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# Emotion, Emotion Regulation, and Psychopathology: An Affective Science Perspective

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## Abstract

Many psychiatric disorders are widely thought to involve problematic patterns of emotional reactivity and emotion regulation. Unfortunately, it has proven far easier to assert the centrality of “emotion dysregulation” than to rigorously document the ways in which individuals with various forms of psychopathology differ from healthy individuals in their patterns of emotional reactivity and emotion regulation. In the first section of this article, we define *emotion* and *emotion regulation*. In the second and third sections, we present a simple framework for examining emotion and emotion regulation in psychopathology. In the fourth section, we conclude by highlighting important challenges and opportunities in assessing and treating disorders that involve problematic patterns of emotion and emotion regulation.

## Keywords

emotion, emotion regulation, emotion dysregulation, psychopathology

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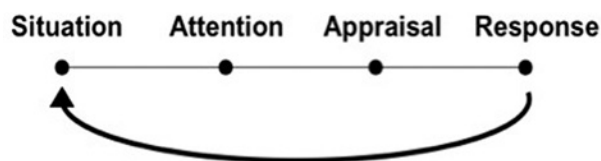
Many psychiatric disorders are said to be characterized by problems with emotion and emotion regulation (estimates range from 40% to more than 75%; see Berenbaum, Raghavan, Le, Vernon, & Gomez, 2003; Gross & Muñoz, 1995; Jazaieri, Urry, & Gross, 2013; Kring, 2008, 2010; Kring & Werner, 2004; Werner & Gross, 2010). This perspective is evident in special journal issues (e.g., *Emotion*, *Emotion Review*, *Cognition & Emotion*, *Journal of Experimental Psychopathology*, *Journal of Abnormal Psychology*, *Motivation and Emotion*, *Journal of Happiness Studies*, *Journal of Child Psychology and Psychiatry*, *Developmental Neuropsychology*, *Journal of Psychopathology and Behavioral Assessment*, and *Nature Neuroscience*, as well as this issue of *Clinical Psychological Science*) and books (e.g., Gross, 2014; Kring & Sloan, 2009) that have focused on the links between emotion and emotion regulation on one hand and psychopathology on the other hand. This view is so widespread that it seems incontrovertible—of course psychopathology involves problems with emotion and emotion regulation.

The closer one looks, however, the murkier the picture becomes. One reason for this is the heterogeneity of emotion-related processes, as well as the considerable

divergence in definitions of *emotion* and *emotion regulation* (Gross & Barrett, 2011). Another difficulty is the tremendous heterogeneity within psychiatric disorders. For example, according to the latest edition of the *Diagnostic and Statistical Manual of Mental Disorders* (5th ed., *DSM-5*; American Psychiatric Association, 2013), two individuals who meet criteria for the same disorder could share only one symptom (e.g., major depressive disorder requires only five out of nine criteria), or in the case of some personality disorders, no symptom overlap at all may exist (e.g., obsessive-compulsive personality disorder, in which meeting four out of eight criteria is required; or antisocial personality disorder, in which meeting three out of seven criteria is required); thus, “convergence on a core set of relevant mechanisms is difficult” (Dillon, Deveney, & Pizzagalli, 2011, p. 75). Although revisions in the *DSM-5* attempt to rectify this problem, clinical heterogeneity still stands as a major challenge for the field.

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**Fig. 1.** The modal model of emotion. From “Emotion Regulation: Conceptual Foundations,” by J. J. Gross and R. A. Thompson, 2007, in *Handbook of Emotion Regulation* (p. 10), J. J. Gross (Ed.), New York, NY, Guilford Press. Copyright by Guilford Press. Adapted with permission.

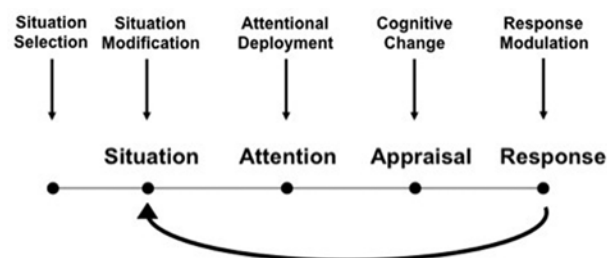
In addition, uncertainty prevails regarding the causal role of emotion and emotion-regulation difficulties. Searches for biomarkers have proven challenging because “similar phenomena (e.g., increased heart rate) are characteristic of multiple emotions” (Dillon et al., 2011, p. 75). These considerations suggest that it may be difficult to precisely specify the problems with emotion and emotion regulation that characterize any given psychiatric disorder.

Despite these challenges, we believe that it is both possible and important to clarify the links among emotion, emotion regulation, and psychopathology. Doing so will require that we sharpen our conceptual and empirical focus. To this end, we begin by providing working definitions of *emotion* and *emotion regulation*. Next, we examine the role of emotion and emotion regulation in different forms of psychopathology; our goal in doing so is to illustrate rather than exhaust the range and diversity of problems evident among the various disorders. Finally, we conclude by considering implications for clinical assessment and intervention.

## Defining Emotion and Emotion Regulation

Emotions such as anger, amusement, fear, and sadness arise when an individual attends to a situation and appraises it as being immediately relevant to his or her currently active goals. As emotions arise, they typically involve loosely coupled experiential, behavioral, and physiological responses: One feels, behaves, and mounts whole-body responses. These changes are what we have in mind when we refer to *emotional reactivity*. This perspective on how emotions arise and unfold over time is referred to as the modal model (see Fig. 1), and it highlights various steps in the emotion-generative process, including the situation that compels attention, the evaluation of that situation, and the multisystem whole-body response (Gross & Thompson, 2007).

*Emotion regulation* occurs when one activates—either implicitly or explicitly—a goal to influence the emotion-generative process (Gross, Sheppes, & Urry, 2011). Emotion regulation can take many different forms, depending on the context. For example, emotion



**Fig. 2.** The process model of emotion regulation. From “Emotion Regulation: Conceptual Foundations,” by J. J. Gross and R. A. Thompson, 2007, in *Handbook of Emotion Regulation* (p. 10), J. J. Gross (Ed.), New York, NY, Guilford Press. Copyright by Guilford Press. Adapted with permission.

regulation may be *intrinsic/intrapersonal* (regulating one’s own emotions) or *extrinsic/interpersonal* (regulating someone else’s emotions). Despite the many forms that emotion regulation can take, there are three important common factors for adaptive regulation—awareness, goals, and strategies.

*Awareness* of emotions, as well as the context in which they are occurring, is a powerful support for adaptive emotion regulation (Barrett, Gross, Conner, & Benvenuto, 2001; Farb, Anderson, Irving, & Segal, 2014). Although emotion regulation may be either *explicit* or *implicit*, emotional awareness seems to enhance both the range of available strategies and the flexibility with which one uses them. A second important factor in adaptive emotion regulation is the emotion-regulation *goal*—that is, what one means to achieve. Emotion-regulation goals include increasing or decreasing the magnitude or intensity of emotion experience, expression, or physiology. A third factor that is important to adaptive emotion regulation is the specific *strategies* that are executed to achieve the emotion-regulation goal. Whereas emotion-regulation goals specify the ends, emotion-regulation strategies specify the means.

Although there are various strategies that may be implemented to achieve emotion-regulation goals, the process model of emotion regulation (Gross, 1998; see Fig. 2) is one widely used framework for organizing emotion regulatory processes (Webb, Miles, & Sheeran, 2012). Following this framework, five families of emotion-regulation processes can be distinguished according to when they have their primary impact on the emotion-generative process. These include situation selection, situation modification, attentional deployment, cognitive change, and response modulation. *Situation selection* refers to influencing whether one will encounter a specific situation that is likely to generate an emotion that is either desired or not desired. *Situation modification* refers to attempts to alter external features of the environment in an effort to influence one’s emotions. When individuals

use *attentional deployment*, they direct (or redirect) attention in such a way as to alter their emotional response. *Cognitive change* refers to efforts to revise the meaning of the situation in an attempt to influence one's emotions. Last, *response modulation* refers to efforts made to influence one's experiential, behavioral, or physiological responses to the emotion-eliciting situation. In any given emotion-generative cycle, an individual moves from left to right through the process model as a function of time, and in everyday life, it is common to engage in behaviors that represent various combinations of different strategies rather than a single isolated strategy. It is also important to be clear that emotion-regulation strategies can be adaptive or maladaptive, depending on the specific individual, the emotion, and the context (Aldao & Nolen-Hoeksema, 2012), and that there is considerable variation both between and within families of emotion-regulation processes (Webb, Miles, & Sheeran, 2012).

In the next two sections, we separately examine (a) the relationship between emotion and psychopathology and (b) the relationship between emotion regulation and psychopathology. We do this by reviewing illustrative *DSM-5* disorders. This organizational scheme suggests a crucial question: Are all problematic patterns of emotion due to problems with emotion regulation? We do not think so. We believe that there are many internal (e.g., temperamental) as well as external (e.g., toxic situational) factors that can give rise to problematic patterns of emotion that are best understood in terms of emotion-generative processes rather than emotion-regulation processes. One further complication is that the degree to which emotion problems arise from emotion regulatory difficulties varies from person to person and from context to context. That said, we believe that one particularly important cause of many problematic patterns of emotion is *emotion dysregulation* due to either *emotion-regulation failures* (i.e., not engaging regulation when it would be helpful to do so) or *emotion misregulation* (i.e., using a form of emotion regulation that is poorly matched to the situation; Gross, 2013).

## Emotion and Psychopathology

More than two thousand years ago, Aristotle suggested that emotions are adaptive if they are expressed in the right way, last the right amount of time, arise in the right circumstances, and are about the right things. On this account, individuals with psychopathology might be expected to have problems with emotional intensity, emotion duration, emotion frequency, or emotion type. In the sections that follow, we link each of these aspects of emotional responding with various forms of psychopathology. However, two caveats are in order. First, disorders often involve several types of emotion-related

difficulties. Second, as a result of the heterogeneous nature of psychological disorders, our descriptions may not apply to all individuals within a single diagnostic category and may apply to individuals outside the referenced diagnostic category. Rather than conduct an exhaustive review, our goal here is to highlight the diversity of emotion-related problems evident in psychiatric disorders in the *DSM*, thereby making the case that it is important to move beyond broad claims about "emotion problems" or "problems with emotion processing" to more specific statements about the precise nature of these problematic emotional responses. Wherever possible, we include empirical research from the field of affective science; although as we discuss, for several disorders that we highlight, much work remains to be done.

### Problematic emotional intensity

Problematic emotional intensity can refer to either too large or too small a response (see Fig. 3a). Hyperreactivity is conceptualized as an *overreaction* to a situation, whereas hyporeactivity is conceptualized as an *underreaction* to a situation (Berenbaum et al., 2003). Intensity problems can occur with negative or positive emotions, and within each disorder, problematic emotional intensity may involve different emotion components (experience, behavior, or physiology). To illustrate, we examine one disorder that exhibits hyperreactivity to specific negative emotions (social anxiety disorder), one disorder that exhibits hyporeactivity to specific positive emotions (antisocial personality disorder), and one disorder that exhibits both hyperreactivity and hyporeactivity (major depressive disorder).

Social anxiety disorder is a fear-based disorder that involves high levels of distress occasioned by the possibility of being evaluated by others (American Psychiatric Association, 2013). Many studies have indicated greater intensity of emotion experience in individuals with social anxiety disorder compared with healthy adults. For example, when they view social-threat stimuli, individuals with social anxiety disorder report feeling greater anxiety than do healthy control individuals (e.g., Goldin, Manber-Ball, Werner, Heimberg, & Gross, 2009). What is less clear, however, is whether individuals with social anxiety disorder also show greater intensity in physiological responses. In a nonclinical study of participants with low versus high trait social anxiety (Mauss, Wilhelm, & Gross, 2004), participants with high trait social anxiety reported feeling greater anxiety, exhibited more anxiety behavior, and perceived greater physiological activation than did participants with low trait social anxiety; however, the two groups did not differ in terms of objectively measured physiological responses.

A similar pattern of decoupling between self-reported anxiety and objective biological measures was shown in a study that compared healthy adults with individuals with social anxiety disorder (Ziv, Goldin, Jazaieri, Hahn, & Gross, 2013). As expected, individuals with social anxiety disorder self-reported greater negative emotions; however, neurally, responses in the amygdala and insula between the two groups were similar. These findings challenge a simple global hyperreactivity account of social anxiety disorder and suggest a more nuanced account in which some—but not all—of the emotion-response components may show greater intensity levels in social anxiety disorder. Much remains to be learned, however, about the nature and extent of response dissociation in social anxiety disorder.

Antisocial personality disorder is an example of a disorder in which individuals exhibit emotional hyporeactivity. This disorder is characterized by a reckless disregard for oneself and others, a failure to conform, aggressiveness, irritability, impulsivity, deceitfulness, irresponsibility, and most notably for the present purposes, a lack of remorse (American Psychiatric Association, 2013). Individuals with antisocial personality disorder often feel indifferent even when they have mistreated, stolen from, or hurt another person. A lack of remorse is a common feature of this disorder: According to a national epidemiologic survey, half of individuals diagnosed with antisocial personality disorder endorse a lack of remorse (Goldstein et al., 2006). Although not recognized within the *DSM*, it has been noted that there are potentially important subgroups within antisocial personality disorder (Poythress et al., 2010). For example, the presence or absence of specific affective patterns (such as a lack of empathy/remorse) may in fact highlight important subgroup distinctions. This observation fuels the debate regarding the “heterogeneity of antisocial personality disorder and the atheoretical orientation of the *DSM* with respect to antisocial personality disorder” (Poythress et al., 2010, p. 399). The apparent hyporeactivity of remorse in individuals with antisocial personality disorder is an area for continued research. For example, there are intriguing indications that the characteristic lack of remorse may be related to the relative incapacity to detect and understand fear expressions in others (Marsh & Blair, 2008).

Sometimes, disorders include both hyperreactivity and hyporeactivity. Consider the case of major depressive disorder, whose core features include an excess of negative emotions as well as a deficit of positive emotions. According to the *DSM*, major depressive disorder includes a depressed mood for most of the day, nearly every day; feelings of worthlessness and guilt; and thoughts of death or pervasive suicidal ideation. Major depressive disorder is also associated with a loss of, or diminished interest or

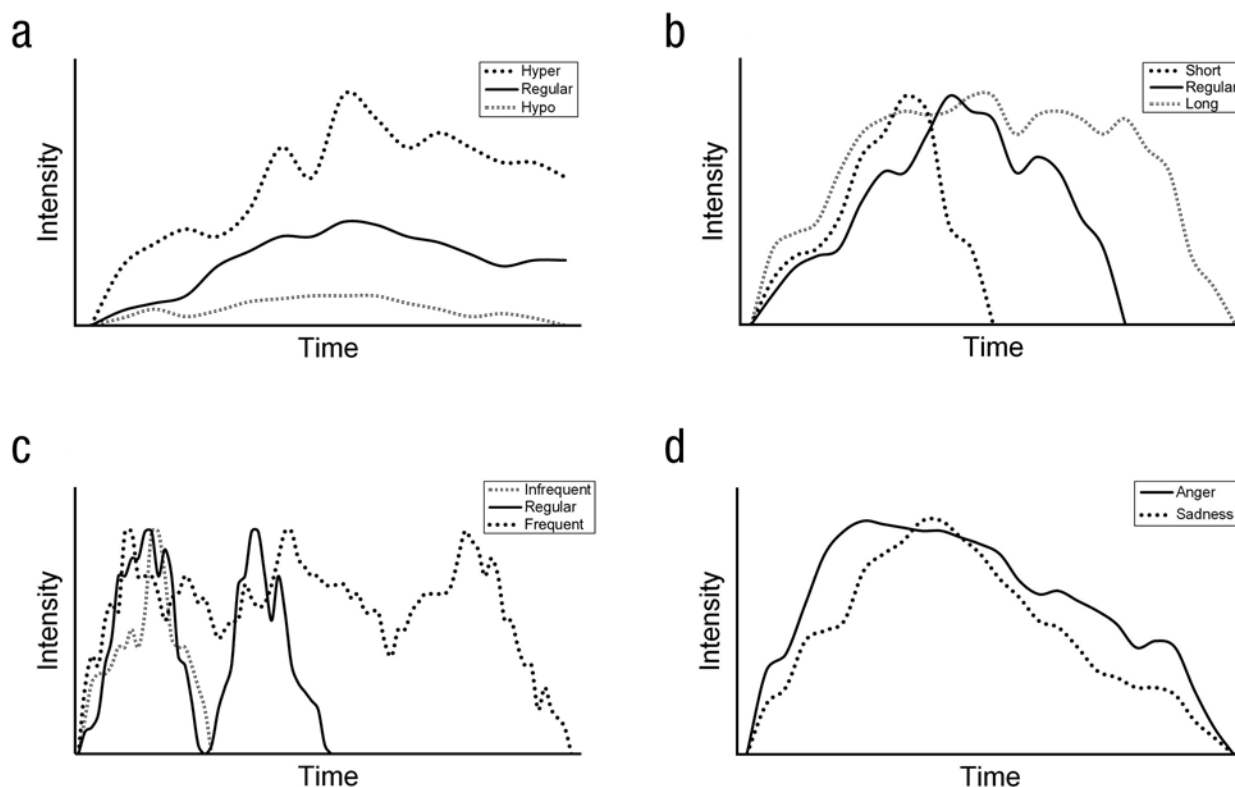
pleasure in, most (if not all) of one’s usual activities that previously generated positive emotions (American Psychiatric Association, 2013). The empirical literature converges with the *DSM* criteria and clinical expectations in terms of hyporeactivity of positive emotion; however, it is a bit more mixed with regard to hyperreactivity of negative emotion. In one experience-sampling study, individuals with major depressive disorder reported greater daily negative affect, lesser positive affect, and fewer pleasant events when compared with healthy individuals (Bylsma, Taylor-Clift, & Rottenberg, 2011). However, a meta-analysis of self-reported experience, expressive behavior, and peripheral physiology suggested “emotion context insensitivity” (Rottenberg, Gross, & Gotlib, 2005)—a profile of reduced positive and reduced negative reactivity in individuals with major depressive disorder compared with healthy adults (Bylsma, Morris, & Rottenberg, 2008). Gaps such as these between clinical expectation and actual responding suggest the pressing need for additional research.

### ***Problematic emotional duration***

Problems with emotion duration occur when emotions are either too short or too long for a particular situation (see Fig. 3b). To illustrate, we examine one disorder in which the duration of negative emotion is too long (specific phobia), one in which the duration of positive emotion is too short (posttraumatic stress disorder, PTSD), and one in which the duration of emotion is both too long and too short (borderline personality disorder).

Specific phobia is a disorder that involves multiple emotion-related difficulties; here, we consider the issue of prolonged duration of negative emotion. *Specific phobia* is defined as a persistent, excessive, and unreasonable fear when anticipating, or in the presence of, a feared object or situation (e.g., air travel, heights, animals). The feared stimuli generally are avoided, and if avoidance is not possible, they are endured with intense anxiety and distress (American Psychiatric Association, 2013). Common phobic stimuli include animals (e.g., snakes, spiders, insects, dogs), natural environments (e.g., heights, storms, water), blood-injection-injury (e.g., needles, medical procedures), and situational contexts (e.g., airplanes, elevators, small enclosed spaces). Like many anxiety disorders, specific phobias are characterized by the extended durations of negative emotions. Although it is almost definitional to specific phobia, if one considers the empirical record on the prolonged duration of negative emotion within specific phobia, it becomes clear that much remains to be done. For example, future research is needed that focuses on individuals within a particular category of specific phobia (e.g., situational) compared with healthy adults, with the aim of





**Fig. 3.** Graphic representations of emotion problems as a function of intensity and time, including problematic emotional (a) intensity, (b) duration, (c) frequency, and (d) type. Hyper = hyperreactivity; Hypo = hyporeactivity.

precisely answering the question of which features of the emotional response are truly persistent and prolonged within specific phobia. It is possible that some features of emotional response are prolonged, but perhaps not all are.

One disorder characterized by too brief emotional responses is PTSD. PTSD is a severe anxiety disorder triggered by exposure to a traumatic event. It is associated with a persistent, involuntary reexperiencing of a previously experienced traumatic event; avoidance of stimuli associated with the trauma; emotional numbing, including a restricted range of affect; and increased arousal and reactivity (American Psychiatric Association, 2013). Although PTSD is often associated with being “quick tempered” (Criterion E1) or with an exaggerated fear response (or overexpression of emotion; Criterion E3/E4), according to a meta-analysis, unlike other anxiety disorders (e.g., social anxiety disorder, specific phobia), PTSD is also associated with hypoactivation in brain structures associated with emotional experience (Etkin & Wager, 2007). As a result of the link between anhedonia (inability to experience pleasure from things that were once enjoyable) and PTSD (Kashdan, Elhai, & Frueh, 2006), this neural hypoactivation may be related to a

decrease in the experience (in the form of emotional duration) of positive emotion. Clinical criteria related to this apparent hypoactivation of positive emotion include diminished interest and participation in once-pleasurable activities (Criterion D5), feelings of detachment or estrangement from others (Criterion D6), and, more specifically, “persistent inability to experience positive emotions (e.g., inability to experience happiness, satisfaction, or loving feelings)” (Criterion D7; American Psychiatric Association, 2013, p. 272).

Borderline personality disorder is characterized by a pervasive pattern of volatile interpersonal relationships, unstable self-image, intense and unstable affects, behavioral impulsivity, and suicidal behavior (American Psychiatric Association, 2013). A situation often will provoke an intense negative emotional reaction that will last for hours or perhaps even days longer than normal (Linehan, 1993). Although greater negative emotional intensity is generally associated with borderline personality disorder, here, we consider emotion duration. Whereas it has been empirically documented that greater negative emotional intensity is associated with slower recovery in borderline personality disorder (startle response, shame; Ebner-Priemer et al., 2005; Gratz, Rosenthal, Tull, Lejuez,

& Gunderson, 2010), a few studies have demonstrated a longer duration emotional response even in the absence of differences in emotional intensity. For example, in a comparison of individuals with borderline personality disorder with healthy adults, Jacob et al. (2008) found that borderline personality disorder was not associated with stronger anger reactions; however, the anger reaction was significantly prolonged in individuals with borderline personality disorder. At the same time, borderline personality disorder also has been associated with shorter emotion duration. For example, individuals with borderline personality disorder often endorse being on an “emotional roller coaster,” experiencing a variety of rapidly changing emotions (e.g., anxiety and depressive symptoms) and, the *DSM-5* suggests, frequent mood changes; however, an empirical record reflecting these observations is conspicuously absent. Future research may use daily experience sampling to capture the shorter durations of emotional experience in borderline personality disorder.

### **Problematic emotion frequency**

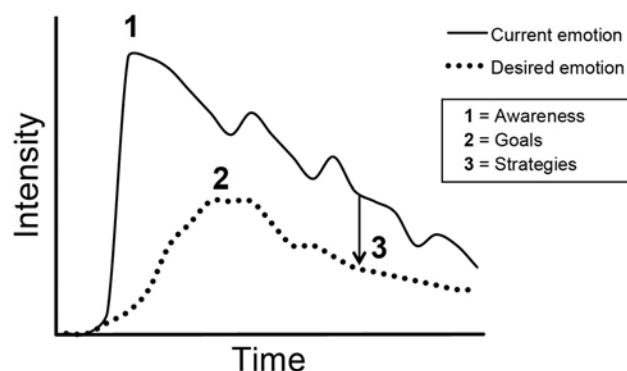
Many individuals who meet criteria for psychopathology experience emotions too frequently or too infrequently (see Fig. 3c). To illustrate, we examine a disorder in which emotions occur too frequently (intermittent explosive disorder), a disorder in which emotions occur too infrequently (dysthymia/persistent depressive disorder), and a disorder in which emotions may occur both too frequently and too infrequently (autism spectrum disorder).

Intermittent explosive disorder is characterized by recurrent and impulsive aggressive outbursts. These outbursts can include verbal or physical aggression occurring on average twice weekly for the past 3 months (Criterion A1) or three behavioral outbursts that involve physical assault occurring within a 12-month period (Criterion A2; American Psychiatric Association, 2013). Intermittent explosive disorder is associated with several types of emotion problems; here, we focus on the issue of negative emotions occurring too frequently. For children (ages 6 years and older), adolescents, and adults with intermittent explosive disorder, although the duration of outbursts is not prolonged (typically lasting less than 30 min), the outbursts occur more frequently (at least twice weekly for a period of 3 months) than is developmentally appropriate, given the precipitating provocation or psychosocial stressors. Unfortunately, because previous editions of the *DSM* did not specify frequency of outbursts in intermittent explosive disorder (and instead indicated “several discrete episodes”), it is not known just how frequent outbursts typically are in this disorder (Coccaro, 2012). Some researchers have previously operationalized this as three or more lifetime

attacks, whereas other researchers have used more narrow definitions of three attacks within the same year (Kessler et al., 2006). Thus, with the concretely defined Criteria A1 with regard to frequency (approximately two outbursts per week) in the *DSM-5*, the issue of negative-emotion frequency in intermittent explosive disorder is an area that can now be examined more precisely in future research.

Individuals with dysthymia (now referred to as persistent depressive disorder in the *DSM-5*) experience a depressed mood for more days than not and for a period of 2 years or longer. In addition to feeling depressed, individuals with dysthymia may experience low energy or fatigue, low self-esteem, and feelings of hopelessness (American Psychiatric Association, 2013). Like other disorders, dysthymia involves multiple emotion problems; here, we focus on problematic infrequency of positive emotions. One study (Casement et al., 2008) investigated the anticipation of future affective events (negative, neutral, and positive) in individuals with dysthymia versus healthy control participants. With regard to behavior, healthy control participants expected fewer negative adjectives to apply to them in the future than either neutral or positive adjectives. In contrast, individuals with dysthymia expected fewer positive adjectives to apply to them in the future than either neutral or negative adjectives. Although affect and emotion are heavily studied in dysthymia's close cousin—major depressive disorder—far less is known about the role of emotion in dysthymia, in part because of high comorbidity rates (e.g., comorbid major depressive disorder in up to 75% of cases, comorbid anxiety disorders in up to 50% of cases; Sansone & Sansone, 2009). Future research on individuals who meet primary criteria for dysthymia without comorbidity is needed. Within these individuals, researchers could employ experience-sampling methods to examine the frequency of specific negative and positive emotions.

Some disorders involve both problematic emotion frequency and infrequency. One example is autism spectrum disorder, a disorder characterized by persistent deficits in social interaction and communication, as well as restricted or repetitive behavioral patterns, activities, or interests (American Psychiatric Association, 2013). These deficits sometimes take the form of too infrequent emotions, such as when an individual with autism spectrum disorder has fewer positive empathic responses than typical when a friend is happy and sharing good news. At other times, the problem is too frequent emotions, such as the high frequency of temper tantrums common in autism spectrum disorder (Maskey, Warnell, Parr, Le Couteur, & McConachie, 2013). Although typically characterized as a disorder of childhood, emotion difficulties often persist into adulthood (Samson, Huber, & Gross, 2012), and much remains to be done to clarify



**Fig. 4.** Important factors in emotion dysregulation as a function of intensity and time. Successful emotion regulation involves appropriate emotional awareness, emotion-regulation goals, and emotion-regulation strategies.

the nature and extent of emotion-related problems in autism spectrum disorder.

### **Problematic emotion type**

In our discussions of problematic emotional intensity, duration, and frequency in the prior subsections, the emotion type was generally appropriate given the context. In other disorders, however, the emotion type is what is problematic for the individual (see Fig. 3d). To illustrate this type of problem, we consider a disorder (schizophrenia) in which, for some individuals, the emotion type that is displayed may be inappropriate for the given context.

Schizophrenia is characterized by delusions, hallucinations, disorganized speech, grossly abnormal psychomotor behavior (e.g., catatonia), and negative symptoms (e.g., restricted affect, avolition), as well as social and occupational dysfunction (American Psychiatric Association, 2013). Emotional disturbances in schizophrenia affect a wide range of emotion processes; here, we examine problems with emotion type and, more specifically, the display of odd or inappropriate emotions. Such displays of inappropriate emotional responses may occur during social interactions, such as displaying anger in a situation in which sadness might be expected. The precise nature of the emotion-generative process that leads to inappropriate emotional responses is not yet well specified, but researchers are interested in responses to both negative (Seok et al., 2006) and positive (Gard, Kring, Gard, Horan, & Green, 2007) stimuli. Furthermore, some research has indicated that individuals with schizophrenia often report emotions that do not match the stimuli (Strauss et al., 2011); a growing body of research has pointed to “affective ambivalence,” with individuals with schizophrenia reporting more negative reactions to

positive stimuli and more positive reactions to negative stimuli (Cohen & Minor, 2010). Taken together, these results indicate a possible impairment in the ability to clearly differentiate and process various emotion types. More research is needed to clarify the role of problematic emotion type in schizophrenia.

## **Emotion Regulation and Psychopathology**

One important cause of many problematic patterns of emotion intensity, duration, frequency, or type—although by no means the only cause—is emotion dysregulation. As noted earlier, *emotion dysregulation* can be considered to be an umbrella term, such that emotion dysregulation may be due to either emotion-regulation failures (i.e., not engaging regulation when it would be helpful to do so) or emotion misregulation (i.e., using a form of emotion regulation that is poorly matched to the situation; Gross, 2013).

Many factors contribute to emotion dysregulation (Rottenberg & Gross, 2003), and a number of frameworks for understanding problems in emotion regulation have been presented (Gross, 2013; see also Gollwitzer, Jaudas, Park-Stamm, & Sheeran, 2008; Koole, 2009; Shah, Friedman, & Kruglanski, 2002; Webb, Schweiger Gallo, Miles, Gollwitzer, & Sheeran, 2012). In the sections that follow, we highlight the role of three important factors in emotion dysregulation, namely, awareness, goals, and strategies. Our view is that, in general, to change problematic emotions, it helps to have (a) an awareness of the emotion and the relevant context, (b) knowledge of one’s short- and long-term goals, and (c) skillful choice and implementation of emotion-regulatory strategies to get from one’s current state to one’s desired goal state (see Fig. 4). To illustrate the role that problems with each of these factors play in psychopathology, we continue with our strategy of selecting representative disorders and including empirical research from the field of affective science if possible. It is again important to note that this is not intended to be an exhaustive review but, rather, an opportunity to examine a variety of disorders that illustrate emotion-regulatory difficulties. As before, it is important to keep in mind that most of the disorders reviewed will fall into multiple categories in relation to emotion-regulation difficulties and that as a result of heterogeneity among disorders, not all of our categorizations will apply to all individuals within a single diagnostic category.

### **Problematic emotional awareness**

Awareness of one’s emotions facilitates adaptive emotion regulation, but more awareness is not always better. To illustrate the role of problematic emotional awareness in



emotion regulation, we consider one disorder in which individuals exhibit hyperawareness of emotion (panic disorder) and one disorder in which individuals appear to exhibit a lack of awareness (bulimia nervosa).

An individual with panic disorder experiences recurrent and unexpected panic attacks. These attacks are followed by at least 1 month of persistent concern about subsequent attacks, worries about implications or consequences of panic attacks, and significant change in behavior (American Psychiatric Association, 2013). Panic disorder is accompanied by intense physiological sensations and discomfort and, thus, often is characterized as a disorder involving a "faulty alarm system," whereby the individual is hyperaware of bodily changes and interprets each change as a cue that something is going terribly wrong. Often, individuals with panic disorder will convince themselves (through their evaluation of their emotional experience) that a catastrophic panic attack will soon ensue (American Psychological Association, 2013). This hypervigilance is assumed to be a core feature of panic disorder (Schmidt, Lerew, & Trakowski, 1997), but some studies have suggested that somatosensory amplification is not always evident in panic disorder (De Berardis et al., 2007). What does seem crucial, however, is attention to physiological changes coupled with a negatively biased appraisal of these changes. This leads to heightened levels of anxiety, which may reach such intensity levels that it becomes very difficult to regulate this anxiety. Continued research on this hallmark feature of panic disorder is needed.

Other disorders are characterized by hypoawareness regarding emotion. One term for such hypoawareness is alexithymia, which refers to the inability to accurately recognize, describe, and communicate one's own emotions, as well as a difficulty with differentiating bodily sensations from feelings (Bagby & Taylor, 1997). Here, we consider the role of alexithymia within eating disorders. In particular, we consider bulimia nervosa, which is characterized by out-of-control binges of large quantities of food often accompanied by compensatory behaviors to avoid gaining weight (American Psychiatric Association, 2013). It has been estimated that more than half of individuals with eating disorders suffer from alexithymia (e.g., Corcos et al., 2000) and, thus, show less awareness of their emotions than do healthy individuals. Without emotional awareness, it is much more difficult to engage sophisticated emotion-regulation strategies, and strategies that are available (e.g., suppression) may be less effective. Despite the role of alexithymia within bulimia nervosa and the various treatments for bulimia nervosa that address emotion and emotion-regulation difficulties (e.g., Safer, Telch, & Agras, 2001), the diagnostic criteria for bulimia nervosa in the *DSM-5* (similar to other editions of the *DSM*) fails to acknowledge any emotion or

emotion-regulatory problems. Continued empirical research as well as conceptual and theoretical sharpening regarding the role of emotional awareness within bulimia nervosa and other eating disorders is sorely needed.

### ***Problematic emotion-regulation goals***

Emotion-regulation goals refer to what the individual would like to achieve with regard to the specific emotion. A healthy profile of emotion-regulation goals requires a judicious weighing of short-term and long-term concerns. Failure to appropriately consider the balance of short- and long-term concerns may lead to problematic emotion-regulation goals.

One disorder that appears to be characterized by dysfunctional emotion-regulatory goals is Bipolar I, a disorder characterized by a period of abnormal and persistently elevated, expansive, or irritable mood that lasts for a minimum of 1 week (though typically longer). During this time, symptoms may include an inflated self-esteem or grandiosity, decreased need for sleep, talkative behavior, insomnia or hypersomnia, psychomotor agitation or retardation, flight of ideas or racing thoughts, an increase in ambitious goal-directed activity, and an increased involvement in pleasurable but risky behaviors (American Psychiatric Association, 2013). During these manic states, the individual with Bipolar I generally reports feeling euphoric, and as a result of the reinforcing nature of "feeling good," the individual is largely uninterested in downregulating his or her emotional state. The empirical literature is beginning to explore the issue of positive emotion within bipolar disorder (e.g., Gruber, 2011). It is not clear, however, whether the failure to downregulate approach-related emotions is due to a failure to foresee negative consequences or to an inability to appropriately consider these long-term consequences. At least some evidence has suggested that the more manic the individual becomes, the less regard he or she has for the adverse longer-term consequences of continued goal pursuit (Meyer, Johnson, & Winters, 2001). Continued investigation into the patterns of emotion-regulatory goals for individuals with Bipolar I disorder (both in the presence and in the absence of a manic state) is needed.

### ***Problematic emotion-regulation strategies***

Emotion-regulatory strategies refer to the ways in which individuals attempt to achieve their emotion-regulatory goals. Many psychiatric disorders appear to involve problematic emotion-regulation strategy choice and problematic emotion-regulation strategy implementation (Jazaieri et al., 2013). In the following discussion, we consider

problematic choice of emotion-regulation strategies separately from problematic implementation. We continue our approach of drawing on examples of disorders in which emotion-regulation choice is problematic (agoraphobia) or emotion-regulation implementation is problematic (attention-deficit/hyperactivity disorder, ADHD).

Many different factors are relevant when choosing the appropriate emotion-regulation strategy to use in a particular context. One important factor is differential overall effectiveness, given that there is now good evidence that strategies vary in terms of their effectiveness (Webb, Miles, & Sheeran, 2012). A second important factor is the availability of resources needed to successfully employ a particular form of emotion regulation. It is becoming clear that different forms of emotion regulation make somewhat different cognitive demands, and, however effective a strategy may be, it is unlikely to be chosen if necessary resources are unavailable (Urry & Gross, 2010). A third important factor is the intensity of the emotion that needs to be regulated. For example, in a series of studies, Sheppes and colleagues (Sheppes, Scheibe, Suri, & Gross, 2011; Sheppes et al., 2014) found that healthy participants chose to implement reappraisal in low-intensity emotional situations and distraction in high-intensity emotional situations. If healthy emotion-regulation choice involves an awareness of the differential efficacy of various forms of emotion regulation, an accurate assessment of the resources needed to implement each strategy, and a clear understanding of key dimensions of the current situation (e.g., the intensity of the emotion), it follows that psychopathology might involve difficulties with one or more of these several steps or with the appropriate weighting of these various (often competing) factors.

One disorder that illustrates problematic emotion-regulation choice is agoraphobia, which has at its core an overuse of situation selection (influencing whether one will encounter a specific situation that is likely to generate an emotion that is either desired or not desired). Agoraphobia is an anxiety disorder that commonly co-occurs with panic disorder whereby the individual avoids or experiences extreme fear or anxiety in situations from/in which he or she believes that it will be difficult to escape/receive help (or in the case of co-occurring panic disorder, situations that the individual believes may induce a panic attack). Similar to other anxiety disorders (e.g., social anxiety disorder), individuals with agoraphobia often feel that their fear or anxiety is out of proportion to the danger posed by the situation (Criterion E). This fear or anxiety typically involves two or more of the following situations: public transportation, open spaces, enclosed spaces, standing in line or being in a crowd, or being outside of the home alone (Criterion A; American Psychiatric Association, 2013). Thus, situations that are avoided may include everyday places, including crowded

spaces, such as stadiums or movie theaters, as well as bridges or other enclosed spaces where escape or receiving help is perceived to be difficult. At its extreme, individuals with agoraphobia go to such great lengths to avoid feared situations that they sequester themselves at home or limit themselves to environments that are deemed “safe.” As a result of the high rates of comorbidity, the majority of research conducted on agoraphobics includes comorbid panic disorder. However, one study of adults with agoraphobia investigated the occurrence of imagery and memories, given that this has been implicated to be involved in the maintenance of anxiety disorders (e.g., Clark & Wells, 1995). Researchers found that all 20 patients reported experiencing distinct, recurrent, and distressing images to agoraphobic situations. Thus, although behavioral avoidance was taking place, these patients were still able to vividly recall images related to these situations (which occurred in their adolescence, approximately 35 years ago; Day, Holmes, & Hackmann, 2004). These findings suggest that when triggered by cues, agoraphobics are involuntarily “reexperiencing” the negative situations even while behaviorally avoiding these feared situations, which may contribute to the maintenance of the disorder.

Even if a specific emotion-regulation strategy is recognized as appropriate given the context, and chosen by the individual, a certain amount of skill and confidence are necessary for proper execution of the regulatory strategy (Tamir & Mauss, 2011). If the skills and confidence required for implementation are absent, then the execution of the emotion-regulatory strategy will likely be ineffective. Two components of implementation are *goal shielding*, which refers to protecting the emotion-regulatory goal from other competing goals, and *goal flexibility*, which refers to adjusting the emotion-regulation goal if needed as the situation changes (Gollwitzer et al., 2008; Gross, 2013; Shah et al., 2002).

One psychiatric disorder that involves problematic emotion-regulation implementation is ADHD. ADHD includes three subtypes: inattentive/disorganized, hyperactive/impulsive, and combined inattentive and hyperactive. Symptoms of ADHD inattentive/disorganized type include being easily distracted, poor concentration and a lack of attention to detail, forgetfulness, and, overall, having a difficult time with organization, following instructions, and completing tasks. Features of fidgetiness, restlessness, aggression, and antisocial traits are generally characteristic of the hyperactive/impulsive subtype only. Symptoms must appear before the age of 7, must be present for a minimum of 6 months, are disruptive, and are inconsistent with the current developmental level of the individual (American Psychiatric Association, 2013). Given the nature of the difficulties observed in ADHD, it stands to reason that an individual with ADHD might

have difficulties consistently implementing emotion-regulation goals, possibly because of difficulties with goal shielding in light of the powerful impact of bottom-up effects of attention on other competing goals. Empirical research has yet to indicate a particular pattern of ADHD-related emotion-regulation difficulties, given that the existing research lacks consistency (see Cole, Martin, & Dennis, 2004; Mullin & Hinshaw, 2007). Additional research is urgently needed to examine emotion-regulation choice and implementation within specific subtypes of ADHD because it is important to understand whether ADHD subtypes differ in their abilities to implement emotion-regulatory strategies.

## Implications for Clinical Assessment and Treatment

The first step in charting a course for future research is to increase awareness of the gap between clinical intuition and empirical findings. It is far easier to make strong claims about “emotion dysregulation” than it is to convincingly demonstrate specific problems with emotion or emotion regulation in a particular disorder. If, as researchers, we are to ground our assessments and interventions on empirical findings—as we must—clarity about the gap between what we think we know and what we actually know is the first step.

As we look to the future, we see a number of important directions for future research that will draw on affective science to test specific hypotheses regarding the role of emotion and emotion regulation in psychiatric disorders. In the following subsections, we consider implications for assessment and treatment.

### Assessment

The *DSM* is currently the gold standard for assessment. Many disorders (e.g., mood and anxiety disorders, borderline personality disorder) clearly have emotional pathology at their core, whereas other disorders (e.g., gender identity disorder, antisocial personality disorder) contain less pervasive and obvious difficulties with emotion and emotion regulation. Although it has been reported that when systematically coding the formal written criteria in the previous edition of the *DSM*, only 40% of disorders included an explicit reference to affective disturbance (Jazaieri et al., 2013), we believe that problematic emotion and emotion regulation may be more pervasive than suggested by the formal written *DSM* criteria. Within the context of the *DSM*, it will also be important to distinguish between problems with emotion and emotion regulation that are part of the definition of the disorder and problems with emotion and emotion regulation that are evident in individuals who have a

particular disorder but that are not part of the defining criteria. Another pressing need is to clarify which diagnostic features should be considered primary deficits of the disorder versus secondary or compensatory deficits (e.g., which result from the individual's attempt to cope with the primary deficit).

It will not be enough, however, to focus on just the formal *DSM* disorders. This is because the *DSM* does not take into account subclinical presentations of emotion and emotion-regulation difficulties. For example, to meet diagnostic criteria for major depressive disorder, an individual must endorse five out of nine symptoms within the last 2 weeks; thus, an individual may endorse all of the affect- and emotion-related difficulties for major depressive disorder (e.g., depressed mood, feelings of worthlessness or excessive or inappropriate guilt) and recurring thoughts of death and yet still not meet formal diagnostic criteria for major depressive disorder. Likewise, individuals who fall under *DSM* V or Z codes (e.g., V71.01/Z72.811 Adult Antisocial Behavior) could experience emotion and emotion-regulation difficulties even though they do not formally meet criteria for psychopathology per se. It is therefore possible that the *DSM*, our current gold-standard tool for psychological classification, excludes individuals who experience emotion and emotion-regulation difficulties and yet do not meet the threshold for formal *DSM* criteria for psychopathology.

Another important direction for future research arises from the observation that quite different psychological disorders (according to clinical convention and the *DSM* criteria) may in fact manifest some of the same underlying emotion or emotion-regulation difficulties. Recent research has even suggested that on a genetic level, risk factors across disorders are quite similar (Serretti & Fabbri, 2013; Smoller, Kendler, & Craddock, 2013). In contrast, some disorders that are considered quite similar to each other may in fact be manifestations of quite different underlying causes. Furthermore, as we have attempted to illustrate, clinical-symptom profiles are in fact a complex mix of primary problems with layers (to varying degrees) of secondary problems that may very well result from dysfunctional attempts at managing the primary deficits (e.g., behavioral avoidance in social anxiety disorder). These issues highlight the importance of considering transdiagnostic vulnerabilities and symptom-based approaches when conceptualizing disorders (e.g., Mansell, Harvey, Watkins, & Shafran, 2009). The National Institute of Mental Health Research Domain Criteria Project is moving the field in this direction by implementing new ways of classifying psychopathology using strong empirical research (from a variety of fields, including affective science) as a basis. By empirically elucidating various dimensions of emotional functioning, it may

be possible to enhance practices related to assessment, diagnosis, and treatment of psychological disorders.

## **Treatment**

A number of existing psychological treatments target problems with emotion and emotion regulation. These include cognitive-behavioral therapy (Beck, 1976), dialectical-behavioral therapy (Linehan, 1993), acceptance- and mindfulness-based interventions (e.g., Hayes, Strosahl, & Wilson, 1999; Roemer et al., 2009; Segal, Williams, & Teasdale, 2002), emotion-focused therapy (Greenberg, 2002), the unified protocol for emotional disorders (Barlow, Allen, & Choate, 2004), and emotion-regulation therapy (Mennin & Fresco, 2009). One important challenge is the clarification of the mechanisms of action within each of these treatments. For example, within the context of social anxiety disorder, we have found that shifts in maladaptive interpersonal beliefs (Boden et al., 2012) and cognitive reappraisal self-efficacy (Goldin et al., 2012) may be important mechanisms by which individual cognitive-behavioral therapy for social anxiety disorder produces immediate and longer-term reductions in social anxiety symptoms. However, much remains to be learned about the mechanism or mechanisms responsible for the efficacy of each of these psychosocial treatments, as well as their pharmacologic counterparts. A second important challenge is the improvement of existing treatments. Although effective treatments for emotion and emotion regulation exist, when one examines clinically significant change, these treatments are far from perfect. Because treatment non-responders as well as individuals who are either unable or unwilling to engage in traditional treatments still exist, we see this as an opportunity to continue to refine existing treatments and explore alternative interventions for this subset of the treatment-seeking population suffering from difficulties with emotion and emotion regulation.

In discussions of treatment approaches, it is important to keep in mind that many if not most individuals who would benefit from clinical interventions do not receive them in a timely fashion (Wang et al., 2005). In our work with social anxiety disorder, we have found that the time between onset of social anxiety symptoms and first contact for treatment (psychotherapy or psychopharmacology) is generally more than a decade and frequently multiple decades. Our observations converge with national epidemiologic surveys. For treatment-seeking individuals, the mean age at first contact for treatment is approximately 27.2 years old (approximately 12.1 years after onset), whereas more than 80% of individuals with social anxiety disorder receive no treatment (Grant et al.,

2005). Individuals who meet criteria for social anxiety disorder often are unaware that there is a name and official diagnostic disorder for the set of symptoms that they experience, let alone efficacious pharmacologic and non-pharmacological treatments (often discounting their difficulties as resulting from being “shy” or “introverted”). Although harm may certainly sometimes be done by providing a “label,” these labels can be useful in pointing individuals to literature, support, and treatment for their set (or subset) of symptoms. It is apparent that continued psychoeducation regarding various psychiatric disorders is sorely needed.

Even if it is clear what the problem is, and how treatment may be obtained, a stigma associated with psychotherapy often still exists and may prevent some individuals from actively seeking out treatment. In addition, a significant financial burden is often involved in obtaining treatments such as cognitive-behavioral therapy. Furthermore, depending on location, it may be difficult to access adherent treatment programs (e.g., dialectical-behavioral therapy). In these instances (stigma, finances, accessibility), we believe the use of telephone- or Internet-based treatment may be a valuable option (e.g., see Andersson, Carlbring, & Furmark, 2012), and it is clear that further work in this arena is needed. Recent epidemiological studies have suggested that less than half of the adult population in the United States experience what is termed as “high mental health,” thereby indicating that the prevalence of “optimal mental health is relatively low” (Catalino & Fredrickson, 2011, p. 938). Such findings hint at the enormity of the need for empirically supported treatments that address problems with emotion and emotion regulation. As other researchers have suggested (e.g., Kazdin & Rabbitt, 2013), a revision of the dominant model of mental-health delivery (individual therapy once a week with a professional) may be required to address these pressing mental-health needs.

Attention typically is paid to emotion and emotion regulation only if a problem has occurred (e.g., avoidance, violence) or after a disorder has been diagnosed. We believe that skillful emotion regulation is important for everyone and would like to see a far broader effort to encourage psychoeducation regarding emotion and emotion regulation prior to any indication of pathology. By using the tools of affective science to further understand the etiology of difficulties with emotion and emotional regulation, researchers may be able to develop or refine existing treatment interventions to prevent disorder onset (e.g., by refining treatments and interventions to the point that when deployed early in life, they may preemptively combat the development of emotion and emotion-regulation difficulties that occur later in life).



## Concluding Comment

It is clear that much remains to be done to translate clinical observations about emotion dysregulation into empirical research whose findings will provide an urgently needed foundation for assessment and intervention. One important task is to sharpen our specification of problems with emotion and emotion regulation. Greater conceptual clarity will make it possible to move beyond broad claims about the role of emotion dysregulation to a deeper understanding of the mechanisms responsible for the onset and maintenance of the many clinical and nonclinical conditions that limit individuals' ability to form and sustain relationships and engage in activities that give their lives meaning. Increased precision will also allow us to "mind the gap" between our assumptions and our empirical findings, thereby enabling clearer clinical assessment and more robust—and potentially individually tailored—treatment strategies. These are exciting times in affective and clinical science. Given the enormous needs in this area, and the growing sophistication of research methods, the future seems very bright indeed.

## Author Contributions

J. J. Gross and H. Jazaieri jointly conceived and wrote the manuscript. Both authors approved the final version of the manuscript for submission.

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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.

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