environmental risk factors. For example, although impulsive male individuals who are reared in protective environments are likely to exhibit only ADHD (Beauchaine, Hinshaw, & Pang, 2010; Beauchaine & McNulty, 2013), their impulsivity often progresses to more serious externalizing conduct if they are reared in high-risk environments characterized by coercive parenting (e.g., Patterson, DeGarmo, & Knutson, 2000), coercive and deviant peer group affiliations (e.g., Dishion & Hiatt Racer, 2013; Snyder et al., 2008), and neighborhood violence and criminality (e.g., Meier, Slutske, Arndt, & Cadoret, 2008). Similarly, environmental risk, including child maltreatment and exposure to protracted stress, increase the likelihood of emerging anxiety and depressive disorders among those with specific genetic vulnerabilities, such as the short allele of the serotonin transporter and certain glucocorticoid polymorphisms (see, e.g., Beauchaine, Crowell, & Hsiao, 2015; Nugent, Tyrka, Carpenter, & Price, 2011; Stein, Schork, & Gelernter, 2008).[2]

PREFRONTAL CORTEX DEVELOPMENT, NEURAL PLASTICITY, AND EMOTION DYSREGULATION

The prefrontal cortex (PFC) comprises several functional subdivisions of the brain's frontal lobes. In contrast to trait anxiety and trait impulsivity, which arise largely from heritable individual differences in activity and reactivity of bottom-up, subcortical brain networks that mature very early in life, emotion regulation is less heritable, considerably socialized (Goldsmith, Pollak, & Davidson, 2008), and subserved by top-down, cortical brain networks that continue to mature into the early 20 s (see Beauchaine & McNulty, 2013; Gogtay et al., 2004; Snyder, in press). Volitional regulation of anxiety occurs through lateral prefrontal inhibition of amygdalar activity and reactivity, whereas volitional regulation of impulsivity occurs through orbitofrontal and dorsolateral prefrontal inhibition of striatal activity and reactivity (see Davidson, 2002; Heatherton, 2011; Heatherton & Wagner, 2011). Children and adolescents with anxiety disorders exhibit less functional connectivity in amygdalar-ventrolateral prefrontal connections than controls (e.g., Monk et al., 2008), and adolescents with externalizing disorders exhibit less functional connectivity in striatal-anterior cingulate connections than controls (e.g., Shannon, Sauder, Beauchaine, & Gatzke-Kopp, 2009). Deficient top-down control of the amygdala by the medial PFC, and reduced functional connectivity between the amygdala and the orbitofrontal cortex, are implicated in emotional lability and deficient self-regulation (see Churchwell, Morris, Heurtelou, & Kesner, 2009; Hilt, Hanson, & Pollak, 2011).

Among typically developing individuals, the PFC reaches peak volume around ages 10–11, with gradual reductions observed thereafter (see Lenroot & Giedd, 2006).[3] This maturational process can be indexed in part by gray matter pruning, which is normally observed from middle childhood into early adulthood (see Gogtay et al., 2004). Among boys with conduct disorder, such reductions in prefrontal gray matter are not observed (De Brito et al., 2009). In addition, children with ADHD evidence a considerable lag in neurodevelopment of the medial PFC compared with controls (Shaw et al., 2012). Such failures of prefrontal maturation likely have implications for difficulties with emotion regulation among those with externalizing behavior disorders (see Beauchaine & McNulty, 2013).

The PFC is also quite sensitive to environmental insults. Orbitofrontal cortex volumes correlate inversely with levels of physical abuse incurred by children and predict both social difficulties with peers and executive functioning deficits (Hanson et al., 2010). In addition, cumulative lifetime adversity is associated with smaller PFC volumes among children (Hanson et al., 2012), as

[2]The assertions that subcortical structures mature very early in life, and that individual differences in motivational and emotional predispositions that they confer are highly heritable, in no way suggests that these neural networks are insensitive to environmentally induced functional alterations across development. As my research group has reviewed elsewhere, epigenetic and other maternal programming effects on functioning of central monoamine systems (dopamine, serotonin, norepinephrine) can be extensive (Beauchaine, Neuhaus, Zalewski, Crowell, & Potapova, 2011; Mead, Beauchaine, & Shannon, 2010), as can effects of postnatal exposure to adversity and substances of abuse (Beauchaine & McNulty, 2013). Of importance, some of these effects reflect active gene–environment correlation, whereby impulsive and anxious individuals seek environments that exacerbate their preexisting neural vulnerabilities. For further reading, interested parties are referred to sources cited herein.

[3]Although neurodevelopment of the prefrontal cortex is more protracted than neurodevelopment of subcortical structures, these differences are a matter of degree. Structural changes are observed in the hippocampus and the amygdala from childhood to adulthood (e.g., Østby et al., 2009), and effects of severe environmental deprivation in toddlerhood are observed on neurodevelopment of the PFC at age 8 (McLaughlin, Sheridan, et al., 2014). Thus, readers are encouraged to view central nervous system maturational processes as dynamic and interactive, not as a set of discrete events. Of importance, this does not invalidate the conjecture that self-control over behavior is mediated more by subcortical mechanisms very early in life and that frontal mechanisms become increasingly important across development (see Beauchaine & McNulty, 2013).

is being reared in impoverished neighborhoods (Hanson, Hair, et al., 2013). Furthermore, child neglect is associated with more diffuse frontal white matter organization (Hanson, Adluru, et al., 2013). Among rodents, chronic exposure to stress results in less dendritic branching and lower neural spine densities in the PFC (e.g., Holmes & Wellman, 2009).

Neurodevelopment of the PFC and its interconnections with subcortical structures is altered further by exposure to alcohol and other drugs of abuse. Substance dependence compromises prefrontal/orbitofrontal cortex structure and function, resulting in more impulsive decision making and susceptibility to relapse (e.g., Schoenbaum & Shaham, 2008). Moreover, addiction is facilitated by use-dependent disruption in top-down regulation of mesolimbic reward regions by the PFC, which has additional adverse effects on self-regulation (see Goldstein & Volkow, 2011; Kalivas, 2008). Chronic elevation of DA neural firing in the nucleus accumbens, which is induced by strong stimulant exposure among rodents and nonhuman primates, downregulates tonic DA activity, sensitizes phasic DA responding to such stimulants, and suppresses the strength of developing connections between mesolimbic structures and the PFC (see Thomas, Beurrier, Bonci, & Malenka 2001; Vezina, 2004). Given the importance of the prefrontal cortex and its interconnections for decision making, planning, and other executive functions (e.g., Floresco & Magyar, 2006), such findings have obvious implications for the development of self-regulatory behavior (e.g., Davidson, Putnam, & Larson, 2000; Quirk & Beer, 2006).

TRAIT ANXIETY, TRAIT IMPULSIVITY, AND EMOTION DYSREGULATION IN PSYCHOPATHOLOGY

Consistent with this discussion, we have suggested that trait anxiety and trait impulsivity are necessary but insufficient for developing internalizing and externalizing spectrum disorders, respectively. Many of those who score high on these traits regulate their emotions effectively and avoid functional impairment. However, when subcortically mediated trait anxiety or trait impulsivity are coupled with cortically mediated deficiencies in emotion regulation, psychopathology is a likely outcome (Beauchaine, 2001; Beauchaine & Gatzke-Kopp, 2012; Beauchaine et al., 2007).

Compelling evidence indicates that emotional lability and emotion dysregulation are socialized within coercive and invalidating families via operant reinforcement contingencies that occur thousands of times across development (see, e.g., Snyder, Schrepferman, & St. Peter, 1997). Socialization mechanisms of emotion dysregulation are specified in modern instantiations of coercion theory (see Beauchaine & Zalewski, in press), which identifies escalation of and escape from negative affective expressions of other family members (i.e., invalidation, anger, violence) as causal agents in reinforcement of emotional lability (e.g., Patterson, DeBaryshe, & Ramsey, 1989; Snyder, 1977). Through meticulous behavioral coding, Snyder and colleagues have quantified negative reinforcement of emotion dysregulation during dyadic exchanges between aggressive children and their parents and linked these reinforcement processes to longitudinal increases in delinquency and affective lability (e.g., Snyder, Edwards, McGraw, Kilgore, & Holton, 1994; Snyder et al., 1997). Aggressive boys and their parents are more likely to initiate aversive interactions with one another than controls, and to reciprocate and exceed one another's levels of negative affect (e.g., Snyder, 1977; Snyder et al., 1994; Snyder & Patterson, 1995). Snyder also demonstrated that aggressive boys become more likely to escalate conflict once in a dysregulated, irritable state (Snyder et al., 1997) and that intense displays of negative affect are more likely to cease conflict in aggressive dyads than in control dyads (Snyder et al., 1994). These landmark studies indicate that negative reinforcement occurs not only via escape from the aversive behaviors of others but also through escape from one's own visceral, dysregulated states (see Beauchaine & Zalewski, in press; Skowron et al., 2011).

Recent research indicates that negative reinforcement of emotional lability also occurs in families of internalizing adolescent girls, whose expressions of negative affect are often met with invalidation by family members. Escape from such invalidation serves a negative reinforcement function. In dyadic interactions between self-injuring adolescents and their mothers, these negative reinforcement contingencies are associated with emotion dysregulation (Crowell et al., 2013). Of importance, dysregulated anger and anxiety are also modelled in families of aggressive and anxious children, respectively (e.g., Bandura, 1978; Burstein & Ginsburg, 2010).

Other research indicates that (a) behavioral inhibition in infancy predicts structural abnormalities in the ventromedial PFC in young adulthood (Schwartz et al., 2010); (b) early life stress mediates—via increased cortisol levels—decreased functional connectivity between the amygdala and the vmPFC 14 years later (Burghy et al., 2012); and (c) childhood maltreatment portends compromised functional connectivity between the subgenual cingulate cortex and both the amygdala and the hippocampus in adolescence (Herringa et al., 2013). Thus, environmental experience appears to play an important role in the development of neural vulnerability to emotion dysregulation among those with internalizing as well as externalizing disorders.

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DEVELOPMENTAL MECHANISMS OF EMOTION DYSREGULATION

Following from the preceding discussion, we are interested in how reinforcement of affective lability leads physiological states (e.g., autonomic reactivity, PFC function) to take on highly canalized, traitlike qualities across development, producing patterns of emotional responding that are highly resistant to change (see Perry, 2008). In our research, we seek to (a) characterize temperamental traits (e.g., anxiety, impulsivity) that render some individuals more vulnerable than others to long-term effects of coercive, invalidating, stressful, and abusive family environments (Beauchaine & Gatzke-Kopp, 2012; Crowell et al., 2013); (b) describe how, via allostatic mechanisms,[4] reinforcement of affective lability alters the operating ranges of biological systems that subserve emotion regulation functions (Beauchaine et al., 2011); (c) identify how these altered biological systems, combined with continued coercion, invalidation, and adversity, confer prospective risk for emotion dysregulation across subtypes of psychopathology (Beauchaine & McNulty, 2013; Beauchaine, Shader, & Hinshaw, in press); and (d) determine how biological vulnerabilities predict intervention effects and are modified by prevention and intervention programs (Beauchaine et al., 2015). In our developmental models of externalizing behavior and intentional self-injury (e.g., Beauchaine, Klein, Crowell, Derbidge, & Gatzke-Kopp, 2009; Beauchaine & McNulty, 2013), socialization of emotion dysregulation plays a primary mediating role in potentiating biological vulnerabilities to psychopathology. We make strong distinctions between individual differences in temperament (trait impulsivity, trait anxiety), which are highly heritable, and socialized deficiencies in emotion dysregulation, which are far less heritable (see above). Our thinking is summarized in Figure 1, where genetic vulnerabilities (top box) predispose to trait impulsivity. In high-risk family contexts such as those characterized by coercion and invalidation (left panel), these genetic vulnerabilities are potentiated, leading to emotion dysregulation, oppositionality, and significant risk for serious conduct problems. Later in development, children on this trajectory are vulnerable to adverse influences of deviant peer groups, and neighborhoods high in violence and criminality. In contrast, children who are reared in protective family environments that are characterized by effective parenting, including de-escalation of affective lability, clear consequences for aggression, and positive reinforcement of prosocial behaviors (right panel), are unlikely to progress to serious conduct problems given well developed emotion regulation skills (Beauchaine et al., 2013; Beauchaine et al., 2010; Beauchaine et al., 2009; Beauchaine et al., in press). Nevertheless, they remain impulsive given the high heritability of this trait.

[4]Allostasis refers to long-term, stress-induced changes in the functional operating ranges of vital biological systems (Sterling & Eyer, 1981). Readers who are interested in allostatic effects on brain function are referred to Beauchaine et al. (2011) for a comprehensive review.

QUANTIFYING EMOTION DYSREGULATION

Emotion dysregulation can be quantified through behavioral observation, informant report, self-report, and/or physiological function. Of importance, studies of emotion dysregulation that appear in high-impact journals tend to use multiple methods of assessment, including at least one physiological measure (see Adrian et al., 2011). Most often, respiratory sinus arrhythmia (RSA), an index of parasympathetic (PNS)-linked cardiac activity (see next), is used. However, some researchers have also used event-related potentials and fMRI in efforts to evaluate biological correlates of emotion regulation/dysregulation among children and adolescents (e.g., Hare et al., 2008; Lewis, Lamm, Segalowitz, Stieben, & Zelazo, 2006). Although these latter two methods are somewhat restricted in ecological validity, their use is likely to increase in upcoming years (see next).

In their recent review of the emotion regulation assessment literature, Adrian et al. (2011) identified 47 different behavioral paradigms, including 13 that have been used with infants, 14 that have been used with toddlers/preschoolers, 13 that have been used with elementary-school-aged children, and 7 that have been used with adolescents. Although emotion regulation/dysregulation assessment methods must be sensitive developmentally, many different tasks are used even within developmental epochs. Given this lack of standardized tasks throughout the literature, it becomes especially important to verify inferences about emotion regulation/dysregulation by using multiple methods, particularly physiological.

A number of self-report measures also exist, most of which are validated for use with older children and/or adolescents given limited insight of younger children. Among the most commonly used are the Children's Emotion Management Scales (Zeman, Shipman, & Penza-Clyve, 2001) and the Emotion Expression Scale for Children (Penza-Clyve & Zeman, 2002). In adolescent samples, the Difficulties with Emotion Regulation Scale (Gratz & Roemer, 2004), which was developed originally for use with adults, is well validated and increasingly popular (Weinberg & Klonsky, 2009). The Emotion/Affect Regulation Interview (Zeman & Garber, 1996), which includes questions about emotional reactions to several vignettes, is also used frequently.

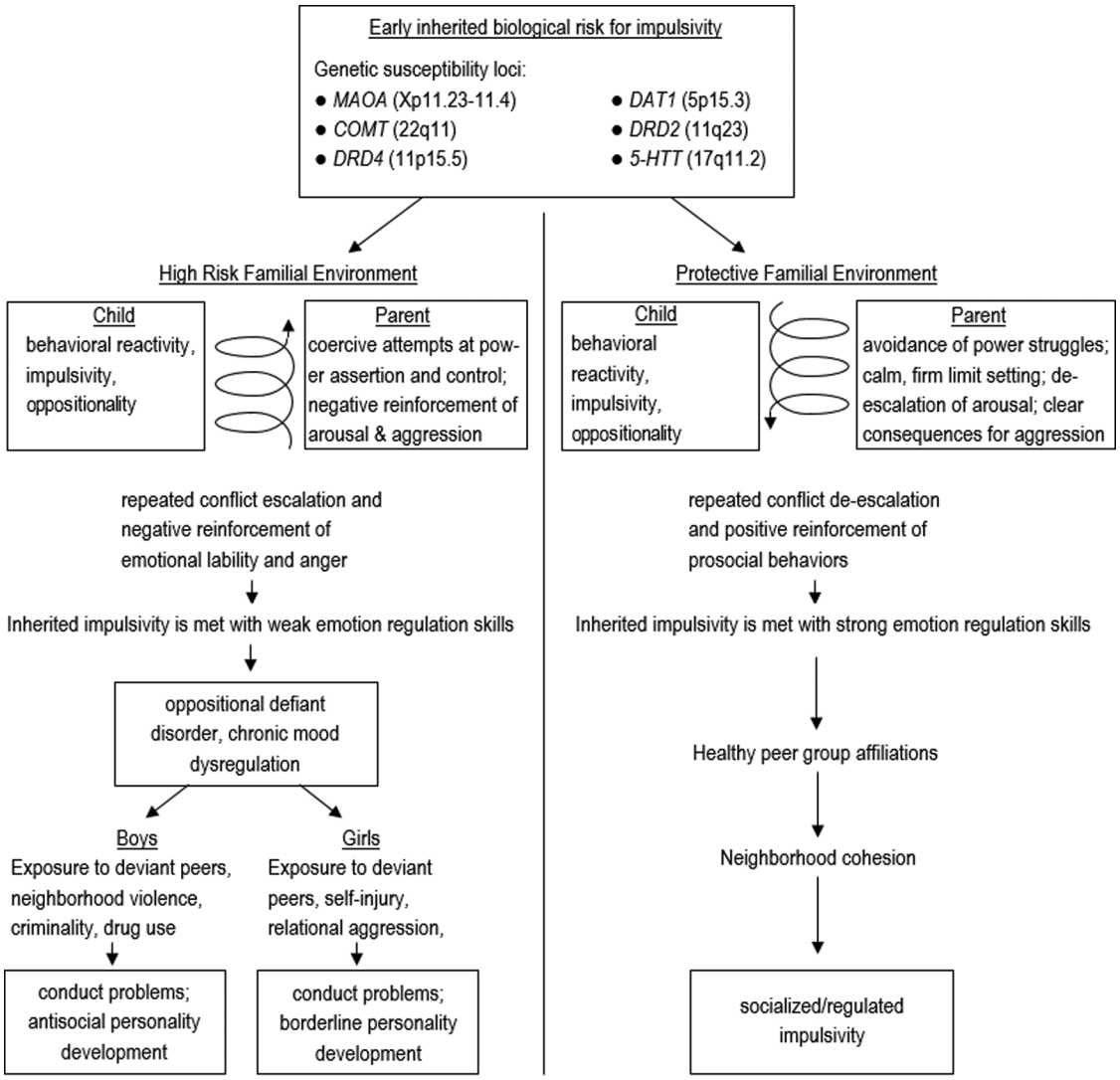

FIGURE 1 A biosocial developmental model of emotion dysregulation. *Note.* Genetic vulnerabilities for impulsivity interact with environmental risk factors to produce oppositionality and chronic emotional lability, which lead to antisocial behavior among boys and borderline behaviors among girls (left). In contrast, protective environments buffer vulnerable individuals from developing emotion dysregulation, so heritable impulsivity does not eventuate in psychopathology. Reproduced with permission from "Multifinality in the Development of Personality Disorders: A Biology × Sex × Environment Interaction Model of Antisocial and Borderline Traits" (Beauchaine et al., 2009, p. 758). Adapted with permission.

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A commonly used parent- and teacher-report measure is the Emotion Regulation Checklist (Shields & Cicchetti, 1997). Many other parent-report measures assess aspects of temperament (see Adrian et al., 2011), which can be problematic given that temperamental predispositions are mediated by subcortical neural networks that are largely independent of cortical emotion regulation networks (see earlier). Thus, tendencies toward behavioral withdrawal may reflect subcortically mediated temperamental inhibition *or* cortically mediated emotion regulation, depending on context. Similarly, tendencies toward behavioral approach may reflect temperamental impulsivity *or* emotion dysregulation. Accordingly, one recommendation for future research is that emotion dysregulation be evaluated with measures that were designed specifically to assess the construct, rather than measures that assess temperament.

The most commonly used physiological index of emotion dysregulation is RSA—which quantifies, usually via spectral analysis of the electrocardiogram R-R time series, parasympathetically mediated ebbing and flowing of heart rate across successive respiratory cycles (see Beauchaine, 2001, 2015; Zisner & Beauchaine, in press-b). Individuals who experience emotion dysregulation often exhibit low resting RSA (i.e., low vagal tone) and large reductions in RSA (i.e., vagal withdrawal) specifically to emotionally evocative stimuli. Indeed, abnormally low resting RSA and excessive RSA

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withdrawal to emotion evocation correlate with symptoms of both internalizing and externalizing psychopathology (see Beauchaine, 2001, 2012; Porges, 2007; Vasilev, Crowell, Beauchaine, Mead, & Gatzke-Kopp, 2009), and with a wide range of psychopathological outcomes, including anxiety (e.g., Hastings et al., 2008; Kemp et al., 2014; Thayer, Friedman, & Borkovec, 1996), phobias (e.g., Åhs, Sollers, Furmark, Fredrikson, & Thayer, 2009), attention problems (see Rash & Aguirre-Camacho, 2012), autism (Neuhaus, Bernier, & Beauchaine, 2014; Patriquin, Scarpa, Friedman, & Porges, 2013), callousness (de Wied, van Boxtel, Matthys, & Meeus, 2012), conduct disorder (Beauchaine et al., 2007; Beauchaine, Katkin, Strassberg, & Snarr, 2001), depression (e.g., Rottenberg, 2007; Rottenberg, Salomon, Gross, & Gotlib, 2005; Rottenberg, Wilhelm, Gross, & Gotlib, 2002), nonsuicidal self-injury (Crowell et al., 2005), panic disorder (e.g., Asmundson & Stein, 1994), trait hostility (Sloan et al., 1994), psychopathy (Hansen, Johnsen, Thornton, Waage, & Thayer, 2007), and schizophrenia (Clamor, Lincoln, Thayer, & Koenig, in press).[5] Moreover, comorbid internalizing and externalizing symptoms predict greater RSA withdrawal to emotion evocation than either internalizing or externalizing symptoms alone (Calkins, Graziano, & Keane, 2007; Pang & Beauchaine, 2013). In contrast, high RSA is associated with resilience among children who are exposed to adversity. For example, children with high RSA who witness marital conflict and hostility or are exposed to problem drinking by their parents appear to be buffered from the associated risk of developing both internalizing and externalizing behavior patterns (El-Sheikh, 2005; El-Sheikh & Erath, 2011; El-Sheikh, Harger, & Whitson, 2001; Katz & Gottman, 1995, 1997). Similarly, high RSA is associated better adjustment among children of depressed mothers, and among children who experience various forms of adversity (McLaughlin, Alves, & Sheridan, 2014; Shannon, Beauchaine, Brenner, Neuhaus, & Gatzke-Kopp, 2007). Furthermore, RSA is associated positively with children's social engagement (Fox & Field, 1989), with teacher reports of social competence (Eisenberg et al., 1995), and with expressions of empathy toward others who are in distress (Fabes, Eisenberg, & Eisenbud, 1993).

This brief review suggests that RSA and RSA reactivity mark one or more core self-regulatory functions that are disrupted across various forms of psychopathology (see Beauchaine, 2001, 2015; Beauchaine & Thayer, in press). Understanding the neurobiological bases of RSA, and determining whether these neural systems exhibit plasticity, may have important implications for treatment. These are therefore core questions in our research as we seek to alter trajectories toward psychopathology among vulnerable individuals (see Beauchaine et al., 2015; Zisner & Beauchaine, in press-b).

CENTRAL NERVOUS SYSTEM SUBSTRATES OF RSA

According to neurovisceral integration theory (Thayer, Hansen, Saus-Rose, & Helge Johnsen, 2009), low RSA and excessive RSA reactivity mark general vulnerability to psychopathology because, under appropriate stimulus conditions, they are peripheral indices of medial PFC function. This assertion is based on three principal considerations, including (a) the existence of inhibitory neural efferent pathways from the medial PFC to the PNS (the peripheral nervous system substrate of RSA), (b) positive associations between resting RSA and performance on executive function tasks, and (c) positive correlations between RSA and medial PFC activity in neuroimaging studies (see Beauchaine & Thayer, in press).

Inhibitory neural efferent pathways from the medial PFC to the PNS have been reviewed by several authors (see, e.g., Barbas, Saha, Rempel-Clower, & Ghashghaei, 2003; Ter Horst & Postema, 1997; Wong, Masse, Kimmerly, Menon, & Shoemaker, 2007). As described by Thayer et al. (2009), the medial prefrontal, insular, and cingulate cortices form an interconnected network that exhibits feedback and feed-forward connections with the amygdala. Activation of the central nucleus of the amygdala through this network inhibits the nucleus solitary tract, which in turn inhibits vagal motor neurons in the dorsal motor nucleus and the nucleus ambiguus. Together, these structures provide inhibitory input to the heart via PNS connections with the sinoatrial node (see Porges, 1995). Through this neural network, efficient PFC function produces high-tonic RSA, and well-regulated RSA reactivity. Because most forms of psychopathology are characterized by deficient PFC function (e.g., Maren, Phan, & Liberzon, 2013; Menon, 2011; Rubia, 2011), they are also characterized by low resting RSA and excessive RSA withdrawal to emotion evocation—both of which mark poor executive control (Thayer et al., 2009).

[5]As we have reviewed elsewhere (e.g., Zisner & Beauchaine, in press-b), findings linking reduced tonic RSA and excessive RSA withdrawal to psychopathology apply specifically to (a) clinical samples and (b) emotion evocation tasks. In contrast, in both normative and high-risk samples, symptoms of psychopathology are often correlated with greater tonic RSA, less RSA withdrawal during lab tasks, or no RSA withdrawal, especially when stimulus conditions are attention demanding and not emotionally evocative (e.g., Dietrich et al., 2007; Graziano & Derefinko, 2013; Obradović, Bush, Stamperdahl, Adler, & Boyce, 2010). Given considerable confusion associated with such discrepant findings, we expand on this topic in the Directions for Future Research section.

Consistent with this interpretation, positive correlations between resting RSA and performance on executive function tasks, which are mediated by the PFC, have been reported. Hansen, Johnsen, and Thayer (2003) median-split a sample of military personnel on RSA scores. Members of the high-RSA group outperformed members of the low-RSA group on stimulus detection and addition tasks. These findings were then replicated and extended in an independent sample (Hansen et al., 2003).

More convincingly, positive correlations between RSA and medial PFC function are observed in positron emission tomography studies. For example, across several emotion-induction tasks, Lane et al. (2009) found that RSA correlated with cerebral blood flow in both the medial PFC and the anterior cingulate cortex. As hypothesized, emotion induction reduced both RSA and blood flow in these regions. Such findings are consistent with the suggestion that RSA marks PFC function.

More recently, associations between RSA and executive function tasks have been reported among patients with panic disorder (Hovland et al., 2012). In addition, associations between RSA and both memory retrieval and cognitive control are observed during executive function tasks (Gillie, Vasey, & Thayer, 2014a, 2014b). RSA is also associated with PFC function as assessed via lesion studies (medial PFC; Buchanan et al., 2010), pharmacological blockade (both hemispheres; Ahern et al., 2001), functional magnetic resonance imaging (ventromedial PFC; see Thayer, Ahs, Fredrikson, Sollers, & Wager, 2012), retinal vessel analysis (Schuster, Jarczok, Fischer, Thayer, & Vossmerbaeumer, 2014), and cerebral blood flow assessed via arterial spin labeling (several regions; Allen, Jennings, Gianaros, Thayer, & Manuck, 2015).

Thayer and colleagues have demonstrated that associations between RSA and PFC activity are mediated in part by cortical-subcortical connectivity and that these connections are disrupted in patients with major depression. RSA correlates positively with BOLD signal activity in several regions of the PFC during affective set shifting among nondepressed but not depressed individuals (Lane et al., 2013). Over the course of 12 week treatment with sertraline, depressed patients evidence increases in RSA-PFC function correlations, and differences between depressed patients and controls disappear (Smith, Allen, Thayer, Fort, & Lane, 2014). Finally, connectivity between the rostral anterior cingulate and the pons correlate positively with RSA, and connectivity is correlated inversely with depression severity (Smith, Allen, Thayer, & Lane, in press). Thus, the ability of the PFC—particularly the medial PFC—to modulate activity in subcortical circuits, consistent with the model proposed herein, is disrupted in patients with major depression. Such findings suggest that RSA–PFC links are important in understanding both normal and psychopathological adjustment.

Nuerovisceral integration theory provides a potential explanation for well-replicated findings of low-resting RSA and excessive RSA withdrawal among psychopathological samples. Broad, nonspecific vulnerability to psychopathology is conferred through poor prefrontal control over behavior (Maren et al., 2013; Menon, 2011; Rubia, 2011), which is reflected in low RSA and excessive RSA reactivity given structural and functional interconnections between the PFC and PNS efferent pathways to the heart. Importantly, although general vulnerability to psychopathology is conferred by PFC dysfunction, more specific vulnerabilities are determined by subcortical neural systems, which determine whether an affected individual exhibits externalizing symptoms (mesolimbic dysfunction), internalizing symptoms (septo-hippocampal dysfunction), or both.

FUTURE DIRECTIONS IN EMOTION DYSREGULATION RESEARCH

So far in this article I have presented a developmental model of emotion dysregulation and made several key points about the existing state of research. *First*, emotion dysregulation is a hallmark—perhaps even the sine qua non—of psychopathology. As previously reviewed, emotion dysregulation is observed across the internalizing and externalizing spectra. Furthermore, it is observed in other disorders, such as schizophrenia, where, as in the case of internalizing and externalizing syndromes, weak subcortical functional connections with the PFC are found (e.g., Williams et al., 2004). *Second*, I have emphasized and reemphasized that subcortically mediated temperamental characteristics such as trait impulsivity and trait anxiety, although highly heritable, are insufficient, in and of themselves, to eventuate in psychopathology. Rather, only when these traits are coupled with deficiencies in emotion regulation, which are considerably socialized, does functional impairment ensue. *Third*, I have noted that emotion dysregulation is acquired through negative reinforcement processes that occur thousands of times across development in at risk families and peer groups, and through exposure to recurrent stress early in life. *Fourth*, I have suggested that these reinforcement processes, through mechanism of allostatis and neural plasticity, alter functioning of the PFC in ways that become highly canalized across development, resulting in (a) deficient top-down executive control over subcortically mediated impulses, and (b) altered patterns of autonomic nervous system (vagal) responding to emotion evocation. In the remainder of this article, I identify

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several directions for future research on emotion dysregulation among youth with psychopathology. I purposefully move from somewhat simple to more complex recommendations.

Distinguish Between Bottom-Up and Top-Down Forms of Self-Regulation

Consistent with a core theme of this article, future research should make strong distinctions between bottom-up, automated, subcortically mediated tendencies toward behavioral approach and withdrawal, versus top-down, volitional, cortically mediated behavior and emotion regulation. The importance of this distinction can be highlighted in part by way of example. Consider two children, both of whom engage in on task and socially competent behaviors most of their time at school. Child 1 has always done so, whereas Child 2 used to engage in considerable off-task behavior, including physical aggression, before completing an intervention that improves emotion regulation (e.g., Webster-Stratton, Reid, & Beauchaine, 2011). If the intervention was recent, it is likely that Child 2 is exerting top-down, volitional control over his or her behavior, whereas Child 1, because he or she has no externalizing predispositions, is not. Because Child 1 is not predisposed to such behaviors, no volitional control over temperamental response tendencies is required for regulation. Although this issue has been pondered by emotion regulation researchers for some time (see Cole et al., 2004), many researchers still treat bottom-up and top-down forms of self-regulation as equivalent.

Avoid Making Inferences About Emotion Regulation/Dysregulation From Cross-Sectional Samples of Behavior Alone

The preceding example illustrates the problem of making inferences about children's emotion regulation from cross sectional behavior samples. When we rely on behavior as the sole criterion variable in research on emotion regulation and dysregulation, we cannot determine whether a specific child's seemingly regulated conduct is due to (a) bottom-up, high temperamental withdrawal; (b) bottom-up, low temperamental approach; (c) some combination of both; or (d) top-down, volitional regulation of high temperamental approach and/or low temperamental withdrawal. In contrast, if we have knowledge about the child's past, including tendencies toward aggression prior to a successful intervention, we are on much stronger footing. Similarly, if we add physiological measures to our behavioral assessment, we are in a position to make inferences about emotion regulation by examining incongruence across methods. For example, if a child exhibits self-control despite large reductions in RSA during emotion evocation, volitional self-regulation might be inferred (see Cole et al., 2013).

A second reason to avoid reliance on behavioral measures alone when making inferences about emotion is that dysynchrony between behavioral and physiological response systems may itself indicate emotion dysregulation. For example, we compared time-linked correspondence between expressions of facial sadness and RSA reactivity during an empathy-eliciting task among boys with conduct problems and controls (Marsh, Beauchaine, & Williams, 2007). Although externalizing and control participants showed equivalent levels of facial sadness, the control group exhibited concordance between RSA reactivity and facial expressions, whereas the conduct problem group did not. Had we evaluated only facial sadness, we would have concluded that the externalizing and control groups exhibited similar emotional reactions to the task.

Conduct More Detailed Studies of Operant Reinforcement of Emotional Lability That Include Physiological Measures

As previously outlined, there is little doubt that negative reinforcement of emotional lability, via coercive family and peer processes, is a primary mechanism through which emotion dysregulation develops (e.g., Beauchaine & Zalewski, in press). Behaviorally, parents and their internalizing and externalizing children tend to match and exceed one another's invalidation and aversiveness levels, respectively, until unpleasant interactions are terminated (hence the term *escape conditioning*). However, despite the inherent social nature of invalidation and coercion, most studies conducted to date have evaluated patterns of emotion dysregulation within individuals, rather than within social relationships that amplify and maintain dysregulation. Using a multilevel actor–partner interdependence model, we recently evaluated concordance of observational and physiological measures of emotion dysregulation during interpersonal conflict among mother–daughter dyads, including depressed adolescents with or without a history of self-injury, and controls (Crowell et al., 2014). Behavior dysregulation was operationalized as coded aversiveness during a laboratory conflict discussion, and physiological dysregulation was indexed by RSA. Of interest, different patterns of concordance were found for control versus depressed participants. Control mothers and daughters were characterized by concordant *partner* (between-person) effects, showing increased physiological regulation (greater RSA) during minutes when their partner was more aversive. In contrast, clinical dyad members (both mothers and daughters) were characterized by a concordant *actor* (within-person) effects, becoming

simultaneously dysregulated in both physiological (reduced RSA) and behavioral (averseness) domains.

Although coordinated physiological responding among mothers and infants has been linked with maternal sensitivity (e.g., Ham & Tronick, 2009; Moore et al., 2009), which itself predicts development of positive behavior and emotion regulation among infants and very young children (e.g., Spanglar, Schieche, Ilg, Maier, & Ackermann, 1994), such studies remain quite rare among older children and adolescents, even though emotion regulation and dysregulation continue to develop well into adulthood (see earlier).

Improve the Internal Validity of Psychophysiological Research on Emotion Dysregulation

Internal validity refers to the degree to which a predictor (independent) variable, and not some alternative explanation, accounts for variance in a criterion (dependent) variable. In psychophysiological research, there is a long history of refining measurement methods and experimental manipulations to improve internal validity. However, many developmentalists and child psychopathologist are not trained formally in psychophysiological data acquisition and analysis. Nevertheless, many use these techniques given advances in hardware and software that make data collection and scoring much easier than is was even a decade ago. Next I list several common mistakes that I have noticed as a reviewer and an associate editor that when addressed, improve the internal validity of psychophysiological research.

Tonic versus phasic responding and true versus vanilla baselines. Tonic psychophysiological measures, by definition, are collected at rest, whereas phasic psychophysiological measures are quantified as change from rest. Preferably, tonic psychophysiological activity, regardless of the measure, is computed toward the end of a movement- and stimulus-free baseline, which must be long enough to induce a wakeful resting state (Obrist, 1981). Such "true" baselines reduce effects of both external (e.g., exposure to stress before a lab visit) and internal (e.g., individual differences in state anxiety) sources of error on measures of both tonic and phasic responding. Establishing true baselines is especially important in psychopathology research. For example, because excessive RSA reactivity is observed in almost all forms of psychopathology (see earlier), failure to collect true baseline RSA will result in underestimates tonic function, which in turn translate into overestimates of phasic RSA to subsequent tasks, potentially resulting in Type II errors (see Hastrup, 1986).

Baselines of 10–20 min may be needed to attain a true resting state (see Hastrup, 1986; Jennings, Kamarck, Stewart, Eddy, & Johnson, 1992). This has led many researchers, especially those who conduct research on children with psychopathology, to use alternatives, such as vanilla baselines, to assess tonic function. With vanilla baselines, participants either engage in a nominally demanding task, such as color detection (Jennings et al., 1992), or watch a calm, age-appropriate video (e.g., Sulik, Eisenberg, Silva, Spinrad, & Kupfer, 2013). In child psychopathology research, use of vanilla baselines usually follows from the assumption that young children, regardless of their level of functioning, cannot tolerate true baselines, or that children with a particular trait being studied (e.g., impulsivity), cannot tolerate true baselines (see e.g., Gavin & Davies, 2008).

Although these assumptions are not entirely without merit, vanilla baselines can be problematic for many psychophysiological measures, including RSA, the most commonly used peripheral index of emotion regulation (see earlier). Of importance, even moderate levels of attention allocation, such as those induced by vanilla baselines, reduce RSA among controls, but not among those with attention problems (see Beauchaine, 2001). In fact, RSA reactivity to lab tasks differs as a function of individual differences in both attention and impulsivity (e.g., Beauchaine et al., 2001). Vanilla baselines may therefore induce attention-related confounds in comparisons of tonic and phasic responding between controls and externalizing samples.

Fortunately, although 10- to 20-min true baselines are ideal, shorter baselines can be effective. In our work, we regularly use 5-min resting baselines with children ages 4 years and older who have clinical levels of attention problems (see Beauchaine et al., 2013; Crowell et al., 2006). We have children sit by themselves in a quiet room, facing a white wall, with no auditory or visual stimulation. Given the novelty of the lab visit, even preschoolers with ADHD can sit almost movement-free for this period. Stillness is required given the functional role of the PNS in supporting metabolic demands (see Bush, Alkon, Obradović, Stamperdahl, & Boyce, 2011).

An additional point regarding tonic versus phasic measures concerns proper use of terms toward facilitating effective communication. As previously noted, tonic measures, by definition, are collected at baseline, whereas phasic measures, by definition, reflect change from baseline. When we refer to phasic measures as tonic measures, literature-wide confusion may result. For example, many articles in the child psychopathology literature, including those published quite recently, use the term *vagal tone* to refer to any measure of RSA, whether tonic or phasic. This is problematic because tonic and phasic measures of RSA often have different external correlates and may reflect different physiological functions (see Beauchaine, 2001).

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Distinguish Between Constructs and Measures

As most readers are undoubtedly aware, a measure is a quantifiable, directly observable variable. In contrast, a construct is an unobservable variable that can only be inferred from multiple measures (see e.g., Nunnally & Bernstein, 1994). Emotion regulation is a construct that emerges from central nervous system processes that cannot be observed directly. Accordingly, it must be inferred from measures, whether behavioral, self-report, biological, or some combination thereof. When appropriate stimulus conditions are chosen (see next), low RSA and excessive RSA reactivity have proved to be reliable peripheral indices of emotion dysregulation. However, they are sometimes equated with emotion dysregulation itself, when they are best considered biomarkers of emotional lability. Emotion dysregulation is a psychological construct that can be assessed only with measures collected across levels of analysis.

Model development. Developmental shifts in tonic and phasic psychophysiological responding are conferred through various mechanisms, including increased body size and correlated alterations in cardiodynamics (e.g., Smulyan et al., 1998); age-related changes in physical fitness (e.g., Tulppo, Mäkikallio, Seppänen, Laukkanen, & Huikuri, 1998); maturation of the PFC, which is implicated in both emotion regulation and autonomic function (see earlier; e.g., Gogtay et al., 2004); migration of neural control over behavior to more fontal neural systems across development (Beauchaine & McNulty, 2013; Geier & Luna, 2009); age-associated changes in physiology (e.g., arterial hardening; Mitchell et al., 2004); and psychological maturation (e.g., more efficient control over attention; e.g., Karatekin, 2004). Given so many age-related influences, failure to model development is likely to introduce confounds and unwanted error variance into psychophysiological measures. Moreover, identifying both maturational effects and maturational failures has always been of direct interest to child psychopathologists and developmental psychopathologists (e.g., Sroufe & Rutter, 1984).

For healthy individuals, measures of heart rate variability, including RSA, increase linearly throughout infancy, childhood, and adolescence (see Massin & von Bernuth, 1997; Silvetti, Drago, & Ragonese, 2001; Zisner & Beauchaine, in press-b). After young adulthood, gradual decreases in RSA occur across the lifespan, except among those who remain physically fit (Byrne, Fleg, Vaitkevicius, Wright, & Porges, 1996). This increase in RSA should not be surprising given the measure's dependence on respiratory frequency, which decreases substantially from childhood to adulthood (see Fleming et al., 2011). Accordingly, it is essential for researchers to either (a) specify the appropriate frequency of respiration for participants' ages in computations of RSA using traditional fast-Fourier transformations (FFT), or (b) use auto-regressive spectral analysis, which identifies the observed respiration frequency of each individual (Kay & Marple, 1981; for an extended discussion, see Zisner & Beauchaine, in press-b). To date, many researchers have used respiratory frequencies that are appropriate for infants or very young children across all age ranges in both cross-sectional and longitudinal studies. This results in larger and larger misspecifications of RSA as individuals mature (others, including us in our early work, have used respiratory frequencies more appropriate for adults in research with child samples). Table 1 provides expected respiratory frequencies (at the 2–1/2 percentile, which captures most individuals) for children, 4–16 years of age (Wallis, Healy, Undy, & Maconochie, 2005). These values should be used to set the lower bound, or cutoff for the high-frequency component of heart rate variability (i.e., RSA). In longitudinal studies, different cutoffs should be used as children mature.

TABLE 1
Respiration Rates (2–1/2 Percentile) in Beats/Minute and Corresponding Hertz (Cycles per Second) for Appropriate "Windowing" of Frequency Domain Metrics of RSA

	Respiration Rate	
Age in Years	*Beats/Min*	*Hertz*
4	20	.33
5	19	.32
6	18	.30
7	17	.28
8	17	.28
9	16	.27
10	15	.25
11	14	.23
12	14	.23
13	13	.22
14	12	.20
15	12	.20
16	11	.18
Adult	9	.15

Note. Respiration rates evaluated among 1,109 children, as reported by Wallis et al. (2005).

Improve the External Validity of Psychophysiological Research on Emotion Dysregulation

External validity refers to the extent to which results from research can be generalized to other samples. One issue in particular merits discussion when considering the external validity of emotion dysregulation research with psychophysiological measures.

Reconcile differences in the developmental and clinical literatures. An important objective of child psychopathologists and developmental psychopathologists is to compare findings obtained from normative samples with findings obtained from clinical samples to (a) advance our understanding of typical and atypical development, and (b) specify mechanisms through which certain individual differences confer vulnerability to later psychopathology (see Cicchetti, 1984, 2006; Sroufe & Rutter, 1984). By conducting such group comparisons across multiple levels of analysis (e.g., genetic, neural, autonomic, social), we seek to identify etiological processes that further our basic understanding of psychopathology, and facilitate more effective intervention (e.g., Beauchaine & Gatzke-Kopp, 2012; Beauchaine, Neuhaus, Brenner, & Gatzke-Kopp, 2008; Cicchetti, 2008). Thus, when apparently different neurodevelopmental processes characterize typical and atypical samples, identifying causal mechanisms should be a high priority.

One such difference concerns relations between psychopathology scores and both tonic RSA and RSA reactivity. In contrast to findings just described in which children, adolescents, and adults with clinical levels of conduct problems, delinquency, and related externalizing behaviors exhibit excessive RSA withdrawal to emotion evocation (e.g., Beauchaine, Hong, & Marsh, 2008; Beauchaine et al., 2001, 2007; de Wied et al., 2012; Mezzacappa, Kindlon, Earls, & Saul, 1994), in both normative and high risk samples, externalizing symptoms are often correlated with higher tonic RSA, less RSA withdrawal during lab tasks, or no RSA withdrawal (e.g., Dietrich et al., 2007; Graziano & Derefinko, 2013; Obradović, Bush, Stamperdahl, Adler, & Boyce, 2010), especially when stimulus conditions are not emotionally evocative. For example, less RSA reactivity to positive film clips has been reported among those who score higher on externalizing symptoms compared with controls (Fortunato, Gatzke-Kopp, & Ram, 2013). In addition, Hinnant and El-Sheikh (2009) found a positive association between RSA change during problem solving and externalizing symptoms among middle schoolers. The consistency of such discrepant findings in clinical versus normative/high risk samples suggests that it is not arbitrary (see Beauchaine, 2009).

There are number of possible explanations for these consistently divergent findings. One is that there are different mechanisms of RSA-behavior relations at the extreme of externalizing scores than closer to the mean of the population distribution. Even in high-risk samples, most children do not meet criteria for clinical levels of conduct problems. Thus, most of the variance in correlations between externalizing scores and RSA reactivity is determined by participants who score less than 1 *SD* above the mean, where almost 85% of observed scores lie. In contrast, most participants in clinical groups by definition either meet criteria for conduct disorder or scored above the 95th percentile on other measures of externalizing behavior. If relations between externalizing symptoms and RSA are different at the extremes of the normal distribution, such a difference will be swamped in normative samples by individual variation among those whose scores fall much closer to the mean (see Beauchaine, 2009). Similar nonlinearities have been described in other neurobiological systems (e.g., Plichta & Scheres, 2014). A recommendation for future research is to identify possible alternative mechanisms of RSA-behavior relations among normative versus clinical samples and to look for differential relations in the tail of the externalizing distribution when sample size permits.

A related explanation concerns the meaning of an externalizing score at different points of a normal distribution. Individual differences in externalizing symptoms are distributed across the entire range of extremely low to extremely high, and low to intermediate scores do not suggest problem behavior. Most participants in both normative and high-risk samples score within normal ranges of externalizing symptoms. As we have noted elsewhere (Zisner & Beauchaine, in press-b), it makes little sense to conclude that a child who scores $T = 60$ (1 *SD* above the population mean) on the Child Behavior Checklist (Achenbach & Edelbrock, 1991) is exhibiting problematic behavior. Rather, externalizing scores in this range more likely reflect temperamental exuberance, positive affectivity, social competence, and adaptive engagement with the environment (see Degnan et al., 2011). Of importance, across numerous studies, these very traits have been associated with modest RSA withdrawal to emotion evocation and attention demanding tasks (e.g., Marcovitch et al., 2010; Richards, 1987; Suess, Porges, & Plude, 1994). To summarize, measures that were designed to assess psychopathology do not capture problematic behavior among those who fall within normal population ranges. Apparent associations between low RSA reactivity to behavioral challenge and behavior "problems" may therefore be misinterpretations.

Choose appropriate stimulus conditions. Third, differences in stimuli may be at play. The functions and CNS origins of psychophysiological responses cannot be inferred without considering stimulus conditions within which psychophysiological responses are evoked (see Beauchaine, 2012; Beauchaine & Gatzke-Kopp, 2012; Brenner & Beauchaine, 2011). Given the astounding complexity of the brain, it is capable of considerably more variegated responding than the PNS. However, because the PNS is a final common pathway between

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CNS responding and many behavioral responses, what might appear to be identical PNS responses can originate from different neural systems. Attempts to link externalizing behavior and RSA reactivity have been conducted using many stimulus conditions, including emotion evocation (e.g., Beauchaine et al., 2001), attention allocation (e.g., Suess et al., 1994), executive function (e.g., Marcovitch et al., 2010), positive mood induction (e.g., Fortunato et al., 2013), and others. Some authors also average RSA scores across a variety of unrelated lab tasks that undoubtedly evoke different CNS responses. As we have reviewed elsewhere (Zisner & Beauchaine, in press-b), externalizing behaviors are associated with excessive RSA withdrawal *specifically during emotion evocation*—not necessarily during other types of tasks. This reflects the emotion dysregulation aspect of externalizing behavior (see Beauchaine et al., 2007; Beauchaine et al., 2009; Beauchaine & Gatzke-Kopp, 2012). In contrast, less RSA withdrawal is found among externalizing samples than controls during attention demanding, executive function, and problem-solving tasks (e.g., Dietrich et al., 2007; Hinnant & El-Sheikh, 2009; Obradović et al., 2010). This likely reflects attentional difficulties among externalizers (see Beauchaine, 2001; Rash & Aguirre-Camacho, 2012).

Integrate Emotion Dysregulation Into Factor Analytic and Behavioral Genetic Models of Psychopathology

Psychopathology researchers have long sought to advance our understanding of mental illness by factor analyzing symptoms among both population-based cohorts and large twin registries. For example, Krueger (1999) factor-analyzed *Diagnostic and Statistical Manual of Mental Disorders* (3rd ed., rev.; American Psychiatric Association, 1987) symptoms of a wide range of psychiatric syndromes among participants in the National Comorbidity Survey ($N = 8{,}098$). Within split-half, male only, female only, and treatment-seeking subsamples, two higher order factors were found. Consistent with existing models in the child psychopathology literature (e.g., Achenbach & Edelbrock, 1991), Krueger labeled these factors internalizing and externalizing. The internalizing factor, which was comprised of two lower order factors (anxious misery and fear) in the split-half and male/female only subsamples, included major depression, dysthymia, generalized anxiety, panic disorder, and phobias. The externalizing factor included alcohol dependence, drug dependence, and antisocial personality disorder. Additional factor analyses conducted since provide support for Krueger's initial findings (e.g., Krueger, Markon, Patrick, Benning, & Kramer, 2007; Krueger et al., 2002) and extend the externalizing factor to include hyperactivity/impulsivity, oppositional defiant disorder, and conduct disorder (e.g., Lahey, Van Hulle, Singh, Waldman, & Rathouz, 2011; Tuvblad, Zheng, Raine, & Baker, 2009). More recent work demonstrates that thought disorder emerges as a third higher order factor when psychotic symptoms are included (e.g., Wright et al., 2013).

Consistent with a major thesis of this article, twin studies indicate that both internalizing and externalizing liabilities are highly heritable, but that specific behavioral syndromes are shaped considerably by environments (Krueger et al., 2002; Lahey et al., 2011; Tuvblad et al., 2009). Thus, whether a child who inherits trait impulsivity (i.e., liability to externalizing disorders; see Beauchaine & McNulty, 2013) develops ADHD or more serious externalizing conduct depends largely on environmental experience (see Figure 1). Similarly, whether a child who inherits trait anxiety develops an anxiety disorder, and if so, which specific anxiety disorder they acquire, depends on environmental context (see Kagan, 2013).

Higher order factors of trait impulsivity (externalizing) and trait anxiety (internalizing) map onto individual differences in functioning of subcortical neural networks that govern approach and avoidance behaviors, respectively (see earlier). As outlined thus far, however, factor analytic models of psychopathology are incomplete because they do not account for emotion dysregulation, which, as we note throughout this article, is cortically mediated and observed in most forms of psychopathology. Thus, contemporary factor analytic and behavioral genetic models of psychopathology should attempt to account for both subcortically mediated vulnerabilities (trait impulsivity [externalizing], trait anxiety [internalizing]) and cortically mediated vulnerabilities (emotion dysregulation).

Recently articulated factor analytic work may accomplish this objective (see Beauchaine & Thayer, in press). Such studies indicate that symptoms of psychopathology are best accounted by a bifactor measurement model that includes a superordinate *p* factor (Caspi et al., 2014; Lahey et al., 2012), in addition to broadband internalizing and externalizing factors. This *p* factor is associated with both functional impairment and executive function deficits—both of which are expected if the factor marks PFC-mediated self- and emotion dysregulation.

In the Caspi et al. (2014) study, 1,037 adults from the Dunedin Multidisciplinary Health and Development Study were assessed longitudinally at ages 18, 21, 26, 32, and 38 years. The bifactor measurement model depicted in Figure 2 provided a good fit to their symptoms. Although externalizing vulnerability, internalizing vulnerability, and thought disorder were all associated with impairment, much of this impairment was

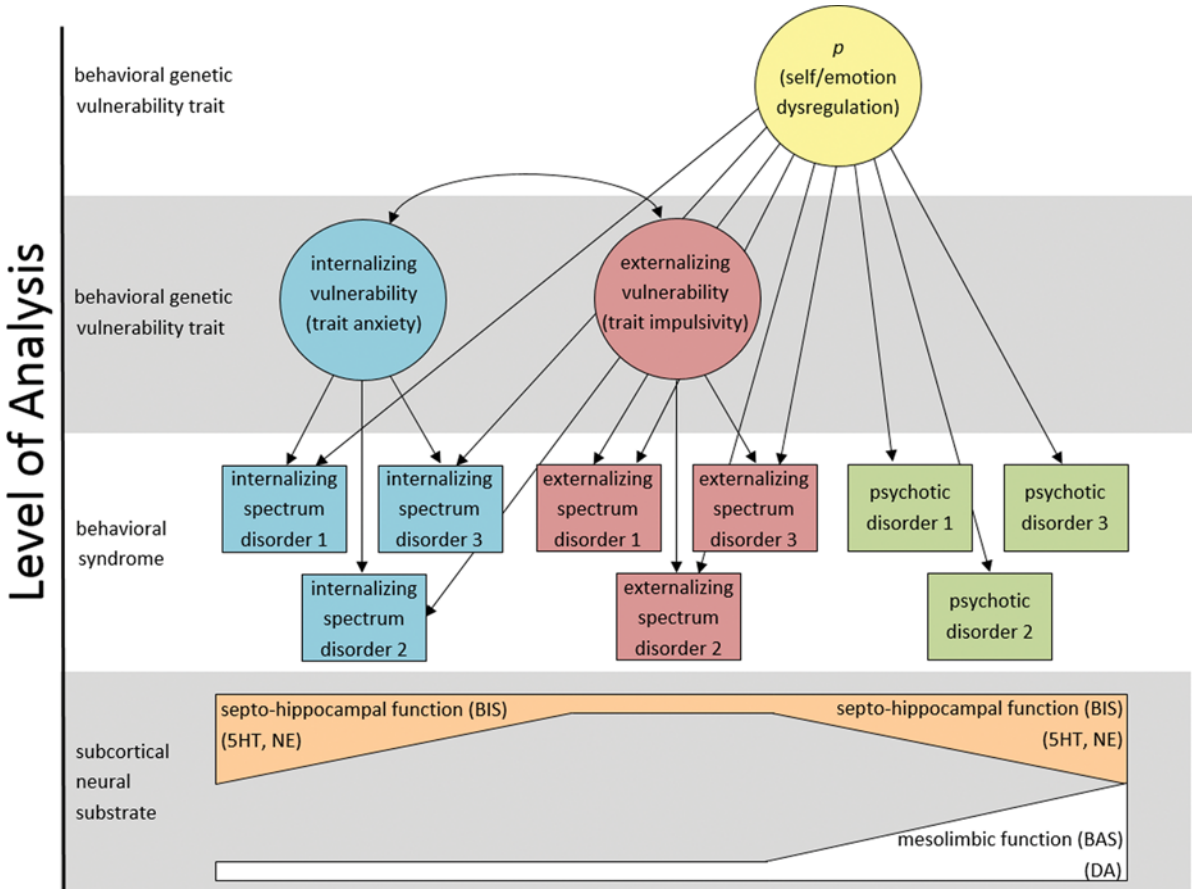

FIGURE 2 Multidimensional conceptualization of psychopathology that includes Caspi et al.'s (2014) *p* factor. Adapted from Beauchaine and Thayer (in press).

accounted for by *p*. Indeed, those who scored highest on *p* exhibited the greatest life impairment. Moreover, *p* was associated more strongly with personal histories of child maltreatment, and with family histories of depression, anxiety, psychosis, conduct disorder, antisocial personality disorder, and substance dependence than were externalizing or internalizing factor scores. Perhaps more important, high *p* scores correlated negatively with cognitive function, as assessed by IQ, executive function tasks, attention, and memory—all of which are subserved by the PFC (Stuss & Knight, 2013)—and all of which are important for self- and emotion regulation. Some of these impairments were evident at ages 3 and 10 years in retrospective analyses. Thus, consistent with previous discussion, trait impulsivity and trait anxiety are far less functionally impairing in the absence of co-occurring PFC dysfunction, as represented by *p*. Without PFC dysfunction and associated deficiencies in self- and emotion regulation, trait impulsivity, and trait anxiety are likely to confer individual differences in personality, not psychopathology (see Beauchaine & Gatzke-Kopp, 2012; Beauchaine & Thayer, in press).

Identify Molecular Genetic Risk for Emotion Dysregulation

Psychiatric genetics is plagued by inconsistent findings, nonreplications, and large gaps between heritability estimates derived from behavioral genetics studies, and variance accounted for in phenotypes by molecular genetic candidates (see, e.g., Beauchaine & Gatzke-Kopp, 2013). Thus, even for highly heritable traits such as impulsivity and anxiety, the molecular genetic bases of which have been studied extensively (for recent reviews, see Gizer, Otto, & Ellingson, in press; Hovatta & Barlow, 2008), much remains to be learned about effects of specific genes, Gene × Gene interactions, and Gene × Environment interactions. Of importance, identifying Gene × Gene and Gene × Environment interactions requires very large sample sizes, especially when effect sizes are small, alleles are stratified across subsamples (e.g., race, ethnicity), and reliabilities of phenotypes are modest. This is an important point because many studies do not have adequate power to detect interaction effects (see, e.g., Whisman & McClelland, 2005). Thus, progress on the molecular genetics of emotion

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regulation is likely to be slow and painstaking and will not be a simple matter of collecting genetic data on typically sized samples. Furthermore, multimodal measurement of emotion regulation is more difficult than measuring constructs such as trait impulsivity and trait anxiety, which can be assessed reliably via informant reports. For reasons just articulated, emotion regulation is best captured across multiple levels of analysis.

With these caveats aside, even though socialization plays an important role in the acquisition of emotion regulation (see earlier; Beauchaine et al., 2007; Beauchaine et al., 2009), the construct is also partly heritable (see Goldsmith et al., 2008). One logical strategy for identifying heritable substrates of emotion regulation is to identify neurobiological correlates of resilience among maltreated children. By definition, these children are functioning well across multiple domains despite past experiences of adversity. As reviewed recently by Cicchetti (2013), this strategy and others have identified both neural systems (e.g., the hypothalamic-pituitary adrenal axis) and genetic polymorphisms (e.g., *MAOA, CRHR1, 5-HTTLPR, 521C/T, OXTR*) that are associated, at least in some studies, with resilience. It bears repeating, however, that very few studies have measured emotion regulation per se (recall that the absence of problem behavior is insufficient for inferring emotion regulation; see earlier). Thus, the molecular genetics of emotion regulation/dysregulation is in a nascent stage of development. Future research studies will need to be designed in which emotion regulation and dysregulation are measured very carefully across levels of analysis, and their interactions with specific genetic variants are evaluated in large samples. Any positive findings from such studies will need to be replicated given that failures to replicate are so common in psychiatric genetics.

Expand Neuroimaging Research on Emotion Dysregulation Among Children and Adolescents

A final recommendation for future research on emotion regulation/dysregulation with children and adolescents is to expand the use of neuroimaging technology. fMRI is used routinely in emotion regulation research with adults, where certain paradigms have emerged as gold standards of assessment. For example, reappraisal tasks, which require participants to interpret highly negative stimuli in unemotional terms, indicate that volitional control of emotion is subserved by the medial PFC and its interconnections with the amygdala (e.g., Ochsner, Bunge, Gross, & Gabrieli, 2002). Thus, consistent with a primary theme of this article, effective emotion regulation requires top-down, prefrontal inhibition of subcortical structures that generate affective experiences (e.g., Ochsner & Gross, 2005). As previously noted, and similar to findings reported among adults, a limited number of neuroimaging studies indicate that children and adolescents with anxiety disorders exhibit less functional connectivity in amygdalar-prefrontal connections than controls (e.g., Monk et al., 2008) and that adolescents with externalizing disorders exhibit less functional connectivity in striatal-prefrontal connections than controls (e.g., Shannon et al., 2009). Of interest, anxiety-disordered adults also require greater PFC involvement than controls to effectively regulate negative emotion (e.g., Campbell-Sills et al., 2011).

The neuroimaging literature on emotion regulation among adults with psychopathology is voluminous, but far fewer studies have been conducted with children and adolescents with psychopathology. Studies of reappraisal, which are common among adults, should be extended to child and adolescent clinical samples in efforts to map the neurodevelopment of volitional control over emotion in high-risk groups. Among typically developing children, reappraisal ability increases with age, as does neural activity in the ventrolateral PFC during reappraisal (McRae et al., 2012). Although structural neuroimaging studies indicate aberrant gray matter pruning in the PFC among delinquent adolescents (De Brito et al., 2009), which may portend co-occurring functional deficiencies in reappraisal and its neural correlates, direct assessments of delinquent samples are lacking.

Numerous studies indicate that trait impulsive, male children and adolescents with ADHD exhibit hypo-responding in the ventral striatum, a subcortical structure implicated in trait impulsivity (for a review, see Plichta & Scheres, 2014). However, few studies have evaluated prefrontal modulation of striatal responding among those with ADHD (for an exception, see Sauder et al., 2009). Future research that includes functional connectivity analyses will be important in evaluating the integrity of top-down neural control over subcortical, striatal neural activity among those who are trait impulsive. Such functional connections are essential for learning and cognition (e.g., Antzoulatos & Miller, 2014; van den Bos, Cohen, Kahnt, & Crone, 2012), and are likely important for regulating trait impulsivity.

CONCLUSIONS

Emotion dysregulation research has burgeoned in the last two decades following (a) improvements in technologies for verifying, quantifying, and inferring emotional states from autonomic and central nervous system processes, and (b) an associated paradigm shift toward emotional models of psychological experience and away from strict behavioral and cognitive models. During this

time, much has been learned about interactions among subcortical neural circuits that generate emotion, and cortical neural circuits that regulate emotion (e.g., Cardinal, Parkinson, Hall, & Everitt, 2002). Effective emotion regulation requires efficient top-down, prefrontal control over bottom-up, subcortically mediated individual differences in trait impulsivity and trait anxiety. Without making critical distinctions between heritable individual differences in trait anxiety and trait impulsivity, versus socialized deficiencies in emotion regulation, progress in understanding the development of psychopathology among children and adolescents will be hampered. In this article, I have offered suggestions for improving future research on emotion dysregulation by (a) distinguishing between bottom-up and top-down forms of self-regulation, (b) using measures of emotion dysregulation that span behavioral and biological levels of analysis, (c) specifying physiological mechanisms through which operant reinforcement and exposure to adversity shape emotional lability, (d) improving the internal and external validity of psychophysiological measures, (e) integrating the emotion dysregulation construct into factor analytic and behavioral genetic models of psychopathology, (f) identifying molecular genetic risk for emotion dysregulation, and (g) expanding neuroimaging research on emotion dysregulation among children and adolescents. Conceptually, none of these suggestions are difficult. Some, however, will be expensive given the need for larger sample sizes, as in the case of molecular genetics, and more sophisticated assessment methods, as in the case of neuroimaging. Others suggestions simply require more care in construct operationalization and model building. Together, I hope that these and additional strategies recommended by others will continue to advance research on emotion regulation/dysregulation in the upcoming decade.

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