

Special Issue Editorial

Emotion dysregulation and emerging psychopathology: A transdiagnostic, transdisciplinary perspective

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In the past quarter century, emotion dysregulation has emerged as an increasingly important construct for understanding diverse adjustment problems in childhood, adolescence, and adulthood. Emotion dysregulation is now recognized across disciplines and theoretical perspectives as a transdiagnostic feature of various mental health outcomes, and is represented in multiple elements of the Research Domain Criteria matrix (e.g., Beauchaine, 2015; Bradley et al., 2011; McLaughlin, Hatzenbuehler, Mennin, & Nolen-Hoeksema, 2011; National Institute of Mental Health, 2019; Sheppes, Suri, & Gross, 2015). This recognition follows from the observations that (a) one or more forms of dysregulated emotion are observed across all empirically derived structural dimensions of psychopathology, including internalizing disorders, externalizing disorders, and psychotic disorders (see Beauchaine, 2001; Beauchaine & Zisner, 2017; Gross & Jazaieri, 2014; Hostinar & Cicchetti, 2019); (b) emotion dysregulation is a common feature of personality disturbance, and is therefore one of four general criteria for personality disorders (American Psychiatric Association, 2013; Trull, 2012); (c) disrupted top-down modulation of emotional reactivity is observed in many forms of psychopathology, even though functional subdivisions of the prefrontal cortex (PFC) that subserve emotion regulation differ depending on the class of emotion considered (see below; Beauchaine, 2015; Palacios-Barrios & Hanson, 2019); and (d) emotion dysregulation in childhood and adolescence confers prospective vulnerability to psychopathology in adulthood (see Cole, Hall, & Hajal, 2017). Thus, specifying and altering complex transactions through which endogenous vulnerabilities transact with social dynamics to reinforce emotion dysregulation and canalize its neuroplastic substrates is of utmost importance to those who seek to prevent and treat various forms of mental illness (e.g., Beauchaine, Constantino, & Hayden, 2018; Beauchaine, Hinshaw, & Bridge, 2019; Eisenberg et al., 2001; Graziano, Keane, & Calkins, 2010; Thompson & Meyer, 2007).

Genesis of Emotion Dysregulation Research

Burgeoning interest in emotion regulation and dysregulation was initiated in part by a collection of papers published in a 1994 *Monograph of the Society for Research in Child Development*

(Fox, 1994a). At the time, most scholars were circumspect about emotion as a topic of scientific inquiry given longstanding proscriptions against studying constructs that were defined exclusively by subjective self-reports. The monograph, which was devoted specifically to development of emotion *regulation*, included a series of articles demonstrating that emotional states can be inferred, quantified, and verified through careful assessment of neurohormonal (Stansbury & Gunnar, 1994), electro-physiological (Dawson, 1994; Fox, 1994b), and cardiovascular function (Porges, Doussard-Roosevelt, & Maiti, 1994). A year later, a special issue of *Development and Psychopathology* appeared in which several prominent scholars contributed articles specifying neurohormonal and psychophysiological correlates of both typical and atypical emotional development (Cicchetti, Ackerman, & Izard, 1995; Hart, Gunnar, & Cicchetti, 1995; Katz & Gottman, 1995; Zahn-Waxler, Cole, Welsh, & Fox, 1995). Soon thereafter, neuroimaging assessment of emotion became mainstream among adults and eventually among children and adolescents (see Cicchetti & Thomas, 2008; Davidson, Jackson, & Kalin, 2000). These developments showed that emotions can be quantified objectively, which rendered emotional processes, including emotion dysregulation, legitimate topics of inquiry. Although addressed in only 37 scientific papers prior to 1994, emotion dysregulation has been the topic of 2,782 articles since (Web of Science, 2019). Most scholars now agree that emotional processes are best captured by research conducted across levels of analyses, including neurobiological, self-report, and behavioral (see Adrian, Zeman, & Veits, 2011; Beauchaine, 2015).

Defining Emotion and Emotion Dysregulation

Scientists who study chronometry and other dynamics of affective experience and expression often distinguish between (a) *emotions*, which are elicited by specific stimuli and events (both endogenous and exogenous), persist from seconds to minutes, and motivate immediate action or inaction; and (b) *moods*, which are imbued by broader internal and external milieus, persist from hours to days and weeks, and bias patterns of cognition and behavior over time (see Fox, 2018). Thus, fluctuations in emotion may influence mood, and mood may bias emotional responses, but mood and emotion are not isomorphic. Although no clear line demarcates emotion from mood, the distinction is nevertheless important for understanding differences between emotional reactions to discrete events (e.g., craving, desire, fear, and sadness)

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versus persistent mood states that transcend immediate situational contexts (e.g., mania, anhedonia, and depression). A practical illustration of the distinction between mood and emotion comes from research on depression, where patients often show *less* negative emotional reactivity to sad stimuli than healthy controls (Rottenberg, 2005). Thus, emotion and mood sometimes fractionate, and mood disturbance is not necessarily attributable to emotion dysregulation (Sheppes et al., 2015).

As is the case for emotion, it is important to define emotion dysregulation carefully. In our own work, we operationalize emotion dysregulation as, “a pattern of emotional experience and/or expression that interferes with appropriate goal-directed behavior” (Beauchaine, 2015, p. 876). This somewhat inclusive definition both follows from and contrasts with situationally functional accounts of emotion regulation, which emphasize emotions as effective initiators, maintainers, and modulators of goal-directed behavior (Campos, Mumme, Kermoian, & Campos, 1994; Cole, Martin, & Dennis, 2004; Thompson, 1990).

Four aspects of our definition of emotion dysregulation are worth elaborating. First, we purposefully emphasize emotion over mood, consistent with distinctions outlined above. Second, our definition is agnostic regarding evolutionary functions of behavior and emotion, and instead refers to “appropriate” goal-directed behavior. In this context, “appropriate” behaviors are those that are *situationally functional* (see, e.g., RamsOOK, Cole, & Fields-Olivieri, 2019). For example, well-mannered, emotionally stable behavior in the classroom is situationally functional in Western culture, whereas impulsive and emotionally exuberant behaviors are often viewed as dysfunctional. However, such impulsivity and exuberance were likely not maladaptive in our environments evolutionary adaptation. Such behaviors likely conferred selective advantages in certain environments (see Mead, Beauchaine, & Shannon, 2010). This example illustrates an important difference between situational and evolutionary functionalism. Emotion researchers are sometimes criticized for subscribing to evolutionary functionalism (e.g., Barrett, 2017), when they instead endorse situational functionalism (Beauchaine & Haines, 2019).

Third, situationally functional and dysfunctional behaviors, including expressions of emotion, often differ across cultures. Emotional reactions that are expected in some cultures may be viewed as threatening, undercontrolled, apprehensive, and so on, in others, and thereby evoke reprisals, social rejection, and other consequences that impede goal-directed behavior. The functional value of expressed emotion therefore cannot be assessed independent of cultural context (e.g., Bhugra & McKenzie, 2003; Mauss & Butler, 2010).

Fourth and finally, implicit in our definition is acknowledgment that many emotional reactions are *afunctional*. For example, fairly intense solitary displays of anger, such as those evoked when one narrowly avoids a traffic accident, may serve no function or dysfunction whatsoever (Beauchaine & Haines, 2019). Thus, whether an emotional reaction is functional, dysfunctional, or afunction; regulated, dysregulated, or unregulated, often depends on eliciting contextual events, and match or mismatch between context and expressive intensity (Aldao, 2013).

Neural Models of Emotion Dysregulation I: Evolutionary Functionalism

Two competing neural theories of emotion dysregulation follow from separate intellectual traditions. According to the more

conventional perspective, broad classes of emotion, including approach (e.g., wanting, enthusiasm), avoidance (e.g., apprehension, fear), and social affiliative (e.g., compassion, affection), evolved to motivate adaptive, survival-related behaviors (e.g., Keltner & Gross, 1999). Evolutionary functionalists propose that subcortical neural circuits subserving approach, avoidance, and social affiliation are preserved across species because they were selected in our environments of adaptation (e.g., Panksepp, 2016). Circuits involved in generating appetitive emotions include the striatum (ventral and dorsal) and its afferent projections from the ventral tegmental area (e.g., Volkow, Wise, & Baler, 2017). In contrast, circuits involved in generating aversive emotions include the septo-hippocampal system and its afferent projections from the amygdala (see Corr, 2013; Strange, Witter, Lein, & Moser, 2014).

Over 50 years of comparative research with animals and humans has yielded general agreement regarding principal functions of these circuits in mediating approach and avoidance emotions and motivating approach and avoidance behaviors (see Beauchaine & Zisner, 2017). It should be noted, however, that subcortical neural systems are structurally interconnected and functionally interdependent (see Beauchaine & Constantino, 2017; Beyeler, 2016; Corr, 2013). For example, the nucleus accumbens (a ventral striatal structure) and the amygdala share interconnections via the paraventricular nucleus and the stria terminalis (e.g., Dong, Li, & Kirouac, 2017). Furthermore, the nucleus accumbens responds to punishment as well as reward, and the amygdala responds to reward as well as punishment (see, e.g., Sauder, Derbidge, & Beauchaine, 2016; Schultz, 2016). Functional specificity of approach and avoidance circuits is therefore relative, not categorical.

According to the traditional perspective, humans behave at the behest of their emotions in life-threatening situations and other survival-relevant contexts (e.g., when one’s children are in danger or when food is scarce). In contrast, given highly elaborated PFCs, humans can suppress emotional responses and engage in more deliberative actions when doing so is deemed advantageous. In such situations, subcortical circuits that subserve emotion-generating functions are modulated by cortical circuits that subserve emotion-regulating functions. This interpretation is consistent with literature linking top-down prefrontal and orbitofrontal control to executive function, self-regulation, and emotion regulation (e.g., Beauchaine, 2015; Etkin, Büchel, & Gross, 2015; Heatherton, 2011; Zelazo, 2015). Dozens of neuroimaging studies show increased activation in functional subdivisions of the prefrontal, anterior cingulate, and insular cortices during volitional downregulation of negative affect (see, e.g., Tone, Garn, & Pine, 2016; Zilverstand, Parvaz, & Goldstein, 2017).

Similarly, effortful reappraisal of negative emotion yields patterns of responding across a distributed network of prefrontal structures (Goldin, McRae, Ramel, & Gross, 2008). Poor top-down functional connectivity between these structures and subcortical regions implicated in generating approach and avoidance emotions is observed in externalizing and internalizing disorders, respectively (Gold et al., 2016; Korponay et al., 2017; Kujawa et al., 2016; Qing et al., 2012; Shannon, Sauder, Beauchaine, & Gatzke-Kopp, 2009). Moreover, emotional lability is associated with poor top-down control of the amygdala by the medial PFC and reduced functional connectivity between the amygdala and the orbitofrontal cortex (Churchwell, Morris, Heurtelou, & Kesner, 2009; Hilt, Hanson, & Pollak, 2011). Collectively, these findings support the notion that emotion dysregulation derives

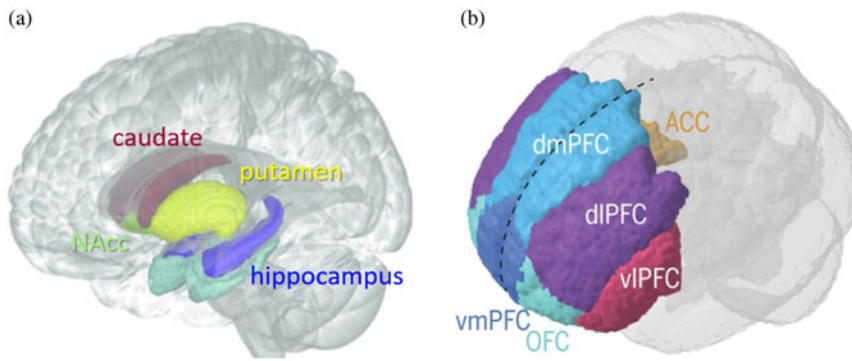


Figure 1. (a) Subcortical and (b) cortical neural structures implicated in emotion generation and emotion regulation. Panel a shows the nucleus accumbens (NAcc), part of the ventral striatum; the caudate nucleus and putamen, parts of the dorsal striatum; and the hippocampus, part of the septo-hippocampal system. The amygdala (not pictured) is a small neural structure positioned at the anterior (forward) face of the hippocampus. Adapted with permission from Krishnan, Watkins, and Bishop (2016). Panel b shows functional subdivisions of the prefrontal cortex, including the anterior cingulate cortex (ACC), the dorsomedial prefrontal cortex (PFC), the dorsolateral PFC, the ventrolateral PFC, the ventromedial PFC, and the orbitofrontal cortex (OFC). Adapted with permission from Carlén (2017).

at least in part from inadequate prefrontal modulation of subcortical neural responding. Subcortical and cortical neural structures implicated in emotion generation and emotion regulation are depicted in Figure 1.

Neural Models of Emotion Dysregulation II: Constructionism

More recently, constructionist models of emotion dysregulation have been described (Barrett, Wilson-Mendenhall, & Barsalou, 2013; Leshin & Lindquist, 2019). In general, constructionists eschew the notion that emotions serve evolutionary functions, and question the value of comparative animal research for making inferences about human emotion and behavior (e.g., Barrett, 2017; Barrett et al., 2013). Constructionists sometimes argue that emotions are distinctly human experiences without homologues in the broader animal kingdom (Leshin & Lindquist, 2019). According to this perspective, experiential states including perception, cognition, and emotion arise from interactions among sensory and neural processes, which humans interpret and categorize based on learning and prior experience (see Barrett, 2009). Constructionist theory specifies *core affective processes*, which transcend various emotional states. In contrast to specific emotions (e.g., sadness, anger, and fear), core affective processes are more general, and include valence (positivity–negativity) and arousal (activation–deactivation). Through recurrent pairings of core affective processes with neural, sensory, and perceptual inputs from our environments, we come to associate core affect with higher order representations of specific emotions (Russell & Barrett, 1999). Neural substrates of core affective processes are presumed to be universal among humans, present at birth, and dependent on the same circuits as decision making and other psychological processes (Duncan & Barrett, 2007).

Constructionist theories have garnered considerable attention in recent years (see Barrett, 2017; Leshin & Lindquist, 2019), and now rival functionalist perspectives as explanatory models of emotion. To date, however, constructionists have written relatively little about emotion dysregulation (Beauchaine & Haines, 2019). This is likely because constructionists view emotions as emergent properties of complex neural systems. Emotions are therefore by-products of highly individualized, experience-dependent neural response patterns. Each person’s unique learning history confers cognitive-affective schemas, attributional biases, and stimulus-response associations that contribute to emotional experience and expression. Thus, emotion and emotion dysregulation are not differentiable because they arise from the same idiosyncratic learning histories and neural processes (see Papa & Epstein, 2018).

More specifically, constructionists argue that emotion dysregulation arises from the same neural processes as core affect

(e.g., valence and arousal), combined with disruptions in *situated conceptualization* (Barrett et al., 2013). This refers to “the brain [as] a situated processing architecture, designed to process situations in the moment and to simulate non-present situations in thought” (Barsalou, 2016, p. 6), including assessment of what immediate events represent, deciding how to act upon such events, and predicting what core affective processes to expect as a consequence. Emotion dysregulation could arise from highly routinized conceptualizations that are not situation dependent (Barrett et al., 2013). Nonsituational conceptualizations may stem from disrupted memory function, altered autonomic regulation, or poor attention deployment, among other processes. These and other deficits correlate with disrupted intrinsic neural network connectivity in the salience network and the frontoparietal network, as seen in diverse forms of psychopathology (Barrett & Satpute, 2013). Thus, although specific foci differ across functionalist and constructionist perspectives, both emphasize connectivity deficits toward explaining emotion dysregulation. Moreover, situational functionalism and constructionism both emphasize the importance of context in shaping and interpreting emotion (see Beauchaine & Haines, 2019).

Neurodevelopmental Considerations

An important consideration for any developmental theory of emotion dysregulation concerns differential neuromaturation of subcortical structures depicted in Figure 1a versus cortical structures depicted in Figure 1b. Neurodevelopment of the PFC lags several years behind neurodevelopment of subcortical structures (Brain Development Cooperative Group, 2012; Casey, Getz, & Galvan, 2008; see also Hauser, Will, Dubois, & Dolan, 2019). As outlined previously, PFC neuromaturation is critical to efficient executive function, self-regulation, and emotion regulation (e.g., Beauchaine & Zisner, 2017; Palacios-Barrios & Hanson, 2019; Zelazo, 2015). Improvements in self-regulation across development occur in part through more efficient top-down modulation of subcortical structures by functional subdivisions of the PFC, and improved connectivity between subcortical structures and the PFC (see Casey, Oliveri, & Insel, 2014; Swartz, Carrasco, Wiggins, Thomason, & Monk, 2014). When exposed to emotion-eliciting events, typically developing children exhibit stronger subcortical responses but weaker and more diffuse frontal responses than adults (Macdonald, Goines, Novacek, & Walker, 2016). Normal PFC neuromaturation yields increasingly effective top-down regulation of these strong subcortical responses (e.g., Arnsten & Rubia, 2012).

Although specific neural structures differ across disorders, neurodevelopment of the PFC, and efficiency of subcortical–cortical

connections, become increasingly compromised for many adolescents with both internalizing and externalizing disorders (e.g., De Brito et al., 2009; Gold et al., 2016; Shaw et al., 2012). Adolescent boys with attention-deficit/hyperactivity and conduct disorder, for example, exhibit reduced anterior cingulate cortex volumes, which co-occur with disrupted connectivity between the anterior cingulate cortex and the striatum (Sauder, Beauchaine, Gatzke-Kopp, Shannon, & Aylward, 2009; Shannon et al., 2009). These findings and others that we have insufficient space to discuss suggest that both internalizing and externalizing progression across development derive at least in part from failures in neuromaturation of prefrontal regions implicated in executive function, self-regulation, and emotion regulation (for recent reviews, see Beauchaine, Zisner, & Hayden, 2019; Cubillo, Halari, Smith, Taylor, & Rubia, 2012). An ever-expanding body of research shows that frontal neuromaturation and cortical-subcortical connectivity are exquisitely sensitive to environmental insults including risk factors that accrue with poverty and heavy substance use (e.g., Hair, Hanson, Wolfe, & Pollak, 2015; Nguyen-Louie et al., 2018; Palacios-Barríos & Hanson, 2019; Pfefferbaum et al., 2018). Environmental effects on frontal neuromaturation begin in infancy (Hanson et al., 2013), which suggests a clear need for expanded prevention and early intervention programs.

Conclusion

We hope this editorial serves as a useful introduction to the Special Issue. We received exceedingly strong transdisciplinary contributions, both empirical and theoretical, from some of the field's top emotion researchers. We believe papers in the volume advance emotion dysregulation research, and will serve as a springboard for future studies. We are therefore eager to share these papers with the readership of *Development and Psychopathology*.

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