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What is This?

How Affective Science Can Inform Clinical Science: An Introduction to the Special Series on Emotions and Psychopathology

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Abstract

The construct of emotion dysregulation has been used to describe and explain diverse psychopathologies. Although this is intuitively appealing and sensible, the application of emotion reactivity and regulation to the study of psychopathology has, to a large extent, proceeded independently from concepts and measures informed by affective science. Utilizing the innovative research approaches, measures, paradigms, and insights that have emerged in the burgeoning field of affective science holds substantial promise for emotion dysregulation theories of psychopathology. In this introduction to the special series on emotions and psychopathology, we review many of these advances, and highlight several broad methodological and conceptual issues that researchers seeking to continue this crosscutting work should bear in mind. We close with a brief review of the six articles that constitute the special series, noting how each exemplifies the pioneering methodological and substantive advances that are typical of the best work in this new interdisciplinary field.

Most forms of psychopathology feature some type of emotional aberration (Gross & Barrett, 2013). Anxiety disorders, for example, are characterized by inappropriate or exaggerated fear responses. Depression is distinguished by sustained low levels of interest and joy (American Psychiatric Association, 2013). These empirically documented associations have led many clinical researchers to increasingly become interested in examining whether emotion dysregulation might serve as an explanatory construct for mental illness. In this view, specific psychopathologies are conceptualized as resulting from, or maintained by, deficits in the ability to appropriately and effectively regulate one's emotional experience.

Historically, theorizing about the relation between psychopathology and emotion dysregulation has proceeded independently of basic research in affective science. Yet, basic research on emotions and emotion regulation has brought to light important conceptual issues and innovative methodological approaches that are relevant to studying the kinds of dysregulation that occur in psychopathology.

Recently, however, scholars have begun to integrate these two streams of research. To give a few exciting examples, borderline personality disorder (BPD) is now viewed as problematic emotional reactivity and regulation (e.g., Glenn & Klonsky, 2009), and the most effective treatment for BPD, dialectical behavior therapy, succeeds largely to the extent that it successfully improves clients' abilities to recognize and regulate their intense emotions (Linehan et al., 2006; McMain et al., 2013). Similarly, the causes and functions of nonsuicidal

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self-injury (NSSI) remained poorly understood for decades, but this behavior pattern is now understood as a coping mechanism that efficiently (albeit temporarily) reduces overwhelming negative emotions and arousal, in individuals disposed to frequent and intense negative emotions (Klonsky, 2007, 2009; Victor & Klonsky, 2014). Furthermore, a treatment designed to increase acceptance and regulation of negative emotion has shown great promise for reducing NSSI (Gratz, Tull, & Levy, 2013). Partly in response to these and other successes, clinical researchers are now beginning to draw on the full range of concepts and methods from affective science to better understand the emotional processes that lie at the heart of a wide range of psychopathologies and to develop emotion-targeted interventions. The articles in this special series all provide examples of this new trend; the researchers represented here have moved past historical limitations to demonstrate the ways in which many of the methods and major findings from affective science can inform psychopathology theory and theory testing.

In this introduction to the special series, we provide a brief overview of conceptualizations of emotions within affective science, and of the historical separation between affective and clinical science. We then review the relatively recent methodological, theoretical, and substantive advances in affective science that have allowed for a newfound integration between these fields, and highlight several broad methodological and conceptual issues that researchers seeking to continue this important integrative work must bear in mind. Finally, we close with a brief review of the six articles that constitute the special series, explaining how each exemplifies the ways in which research in this area must proceed.

From Emotion to Emotion Regulation

Although there have been numerous disagreements over the precise definition of emotion, there is growing consensus that an emotion involves transient experiential, behavioral, and physiological responses to motivationally salient internal and external stimuli (Gross & Thompson, 2007). From an evolutionary perspective, emotions are "specialized modes of operation shaped by natural selection to adjust the physiological, psychological, and behavioral parameters of the organism in ways that increase its capacity and tendency to respond adaptively to the threats and opportunities characteristic of specific kinds of situations" (Nesse, 1990, p. 268). Emotions, then, are associated with physical and psychological changes that can be measured across multiple response systems, and understood in terms of their underlying motivational functions: Emotions compel us to act, and typically in a way that is useful or adaptive.

The multifaceted nature of emotion is highlighted if we imagine a person threatened by an assailant. The activation of an emotional response involves behavioral changes, which are evident as the individual freezes, fights, or flees. At the same time, physiological changes reflect the heightened attention, perceptual processing, and readiness for action that are characteristic of emotion. When asked, the individual might describe his or her subjective experience as terrifying, based on very real subjective *feelings* that are also an important part of the emotional experience.

In this example, the various components of the emotion work together to produce a highly adaptive response to the eliciting stimulus, and it is likely that this is how emotions typically work in nonhuman animals, who lack the ability to think about their emotions and intentionally regulate them. In humans, however, emotions are not entirely functional each time they are experienced, and they are also not always fully expressed. If we feel frightened by a horror movie, we do not run from the theater; along similar lines, most people inhibit the urge to fight when the person they are furious at is their boss. Although emotional responses are generally assumed to be relatively automatic, such inhibition suggests that emotional responses are not fully obligatory, and can be modulated. Emotions, then, might best be characterized in terms of *dispositions* toward action (Bradley & Lang, 2000; Lang, Bradley, & Cuthbert, 1997). Sitting still in the dark movie theater, our hearts race; engrossed in the film, we are primed to jump out of our seats—but we do not.

The reason for this gap between emotional experience and emotional behavior, in humans, is emotion regulation—a process that occurs so regularly it must be considered part of the emotion experience itself. Emotion regulation has been conceptualized in a number of ways; for example, Cole, Martin, and Dennis (2004) have noted that emotions influence, and therefore *regulate*, a number of cognitive functions (e.g., memory)—and that this impact of emotion on cognitive processes could be called emotion regulation, even though that is not the most typical conceptualization. The kind of emotion regulation that is most relevant to psychopathology, however, is that composed of the cognitive and behavioral processes that influence which emotions are experienced, as well as when and how they are experienced (Gross, 1998; Gross & Thompson, 2007). This conception of emotion regulation is consistent with Thompson's (1994) definition: "the extrinsic and intrinsic processes responsible for monitoring, evaluating, and modifying emotional reactions, especially their intensity and temporal features" (pp. 27–28). According to Gross and Jazaieri (2014), emotion regulation can be considered a fairly strategic process—it occurs when we "activate—either implicitly or explicitly—a goal to influence the emotion-generative process."

This is the form of regulation that is invoked when we choose to sit through horror films, or withhold an angry response in the presence of a superior. But how does it work?

Gross and his colleagues have conducted extensive research comparing the effectiveness and consequences of various emotion regulation strategies, and their model of emotion regulation has guided many recent studies on this topic. In their article in this special series, Gross and Jazaieri outline this model and explain how it applies to the processes involved in psychopathology. In addition, Joormann and Vanderlind (2014) adopt Gross's approach to explain how emotion regulation and regulatory failures can account for the cognitive biases and deficits in cognitive control that underlie depression. In both of these articles, the authors suggest that understanding the ways in which emotion regulation can go awry is essential to understanding psychopathologies that result from aberrant emotional experiences. In the remaining articles in the series, the focus is less explicitly on deficits in emotion regulation, per se, but rather on the ways in which emotional experiences, and the cognitive processes that elicit them, differ for those suffering from various psychopathologies. In all cases, these researchers suggest that by examining the emotional process at the root of a particular psychological disorder, treatments can be targeted to the source of the problem. Because emotions encompass feelings, thoughts, physiology, and behavior, changing one's emotion can have widespread downstream consequences relevant to the symptoms of a given disorder. As a result, our emphasis on emotions in psychopathology allows for not only more complex and nuanced theory building but also potential new interventions.

Historical Reasons for the Neglect of Emotions Within Psychopathology Research

For years, dysfunctions of emotion have figured implicitly in conceptions of psychopathology. More recently, however, many psychopathologies have been recast explicitly in terms of emotion and emotion regulation deficits (Berenbaum, Raghavan, Le, Vernon, & Gomez, 2003; Gross & Muñoz, 1995; Keenan, 2000; Kring & Bachorowski, 1999; Watson, 2003, 2005). In particular, elaborated theories of emotion dysregulation have been applied to depression, generalized anxiety disorder, BPD, and others. What accounts for this change? In our view, it can largely be understood by tracing—in broad strokes—the history of psychopathology research. Prior to the 1950s, clinical research and theory were largely centered on psychoanalysis, which focused on emotions, but in ways

that were scientifically untenable. Freudian theory made emotions—and most notably, anxiety—a critical player, responsible for triggering essentially all observed psychopathology, but Freud's account of emotion regulation via repression was largely unfalsifiable (Grunbaum, 1985). As behaviorism emerged as the strong counterpoint to psychoanalysis—eventually becoming the more prominent psychological subfield (see Tracy, Robins, & Gosling, 2003)—any research approach that involved the assessment of unobservable internal mental contents—such as repressed anxiety—was considered unscientific. In this context, internal experiences such as thought and emotion were not considered amenable to scientific study. This all changed in the 1970s, with the cognitive revolution, which allowed researchers to study thoughts as well as behavior. At the time, however, thoughts were considered to be largely separate entities from emotions (e.g., Zajonc, 1980). Moreover, cognitive psychologists found ways to test clearly articulated predictions—many of which were derived from observed behavior—giving scientific credence to their work and taking care to leave behind unobservable subjective feelings (which, at that time, were considered synonymous with emotion).

Only in the 1980s and 1990s were emotions understood as constructs that could be reliably measured, in ways that involve self-report but do not rely on it. Ekman and his colleagues' (Ekman & Friesen, 1971; Ekman, Sorenson, & Friesen, 1969) and Izard's (1971) work on the face—demonstrating that emotions can be assessed through concrete, observable nonverbal facial behaviors—was a large part of what led to this pendulum swing. Following Ekman's and Izard's work demonstrating universal facial expressions of emotion, others began to study emotions as potentially adaptive, biological phenomena, which could be assessed not only through self-report and facial expressions but also through physiology (e.g., Gross & Levenson, 1995). Emotions eventually came to be seen as functional rather than irrational targets of repression, and clinicians began to incorporate them in their models. However, this trend occurred recently enough that the merging of affective science and psychopathology research can still be considered to be in its infancy, underlining the importance of this special series.

Advances in Affective Science That Allow for a Fruitful Integration With Clinical Research

Affective science researchers comprise psychologists from all areas (clinical, cognitive, developmental, health, psychobiological, social, and personality) as well as many scientists outside of psychology (e.g., anthropologists,

biologists, economists, and neuroscientists). In the past few years, more articles have been published with the phrase "affective science" than in all prior years combined (Gross & Barrett, 2013). With this expansion have come new methods, research approaches, and substantive advances, all of which can inform psychopathology research. The time is thus ripe for clinical researchers to integrate emotions into their work. Here, we highlight several conceptual and methodological innovations that could substantially advance research and theory in this promising area.

Emotions are inherently biological

The understanding that emotions are generated in the brain, and are largely universal in humans and, in many cases, present in some form in most mammals, has allowed for major advances in emotion-based accounts of psychiatric disorders (e.g., Panksepp, 1998). All of the articles in this special series take for granted that emotions are manifested in the body as well as the mind, and all use knowledge of specific emotions to explain the various experiential, behavioral, and physiological symptoms seen in major psychopathologies. Some of these articles go even further; Panksepp, Wright, Dobrossy, Schlaepfer, and Coenen (2014), for example, provide an account of depression that relies on the understanding that deficits in three distinct brain systems entail dysfunction in three distinct emotions. In this view, the relevant emotional system and corresponding brain system are, essentially, one and the same. Others, such as Siegle and colleagues (2014) focus more on the utility of neurological indicators of shifts in cognitive-affective processes, but in all cases, there is an explicit acceptance that emotions are fully embodied experiences, orchestrated by distinct and overlapping neurological mechanisms. Although it may seem obvious that a phenomenon as multifaceted as an emotion is inherently biological, this view has become widely accepted only within recent decades. Contemporary affective scientists seek to understand the affective dysfunction that is characteristic of various psychopathologies in terms of core neural systems implicated in emotion, and how these systems interact with the environment. Situating psychological disease within an affective neuroscience framework allows for major advances in our understanding of the etiopathogenesis and treatment of psychopathology.

Emotion regulation is cognitive and biological

The emotional process that may be most relevant to psychopathology is emotion regulation. This is because emotion-based dysfunctions often result from dysregulation—a failure to appropriately regulate one's emotions, resulting in emotional experiences that are extreme or discordant with the situation. In addition, even though some psychopathological conditions are best conceptualized as stemming from extreme emotional reactivity or sensitivity, rather than deficient regulation, emotion regulation may nonetheless provide an important means of intervention. Most of the articles in this special series touch on regulation, and, as mentioned earlier, two explicitly focus on it. In recent years, our understanding of the cognitive processes involved in emotion regulation has increased dramatically, as has our understanding of how these processes are manifested biologically. These findings have several implications for psychopathology.

A growing body of research by Gross and his colleagues (e.g., Gross, 2002) has focused on expressive suppression as a form of response-focused emotion regulation, and reappraisal as a form of antecedent-focused emotion regulation. Expressive suppression refers to inhibiting the behavioral expression of an emotional response, whereas reappraisal involves consciously altering the meaning of an emotion-eliciting stimulus as it is perceived. Reappraisal is rooted in the work of Lazarus, who demonstrated that an emotional response is determined by the way a stimulus is interpreted (Lazarus, 1991).

Several studies indicate that reappraisal is effective in modulating physiological and neural responses to emotional stimuli, as well as self-reported feelings (Ochsner & Gross, 2005). For example, studies using functional neuroimaging found that reappraising unpleasant stimuli is associated with increased activation in areas of the lateral and medial prefrontal cortex, and also with decreased activation of the amygdala (Ochsner, Bunge, Gross, & Gabrieli, 2002). In related work, reducing the intensity of negative emotions through reappraisal led to increased activity in similar areas of the prefrontal cortex and decreased activity in limbic regions (Phan et al., 2005; see also Beauregard, Lévesque, & Bourgouin, 2001; Harenski & Hamann, 2006; Kalisch et al., 2005; Lévesque et al., 2003; Ohira et al., 2006).

Using multiple reappraisal strategies, another set of studies demonstrated that some regions in the prefrontal cortex were activated during both the down- and up-regulation of emotional response to unpleasant stimuli (Ochsner et al., 2004). Another lab reported consistent findings: Many areas of the prefrontal cortex were active in both up-regulation and down-regulation—though some regions demonstrated specificity to the direction of emotion regulation (Eippert et al., 2007). In both sets of studies, amygdala activation was, in contrast, specifically increased or decreased by instructions to up-regulate or down-regulate, respectively. Furthermore, trial-by-trial

ratings of emotion regulation *success* were correlated with increased amygdala activity during up-regulation, and with increased prefrontal cortex activity during down-regulation (Eippert et al., 2007).

In terms of the functional relationship between the amygdala and prefrontal cortex, studies using a within-subjects approach showed that participants who decreased amygdala activity during reappraisal also increased activity in areas of the prefrontal cortex (Urry et al., 2006). Other studies have similarly demonstrated increased coupling between prefrontal cortex and amygdala during emotion regulation (Banks, Eddy, Angstadt, Nathan, & Phan, 2007; see also Lee, Heller, Van Reekum, Nelson, & Davidson, 2012). Furthermore, this circuitry is linked to functioning of the hypothalamic-pituitary-adrenal axis: The coupling of frontal and limbic regions during emotion regulation predicts more normative changes in cortisol over the course of the day (Urry et al., 2006). Overall, these results suggest that areas of the prefrontal cortex interact with the amygdala and other neural structures that respond to emotionally salient stimuli during reappraisal and, important for clinical research, individual differences in prefrontal-amygdala coupling may underlie individual differences in emotion regulation.

Reappraisal studies have highlighted the way appraisal and explicit meaning changes can alter emotional responses; however, even more subtle manipulations during emotional processing seem to have similar effects. For instance, the ways in which emotional stimuli are evaluated and attended to influence a variety of indices of emotional processing (Ochsner & Gross, 2005; MacNamara, Ochsner, & Hajcak, 2011). For example, if emotional stimuli are described in more neutral terms before they are viewed (i.e., preappraisal), they elicit reduced self-reported arousal and neural activity (Foti & Hajcak, 2008; MacNamara, Foti, & Hajcak, 2009). Moreover, simply making nonemotional compared with emotional judgments of unpleasant stimuli results in reduced amygdala activity and increased prefrontal activation (Hariri, Mattay, Tessitore, Fera, & Weinberger, 2003; Keightley et al., 2003; Mathews, Yiend, & Lawrence, 2004). Along similar lines, explicitly directing attention to less arousing aspects of emotional stimuli reduces electrocortical measures of emotional processing (Hajcak, MacNamara, Foti, Ferri, & Keil, 2013), decreases amygdala response, increases fronto-parietal activation, and decreases the intensity of subjective experience (Ferri, Schmidt, Hajcak, & Canli, 2013). Collectively, these findings suggest not only that emotion regulates attention, but also that attention and appraisal are powerful means for altering emotional experience.

Structure and measurement of emotional response and regulation

The understanding that emotions are multifaceted has paved the way for diverse methodological innovations, geared toward measuring each facet of emotional experience and emotion regulation in complex ways that go beyond self-report. Psychopathology researchers who are already interested in moving beyond self-report can use these approaches—which build on innovations in cognition, neuroscience, behavior, and biology—to directly target the emotional processes at play in the disorders they seek to understand.

For example, emotion research has been substantially facilitated by the development of standardized emotion-eliciting stimuli, such as the International Affective Picture System (IAPS; Lang, Bradley, & Cuthbert, 1999). The IAPS contains a range of emotional content that produce changes in self-reported, behavioral, psychophysiological, and neural measures. In early studies using this stimulus set, participants were asked to rate each image on a Likert-type scale that ranged from pleasant to unpleasant, and on a separate scale that indicated a strong to weak emotional response (Lang, 1980). Pictures rated as very pleasant or very unpleasant were also rated as eliciting an intense emotional response (Lang et al., 1999). This finding suggests that both increases and decreases in valence ratings were accompanied by increased arousal. In contrast, pictures rated low in arousal tended to be neutral with respect to valence. In terms of the generality of these findings, similar patterns been reported for both word- and sound-based stimuli (Bradley & Lang, 1991).

This pattern of self-report data is consistent with the existence of two fundamental motivational systems that support emotional responses: Pleasant ratings reflect *appetitive* activation, whereas unpleasant ratings reflect *defensive* activation (Bradley, Codispoti, Cuthbert, & Lang, 2001; Bradley & Lang, 2000; Lang et al., 1997).[1] In this view, the intensity of appetitive or defensive activation elicited by a stimulus is reflected in ratings of arousal. This differentiation (i.e., pleasant/unpleasant versus weak/strong emotional response) has important consequences for studies in emotion and psychopathology: *An emotional response* might reflect the valence of the stimulus, the degree to which it is emotionally arousing, or both. Similarly then, emotion dysfunction might reflect abnormalities related to arousal, valence, or both higher-order dimensions of emotion. These findings mean that differentiating valence from arousal is important in terms of specifying the nature of emotion and its dysfunction.

Several behavioral and psychophysiological measures appear to uniquely index the valence dimension of emotional response. A furrowed brow is linked to facial

displays of several negative emotions (Ekman, 1993; Ekman et al., 1969; Ekman et al., 1987), and it can be quantified by measuring the amount of EMG activity in the corrugator muscle; furthermore, corrugator EMG activity during picture viewing correlates negatively with valence ratings (Bradley and Lang, 2000; Cuthbert, Bradley, & Lang, 1996; Lang, Bradley, & Cuthbert, 1998b; Lang et al., 1997). Conversely, activity of the zygomatic muscle is involved in smiling, and EMG activity in this muscle correlates positively with ratings of valence (Bradley & Lang, 2000; Bradley et al., 2001; Lang et al., 1997; Lang et al., 1998b; Lang, Greenwald, Bradley, & Hamm, 1993). It is worth noting that facial EMG activity is sensitive to the perceived valence of stimuli even in the absence of overtly observable facial movements (Bradley & Lang, 2000).

The human startle eyeblink reflex has also been linked to particular aspects of emotional processing (Lang, Davis, & Öhman, 2000; Vrana, Spence, & Lang, 1988). The startle response is a reflex in which the body contracts into a defensive posture, and is typically measured in humans by recording EMG from the obicularis oculi (i.e., blink magnitude) in response to a sudden and loud acoustic probe. The magnitude of the startle eyeblink response is larger when participants view threatening stimuli, indicating that defensive reflexes are primed by aversive stimuli (Bradley, Codispoti, & Lang, 2006; Bradley, Moulder, & Lang, 2005; Lang et al., 2000).[2]

Unlike startle, reflexes that are not inherently defensive appear to be potentiated by both appetitive and aversive emotional stimuli. Two studies found that spinal reflexes were enhanced while participants viewed both pleasant and unpleasant stimuli (Bonnet, Bradley, Lang, & Requin, 1995; Both, Everaerd, & Laan, 2003). For instance, in one of these, researchers recorded the EMG in the soleus muscle of the lower leg following a hammer tap at the heel tendon, and found that this response was larger when participants viewed appetitive or aversive, compared with neutral images (Both et al., 2003). Similarly, the EMG response elicited by transcranial magnetic stimulation over the motor cortex was amplified when participants viewed both pleasant and unpleasant, compared with neutral, IAPS images (Hajcak et al., 2007). Collectively, these data suggest that both pleasant and unpleasant emotional stimuli prime or facilitate action, consistent with the view that emotional processing mobilizes the body for action (Fridja, 1986; Lang, 1994).

Like the general (i.e., nondefensive) reflexes described earlier, several other behavioral and psychophysiological measures appear sensitive to the arousal dimension of emotion—and insensitive to valence. These measures appear to index the *degree* to which motivational systems are activated, regardless of *which* system has been activated. For example, both pleasant and unpleasant stimuli elicit larger skin conductance responses compared with neutral images, and the magnitude of the skin conductance response relates to arousal ratings (Bradley & Lang, 2000; Bradley et al., 2001; Cuthbert et al., 1996; Lang et al., 1993; Lang et al., 1997; Lang et al., 1998b; Winton, Putnam, & Krauss, 1984). Pupil diameter is also increased for emotional (i.e., both unpleasant and pleasant) compared to neutral stimuli, and pupillary and skin conductance changes follow similar patterns across emotional stimuli (Bradley, Miccoli, Escrig, & Lang, 2008).

Turning to behavioral indices, viewing time is positively related to arousal, such that both highly pleasant and unpleasant images are viewed the longest by participants (Bradley & Lang, 2000; Bradley et al., 2001; Lang, Bradley, & Cuthbert, 1998a; Lang et al., 1993; Lang et al., 1997, 1998b). Consistent with these findings, studies using eye tracking have found that individuals are more likely to look at emotional compared with neutral images, in terms of both their initial and subsequent fixations (Calvo & Lang, 2004; Nummenmaa, Hyönä, & Calvo, 2006)—even when they are instructed to look only at neutral images (Nummenmaa et al., 2006).

Another indicator of arousal is the late positive potential (LPP)—an electrocortical response that is larger following the presentation of emotional compared with neutral images. The LPP is a positive deflection in the stimulus-elicited event-related potential (ERP) that is maximal at central-parietal midline recording sites. Compared with neutral images, both pleasant and unpleasant images elicit a more positive LPP—a difference that begins approximately 200 ms after stimulus presentation (Cacioppo, Crites, Gardner, & Berntson, 1994; Cuthbert, Schupp, Bradley, Birbaumer, & Lang, 2000; Keil et al., 2002; Lang et al., 1997; Schupp et al., 2000; Schupp, Markus, Weike, & Hamm, 2003). Functionally, the LPP is thought to reflect sustained engagement with emotional content (Proudfit, Dunning, Foti, & Weinberg, 2013).

In many ways, the LPP mirrors data obtained using functional neuroimaging techniques; using both positron emission tomography (PET) and functional magnetic resonance imaging (fMRI), studies have demonstrated emotional stimuli to activate the visual and extrastriate cortex to a greater degree than neutral stimuli (Bradley et al., 2003; Breiter et al., 1996; Lane et al., 1997; Sabatinelli, Flaisch, Bradley, Fitzsimmons, & Lang, 2004). In fact, the increased perceptual processing of emotional stimuli likely depends on projections from the amygdala to visual cortex (Bradley et al., 2003; Lang et al., 1998a; Morris et al., 1998; Sabatinelli, Bradley, Fitzsimmons, & Lang, 2005). A recent study that combined ERP and fMRI methods indicated that the increased LPP elicited by emotional stimuli corresponded to

increased blood flow in occipital, parietal, and infero-temporal regions in the brain (Sabatinelli, Lang, Keil, & Bradley, 2007). Overall, then, both pleasant and unpleasant stimuli capture attention and receive increased perceptual processing resources, which can be measured using both fMRI and ERPs.

Broadly, these studies indicate that many dependent measures of emotional response are differentially sensitive to the valence and arousal dimensions of emotion. However, it is also important to note that correlations between, and coherence among, these various measures of emotion can be weak (Davidson, 1998; Lang, 1968). This finding, that measures of emotional reactivity are not redundant across response modalities, highlights the importance of multimodal assessment. Such an approach may be necessary for fully characterizing emotional deficits in psychopathology, that is, to determine whether abnormalities of emotional response reflect the valence, arousal, or both dimensions of emotional response—and to quantify emotion across multiple response modalities.

Finally, we turn to the self-report method of assessing emotion. Many, if not most, studies of emotion rely solely on self-report measures to quantify emotional response. Indeed, emotion dysregulation theories of psychopathology were based on such data. Often, self-report is treated as the criterion measure against which other indices are compared—the standard used to validate other assessments. Although self-report is an important method in emotion research, it has a number of methodological shortcomings. Perhaps most important is the implicit assumption that emotions are accessible to consciousness, in other words, that *emotion is equivalent to feeling* (Davidson, 2003). Although feelings have a role in adaptive functioning (Bechara, Damasio, & Damasio, 2000; Damasio, Everitt, & Bishop, 1996), there is growing evidence that the generation of emotion takes place under the radar of consciousness (Berridge, 2003; Monahan, Murphy, & Zajonc, 2000; Öhman, 2005). Thus, fully characterizing emotional responses requires complementing self-report measures with more objective indices of emotion that do not rely on introspection.

Many of the measures reviewed earlier appear promising for studying the role of emotion regulation and dysregulation, in particular in psychopathology. Jackson, Malmstadt, Larson, and Davidson (2000) measured the modulation of the startle response and corrugator activity by unpleasant and neutral images, under conditions in which participants were instructed to increase or decrease their emotional response. They found that both the magnitude of the startle response and corrugator EMG activity were sensitive to emotion regulation instructions (see also Dillon & LaBar, 2005; Eippert et al., 2007; Lissek et al., 2007). Corrugator facial EMG is also sensitive to emotion regulation (Ray, McRae, Ochsner, & Gross, 2010; Wu, Winkler, Andreatta, Hajcak, & Pauli, 2012), as is the skin conductance response to emotional stimuli (Driscoll, Tranel, & Anderson, 2009; Eippert et al., 2007; Hariri et al., 2003).

Amplitude of the LPP appears similarly sensitive to emotion regulation instructions. In an initial study of the effect of regulation on LPP responsivity, results revealed an LPP reduction when participants intentionally decreased the intensity of their emotional response to unpleasant images (Moser, Hajcak, Bukay, & Simons, 2006). The LPP is also reliably reduced following reappraisal instructions (Hajcak & Nieuwenhuis, 2006). In both of these studies, LPP modulation related to emotion regulation began just 200 ms after stimulus onset, and the difference was maintained throughout most of the stimulus presentation. In another study, the observed LPP elicited by emotional pictures was smaller when participants made nonemotional compared with emotional decisions about pictures, suggesting that the way emotional content is appraised also affects the LPP (Hajcak, Moser, & Simons, 2006). Finally, attentional deployment—directing attention to less arousing aspects of emotional stimuli—can reduce the amplitude of the LPP (Dunning & Hajcak, 2009; Hajcak, Dunning, & Foti, 2009; Hajcak et al., 2013). The LPP may be ideally suited for studying the modulation of early neural responses to emotional stimuli, in the context of both relatively automatic and conscious emotion regulation strategies.

It thus appears that nearly all physiological and neurological measures of emotional reactivity—functional activity in the amygdala and visual cortex, facial EMG, startle, the LPP, and skin conductance—are sensitive to emotion regulation instructions. As a result, clinical researchers who wish to examine the role of emotion dysregulation in a particular disorder, or the effectiveness of interventions that involve training patients to engage in certain regulatory strategies, might consider adopting these measures as a way of avoiding reliance on self-report. Given weak coherence across measures, multiple measures of emotion regulation might be preferable.

Broad Conceptual and Methodological Issues

Historically, emotion dysregulation theories of psychopathology have suffered from a number of conceptual and methodological limitations. Newer work in this area has made major steps to overcome these issues, and the six articles in this special series are examples of this trend. Nonetheless, as research in this field moves forward, it is essential that researchers bear in mind these potential pitfalls, so as to avoid repeating the mistakes of the past.

As reviewed earlier, emotion can be measured across multiple response channels. Moreover, specific measures of emotion can differ from one another in crucial ways. For instance, arousal ratings (Codispoti, Ferrari, & Bradley, 2006), corrugator muscle measured via facial EMG (Bradley, Lang, & Cuthbert, 1993), skin conductance (Codispoti et al., 2006), and amygdala response (Breiter et al., 1996) all habituate to the repeated presentation of stimuli; however, affective modulation of startle (Bradley et al., 1993) and the LPP (Codispoti et al., 2006) do not. Moreover, each measure of emotion will have its own unique psychometric properties—and its ability to index trait-like differences will be limited by measures of reliability. To take one example, amygdala response to emotional faces shows relatively low test-retest reliability (Sauder, Hajcak, Angstadt, & Phan, 2013); contrast that with moderate reliability of the LPP over 2 years (Kujawa, Klein, & Proudfit, 2013; see also Moran, Jendrusina, & Moser, 2013). Very few studies have evaluated psychometric properties of physiological measures in the context of emotion regulation—although there is already evidence for clear differences in psychometric properties between dependent measures (cf. Hibbert, Weinberg, & Klonsky, 2012; Lee, Shackman, Jackson, & Davidson, 2009; Moran et al., 2013). Thus, it will be crucial for future work to carefully assess multiple measures of emotion regulation in light of these issues.

A second conceptual issue is an absence, in many theories, of clear distinctions between specific aspects of emotion. Given mounting data, reviewed earlier, suggesting that emotional experiences (subjective, physiological, and behavioral) can be characterized by the higher-order constructs of valence and arousal, it would be possible for a given disorder to be characterized by hyperactive defensive reactivity (a valence-related abnormality), or hyperreactivity of both defensive and appetitive systems (an abnormality of arousal that cuts across the valence dimension of emotion). For example, individuals who self-injure appear to be characterized by elevated negative emotionality (i.e., defensive reactivity) but normative positive emotionality (i.e., appetitive reactivity; Klonsky, Oltmanns, & Turkheimer, 2003; Victor & Klonsky, 2014), and the behavior appears to be reinforced by the subsequent reduction in negative emotion rather than increases in positive emotion (Klonsky, 2009).

Likewise, as more and more mental disorders are discussed in terms of emotion regulation abnormalities, it is increasingly important for these accounts to differentiate themselves. For example, emotion dysregulation has been conceptualized as the core feature of depression, generalized anxiety disorder, and BPD. Yet, these three disorders are associated with different presentations and interventions. To be useful clinically or theoretically, emotion regulation theories of these disorders will need

to account for and specify the distinct patterns of emotional dysfunction apparent in these disorders. Current theories appear to insufficiently differentiate among potential sources of emotional difficulties (but see Gross & Jazaieri, 2014).

Third, theories of psychopathology have not adequately assessed the time course of emotion. Rothbart and Derryberry (1981) delineated several characteristics of emotional reactivity, which were later described as metrics for quantifying *affective chronometry* (Davidson, 1998). For instance, some emotional responses might reach their maximum very rapidly, whereas other responses may take a longer time to develop; Davidson (1998) refers to this metric as *rise time to peak*. Similarly, the time it takes to return from maximum response to baseline, *recovery time*, is another metric that can be used to characterize the time course of emotional reactivity. Davidson also highlights *duration of response* as an additional aspect of affective chronometry—that is, the amount of time that responding stays above some threshold.

Consider the hypothetical emotional responses depicted in Figure 1; Responses A through E could represent averages from several individuals or groups of individuals or even within-individual responses across types of stimuli or measures. If peak or average measurement relative to baseline is employed, nearly all of these responses would be scored identically; in fact, only E reflects a smaller baseline-to-peak response. In terms of Davidson's metrics of affective chronometry, C and D have a faster rise time to peak, and thus reach their maximum response earlier and have a steeper increase in response amplitude. In addition, C takes longer than D to return to baseline and could be described in terms of prolonged recovery time; it would also be fair to say that the duration of response depicted by C exceeds that of D. On the other hand, A is identical to B except for a difference in baseline. These meaningful differences can be captured only by assessing the time course of emotional response.

In addition to these metrics of affective chronometry, the intensity of a stimulus required to elicit an emotional response may vary in meaningful ways—thus, *threshold* for response is another important variable in emotional reactivity (Davidson, 1998). Specifically, the relationship between the emotional intensity of a stimulus and response magnitude may vary across individuals: Maximum response may be reached by lower intensity stimuli for some, whereas the response magnitude for others might follow a more linear dose-response curve. Moving forward, emotion dysregulation theories of psychopathology should seek to distinguish the specific temporal processes that are supposedly abnormal (for example, see Heller et al., 2009).
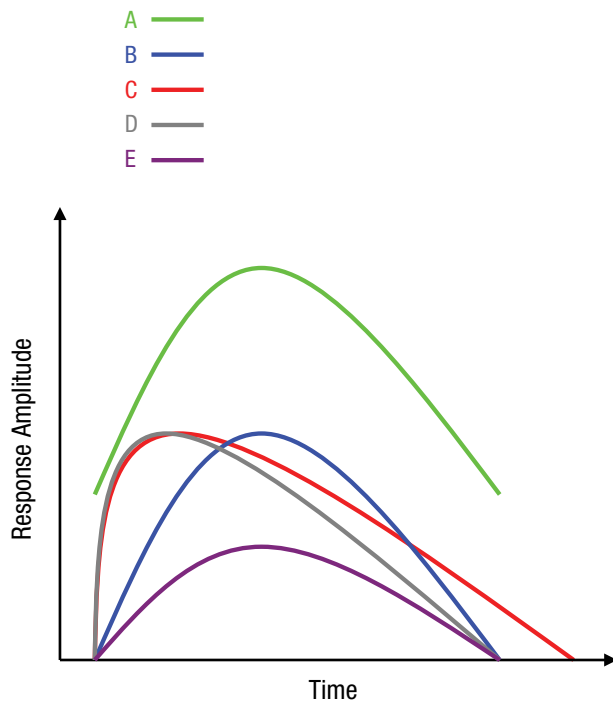
Figure 1. Five hypothetical emotional responses. Affective chronometry refers to the time course of emotional response; A to E represent five hypothetical emotional responses that vary in baseline, peak response, rise time to peak, and duration.

A fourth conceptual issue is, by this point, probably obvious given our emphasis on the role of regulatory processes and dysregulation. Previously, theoretical accounts often failed to make clear conceptual distinctions between emotional reactivity and regulation. Indeed, it was relatively common for researchers to make conclusions about emotion regulation without assessing regulatory processes or distinguishing them from reactivity (see Cole et al., 2004). For instance, a positive relationship between scores on the Affect Control Scale and BPD symptoms was interpreted to support an emotion dysregulation theory of BPD (Yen, Zlotnick, & Costello, 2002). However, the Affect Control Scale was developed to assess fear of emotions, which—although related to emotion regulation—is not equivalent to regulation. Furthermore, although the presence of affective psychopathology suggests emotion dysfunction, this does not *necessarily* imply regulatory deficits. Too much sadness, fear, or anger *could* result from a lower threshold for reacting to emotional stimuli, or choosing not to regulate these emotions.

It is important to note that the conceptual difficulty in distinguishing between emotional reactivity and regulation applies to all measures of emotional response. In Figure 1, suppose response B and E reflect activation of the amygdala from two individuals. Individual B, then,

has increased peak activation in the amygdala compared with Individual E, although they have comparable rise times, duration, and return to baseline. This could reflect the fact that Individual B is *more reactive* than E; however, one could also argue that Individual E is simply engaging in *more regulation* than B (Rothbart & Derryberry, 1981). Conceptually, increased reactivity can always be explained in terms of decreased regulation, and vice versa. Our practical suggestion is that reactivity and regulation can be teased apart, empirically, through experimental manipulation—that is, by way of altering task demands. For instance, the influence of attentional focus can be manipulated by directing attention to particular aspects of emotional stimuli, or by asking participants to make emotional versus nonemotional decisions about emotional stimuli (Ferri et al., 2013; Hajcak et al., 2006; Hariri et al., 2003; Keightley et al., 2003). This approach has proved effective in shedding light on neural correlates of emotion regulation, and can now be used to examine individual differences in the efficacy of particular emotion regulation strategies.

In addition to differentiating emotion regulation from other emotion variables, it is also necessary to identify distinct classes of emotion regulation difficulties. For example, emotion regulation can range from an intentional and controlled process on the one hand to a completely automatic process on the other hand (Gross et al., 2007). Although the controlled versus automatic distinction may be more of a continuum than a dichotomy, many theories of psychopathology that have posited core emotion regulation deficits have not specified whether these deficits reflect failures of controlled or relatively automatic emotion regulation strategies. Furthermore, for a given psychopathology, it is important to assess emotion regulation abilities across a number of strategies; a disorder could be characterized by a normal ability to reappraise but abnormal ability to utilize directed attention, for example. Thus, assessing more fine-grained conceptualizations of emotion regulation—as many researchers are beginning to do (see Gross & Jazaieri, 2014; Joormann & Vanderlind, 2014; Siegle et al., 2014)—will shed important light on the specific deficits, or patterns of deficits, that characterize a given disorder.

Along these lines, it is also important to distinguish emotion regulation deficits from regulation that successfully modulates emotion in the short term but leads to maladaptive outcomes in the long-term. Campbell-Sills and Barlow (2007) suggest that "individuals with anxiety and mood disorders may make counterproductive attempts to regulate acute affective episodes that lead to the exacerbation and persistence of unwanted emotion" (p. 543). For instance, patients with posttraumatic stress disorder may purposely avoid trauma cues and reminders; within Gross's

model, these individuals are utilizing situation selection to reduce unpleasant emotional experience. This type of avoidance does not characterize an emotion regulation *deficit*; rather, patients who avoid reminders are *successfully* regulating their problematic emotions. Ironically, though, their successful regulation appears to maintain anxiety disorders, suggesting that effective emotion regulation can have deleterious effects under some conditions. An individual who always avoids reminders of a trauma can never learn that the reminder is not dangerous and may continue to avoid and fear such reminders throughout his or her life (Foa & Kozak, 1986).

Using Affective Science to Inform Psychopathology: The Special Series

In summary, emotional dysfunction and dysregulation, as an explanatory construct, holds great promise for understanding mechanisms that put individuals at risk for, and contribute to the maintenance of, a number of major psychopathologies. To realize their full potential, emotion-based theories of psychopathology should bear in mind several critical aims: (a) specify whether deficits are specific to pleasant or unpleasant stimuli, and consider the distinction between valence and arousal; (b) employ multiple measures of emotional reactivity; (c) focus on measures capable of shedding light on affective chronometry and the time course of emotional response, especially moving beyond peak response measures to assess rise time, return to baseline, and threshold; (d) assess multiple types of emotion regulation, especially with existing paradigms and frameworks of emotion regulation; (e) seek to distinguish reactivity from regulation (see, for example, Gross, Sheppes, & Urry, 2011); and (f) attempt to elucidate disorder-specific patterns of emotion dysregulation. The articles in this special series are all examples of research programs and findings that take these suggestions to heart.

These articles fall into two categories. First, several articles examine how emotional processes or failures in emotion processing contribute to the etiology of a disorder or disorders. Second, several articles take the next essential step in this line of work, examining how intervention research can target the specific emotional processes or failures at play. Within the first category, articles examine psychopathology at both the broadest possible level, covering a wide range of disorders (Gross & Jazaieri; Watson & Naragon-Gainey, 2014), and at a narrower level, tackling the emotional processes that go awry within one particular disorder (Joormann & Vanderlind, major depressive disorder; Kring, Siegel, & Barrett, 2014, schizophrenia). All of these articles follow our recommendation to make clear distinctions between emotion experience (including the dispositional

tendency to experience emotions, as is relevant to the personality traits examined by Watson & Naragon-Gainey), emotional reactivity, and regulation.

Beginning the series is Gross and Jazaieri's broad review of how emotion regulation failures are central to a number of major psychiatric disorders. These authors emphasize the importance of distinguishing between specific emotional processes and changing our focus from "emotion dysregulation" broadly construed, toward examining how different disorders involve different kinds of dysregulation. As a result, the model they articulate provides a strong overarching framework for how psychopathology researchers might most effectively integrate emotion regulation research into their work.

Building directly on this work is Joormann and Vanderlind's article discussing how emotion regulation research can inform theory and research on depression. These authors focus on the cognitive factors that increase depression, specifically examining how many of these processes can be understood as regulation failures. They also pinpoint how these failures directly promote the dysregulated emotional experiences seen among those suffering from depression.

In a somewhat different vein, Watson and Naragon-Gainey report results from a large-scale study of the associations between specific personality traits and a wide range of affective disorders—which they define as psychological disturbances that are particularly emotional in nature. This work takes important steps toward connecting personality trait research with psychopathology research by way of emotions and emotion dysregulation. Personality researchers have long been interested in psychopathology, but this study exemplifies the new importance placed on the role of emotion in these associations. Watson and Naragon-Gainey also go beyond self-report—using clinical interviews as an assessment tool—to compellingly demonstrate how the major symptoms of many disorders and the major characteristics of at least two of the Big Five personality traits (Neuroticism and Extraversion) can be understood in emotional terms.

The final contribution targeting etiology is from Kring et al., who report the results of an experiment testing whether early emotional processing (i.e., unconscious processing) is intact in schizophrenics. Given prior evidence that schizophrenics suffer certain emotional deficits, including an absence of anticipatory pleasure, the goal of this research was to test whether these deficits could be explained by failures in early unconscious emotional processing. Results suggest, however, that early processing is intact in these individuals, indicating that it is the subsequent semantic understanding of emotions that is likely to be the source of problems in schizophrenia. This research demonstrates how the examination of a specific emotional process can be informative about the

basic etiology of a major disorder. It also exemplifies the importance of moving beyond self-report to address these issues, as these findings—which emerged from a continuous flash suppression paradigm—would not have been attainable from a study that more simply asked participants to report on their feelings or their explicit perceptions of emotion stimuli.

The final two articles in the series move beyond the emotion-based causes of disorders to begin to examine treatments that directly target these emotional processes. Siegle and colleagues test the effectiveness of cognitive control exercises designed to reduce rumination and thereby improve outcomes for patients suffering from depression. Their novel approach emerges directly from an understanding that treatments targeting the emotional processes known to influence disorders are likely to be most effective. Furthermore, they found the greatest improvement (reduced rumination) among patients who showed physiological indicators of task engagement during the exercises. This finding has important implications, as it highlights the need to assess emotional responses through physiological measures that are impervious to potential participant biases in self-reporting task engagement. It also allows for inferences regarding precisely which aspect of the treatment was responsible for its positive effects.

Finally, our series closes with a contribution from Panksepp et al., who discuss the specific importance of three emotional brain systems shared across species—which Panksepp has previously labeled the PLAY, PANIC, and SEEKING systems (e.g., 1998)—in conceptualizing and treating depression. In addition to describing Panksepp and colleagues' large body of work demonstrating what these brain circuits do and how they work, these authors discuss the novel development of neurochemical and direct brain stimulation interventions that seek to treat depression by targeting these specific brain regions and their functionality.

Collectively, these six articles point to exciting new directions in research at the intersection of emotions and psychopathology. Each demonstrates how this research should be done and exemplifies the benefits of this kind of integrative work, in terms of major advances in our understanding of a range of psychiatric disorders. It is our hope that future researchers will use these articles as a road map for how work in this growing area might most effectively move forward.

Author Contributions

All three authors contributed equally to the ideas and review in this article.

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The authors declared that they had no conflicts of interest with respect to their authorship or the publication of this article.

Notes

1. We have highlighted the two-factor approach advocated by Lang and colleagues, mainly because their group has systematically measured responses across multiple modalities and utilized standardized stimuli to do so. It is important to note, however, that this type of two-factor approach to emotion is consistent with a number of other models of emotion and emotional experience. For instance, Watson and colleagues have emphasized the distinction between positive and negative activation (Watson, Wiese, Vaidya, & Tellegen, 1999), and drawn parallels between their account of emotion and the broader temperamental dimensions of extraversion and neuroticism, respectively (Clark, Watson, & Mineka, 1994). In addition, Gray's theory relies on the distinction between the behavioral inhibition and activation systems (Gray, 1994; Gray & McNaughton, 2003). Davidson and colleagues have described neural systems supporting individual differences in approach- and withdrawal-related emotion, especially differences in frontal asymmetry (Davidson, 2002, 2003; Davidson, Abercrombie, Nitschke, & Putnam, 1999; Davidson & Irwin, 1999; Davidson, Pizzagalli, Nitschke, & Putnam, 2002).

2. When the interstimulus interval between picture onset and the startle probe is relatively short (e.g., less than approximately 1,000 ms), the startle response is actually inhibited. These data have been taken to indicate that early attention to emotional stimuli inhibits the startle response and that the subsequent facilitation of startle by defensive activation takes some time to develop (Bradley, Codispoti, & Lang, 2006). In addition, the startle reflex is facilitated by cues that signal subsequent impending pleasant and unpleasant stimuli—suggesting that arousal modulates startle during anticipation of emotional stimuli (Sabatinelli, Bradley, & Lang, 2001).

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