

## When Emotion Goes Wrong: Realizing the Promise of Affective Science

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**One of the fundamental questions that clinical scientists and practitioners alike must address is when emotions are functional and when they are dysfunctional. Recent advances in affective science have provided new tools with which to address this age-old question. In particular, affective science is teaching us a great deal about both the *generation* and the *regulation* of emotion. We argue that it is only by harnessing the insights of contemporary affective science that it will be possible to develop a nuanced taxonomy of emotional disturbances that is grounded in a causal analysis of underlying processes. We illustrate our points by drawing upon our recent studies of depression and discuss several important challenges that lie ahead as we build much-needed bridges between affective and clinical science.**

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In his *Nicomachean Ethics*, Aristotle argued that emotions are adaptive when they are about the right things, are expressed in the right way, arise at the right time, and last the right amount of time. By contrast, emotions are maladapt-

tive when they are about the wrong things, are expressed in the wrong way, arise at the wrong time, or are the wrong duration. This analysis led Aristotle to recommend moderation, and he provided vivid cautionary examples of emotional excesses and deficiencies of both positive and negative emotions.

The issue of how best to distinguish adaptive from maladaptive emotion remains as important to clinical scientists and practitioners today as it was when Aristotle wrote more than two thousand years ago. Happily for us, the past few decades have witnessed an explosion of research on emotion, accompanied by new theories (e.g., evolutionary analyses of emotion; Tooby & Cosmides, 1990), methods (e.g., anatomically based systems for coding facial expressive behavior; Ekman & Rosenberg, 1997), and findings (Cacioppo & Gardner, 1999). These developments have provided new tools with which to address the role that emotions play in health and illness. Although it remains a daunting task to arrange into a meaningful conceptual scheme the many ways that emotions can go wrong, these tools promise to enable researchers and clinicians to formulate more sophisticated conceptualizations of emotional disturbance that will enhance their capacity to describe, diagnose, and treat disorders.

In the rich tradition of Aristotle, Berenbaum and colleagues (2003; this issue) classify emotional disturbances on the basis of observed excesses or deficiencies in emotion. In this comment, we consider what affective science is teaching us about both the *generation* and the *regulation* of emotional responses, and how each of these processes may relate to clinical phenomena. We argue that it is only by fully harnessing the insights of contemporary affective science that it will be possible to develop a comprehensive taxonomy of emotional disturbances that is grounded in a causal analysis of the underlying processes. Throughout, we

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illustrate our points by drawing upon examples from our recent studies of depression.

#### **EMOTION: DEFINITION AND TEMPORAL COURSE**

Discussions of emotion disturbance presuppose and build upon an understanding of what emotion is. Unfortunately, for many years, considerable confusion has clouded the use of emotion-related constructs, with different investigators employing their own, often idiosyncratic, definitions and operationalizations of terms such as *affect*, *emotion*, and *mood*. Recently, affective science has increasingly moved towards standardized nomenclature that provides researchers with common reference points (Gross, 1999).

*Affect*, in this nomenclature, is considered a superordinate category for all valenced states (Scherer, 1984). *Emotion*, a subtype of affect, refers to coordinated responses that occur when an organism encounters meaningful stimuli that exercise its adaptive capacities. Emotional responses prepare an organism for situationally appropriate actions that have generally facilitated the survival of species and individuals over evolutionary time (Tooby & Cosmides, 1990). Importantly, emotions are *multi-componential*—and can be indexed by cognitive changes, by experiential changes, by central and peripheral physiological responses, and by overt behavior. For example, anger may be reflected by thoughts about exacting revenge, feelings of great distress, an elevated heart rate, and an attack on the source of one's anger. *Moods*, though also complex multi-system affective responses, differ in several important ways from emotions. Relative to emotions, moods are generally thought to be longer, slower moving, and less tied to specific objects or elicitors (Watson, 2000). If emotional reactions are like storms, then moods are like seasonal climate change.

Our recent work on the emotional features of depression illustrates the potential payoffs of carefully distinguishing between these different forms of affect. One of the most striking features of depression noted from early psychoanalytic formulations onwards is the pervasive negativity of depressed patients' thinking and affect. Not surprisingly, most investigators have assumed that depression enhances *all* forms of negative affect (i.e., both moods and emotions). Perhaps most notably, cognitive theorists have advanced a view of depression in which negative moods and negative emotions are mutually reinforcing (Beck, 1967, 1976). According to this view, depression can be

conceptualized in terms of cognitive structures that negatively distort the processing of emotional stimuli (e.g., by directing attention to negative features of the environment). Importantly, negative mood states strengthen these cognitive structures, magnifying emotional responses when depressed persons encounter a negative stimulus.

Although the hypothesis that negative moods potentiate negative emotions in depressed individuals is a reasonable one, emotional reactivity to standardized negative emotion-eliciting stimuli has seldom been assessed in depressed persons. In a recent study, we found that, relative to healthy controls, depressed individuals in fact exhibited *less* differential reactivity to sad stimuli, and instead exhibited sadness in a stereotyped, context-insensitive fashion (Rottenberg, Kasch, Gross, & Gotlib, 2002). Moreover, even when displaying a potent form of sadness—tearful crying—depressed persons exhibited less emotional reactivity than healthy controls (e.g., less heart rate acceleration, Rottenberg, Gross, Wilhelm, Najmi, & Gotlib, 2002). Paradoxically, then, our findings suggest that pervasive sad moods in depression may impede (rather than facilitate) the generation of sad emotions. These findings confirm other clinical observations of emotional numbing in depression, and raise the possibility that this disorder can be treated with techniques that enhance patients' emotional reactivity.

In addition to clarifying the nature of its key constructs, contemporary affective science has also begun to clarify the temporal course of affective processes as they unfold over time. For example it has been demonstrated that the course of an emotional response can be profitably decomposed and studied in terms of its temporal constituents, such as rise time to peak or time to recovery (Davidson, 1998). Therefore, in addition to excesses or deficiencies in the overall magnitude of emotional responding, it seems very likely that dysfunctions of emotion will involve abnormalities in one or more of these temporal parameters as well.

For example, recent empirical work suggests that variations in the offset of negative emotion may be particularly important for understanding psychopathology (e.g., Siegle, Steinhauer, Thase, Stenger, & Carter, 2002). One mechanism that may play a role in negative emotion offset is parasympathetic tone, usually indexed by respiratory sinus arrhythmia (RSA), which refers to the variations in heart period that coincide with respiration. In prior work, RSA has been related to organismic flexibility and resiliency in

the face of stressors (reviewed in Rottenberg, Wilhelm, Gross, & Gotlib, 2002). To test whether RSA tracks recovery from negative emotion, we measured RSA fluctuations that occurred over the course of tearful crying episodes. Interestingly, healthy control participants who cried exhibited large increases in RSA that coincided with the resolution of their crying. By contrast, depressed participants who cried did not exhibit RSA rebound over this period, consistent with the idea that depression compromises emotion offset (Rottenberg, Wilhelm, Gross, & Gotlib, 2003).

### **EMOTION REGULATION: ALTERING THE TRAJECTORY OF EMOTION**

Because emotions are flexible behavioral guidance systems (rather than fixed-action patterns), individuals are typically able to shape the course of an emotion episode as it unfolds. It is, therefore, the modulation of emotional response tendencies that determines the final shape of the emotional response (Gross, 1998a). *Emotion regulation* refers to the processes by which individuals influence which emotions they have, when they have them, and how they experience and express these emotions. Emotion regulation is, along with emotion generation, a major theme of contemporary affective science.

Research on emotion regulation suggests that emotion regulatory processes may be automatic or controlled, conscious or unconscious, and that they may have their effects at one or more points in the emotion-generative process (Gross, 2001). In everyday life, individuals use a wide variety of automatic and conscious strategies to influence how and when they have emotions (e.g., Gross, 1998a). To an impressive degree, these self-regulatory efforts are successful. For example, college students are able to down-regulate their expressions of emotion in the laboratory, even in the face of a powerful emotion elicitor and regardless of whether they are instructed to do so through the direct control of their facial musculature or through cognitive reappraisal of the situation (Gross, 1998b). So pervasive is effective emotion regulation, it is often taken for granted. Nevertheless, there is increasing recognition that many people experience serious difficulties in emotion regulation that result in ordinary unhappiness as well as in outright psychopathology.

Given the importance of emotion regulation, its pervasiveness in everyday life, and its likely role in psychopathology, we believe it is critical for emotion regulation

to be thoroughly integrated into taxonomies of emotional disturbance. Emotion generation and emotion regulation are conceptually distinguishable processes that may represent distinct pathways into disorder. Indeed, the pathological nature of many emotion regulation strategies is plain. One might, for example, think of a person's efforts to reduce feelings of shame by refusing to eat, or his or her efforts to ventilate anger by beating up a co-worker. Other emotion regulation strategies, such as avoiding fearful situations, may sometimes assist successful adaptation (e.g., when there is a real threat), but at other times lead to disorder. Although we are still learning about the significance of emotion regulation for mental health, the importance of this idea has already been assimilated by several successful psychological therapies, such as cognitive-behavioral therapies, which are premised on the idea that psychological adjustment can be improved by altering habitual responses to uncomfortable emotions (Gross & Muñoz, 1995).

Distinguishing difficulties with emotion regulation from problems with emotion generation is not always easy. Indeed, although psychopathology often reflects emotion regulation failures, many instances of psychopathology may have less to do with emotion regulation than with abnormalities in the mechanisms of emotion generation. For example, twin concordance data from behavioral genetics studies strongly suggests that some individuals are born with emotion-generative mechanisms that confer risk for later psychopathology (Pedersen, Plomin, McClearn, & Friberg, 1988). This idea is reinforced by temperament research indicating that a person's characteristic affective style (Davidson, 1998) develops from an early age, is stable, and has a significant biological basis (Kagan, 1998). In fact, stable differences in the thresholds for emotion activation are observable in young infants. On the other hand, even these early differences in emotion-generation are to some degree plastic, modulated by an infant's developing self-regulatory skills, such as approach and avoidance behavior and attentional control (Rothbart & Ahadi, 1994).

Depression exemplifies many psychological disorders in that it is believed to have a heterogeneous etiology. Distinctions between emotion generation and emotion regulation pathways into depression may prove useful for explaining aspects of this heterogeneity. For example, a heritable trait such as neuroticism that is associated with a vulnerability to depression likely confers this vulnerability via a lower threshold for the generation of negative emo-

tion (Fanous, Gardner, Prescott, Cancro, & Kendler 2002). However, emotion regulatory pathways to depression also may emerge from dysfunctional ways of attempting to control, or cope with, affective impulses. Depressogenic emotion regulatory strategies are likely to range widely, from rumination (Morrow & Nolen-Hoeksema, 1990), a cognitive style of repetitive self-focus that prolongs and deepens negative mood, to self-medication with alcohol and other drugs. Additional research is needed to examine the role of emotion generation and emotion regulation in depression (and other disorders) and to assess their implications for etiology.

#### **TOWARD AN AFFECTIVE SCIENCE OF PSYCHOPATHOLOGY**

Empirical studies of the processes underlying emotion have already produced valuable insights into psychopathology (Barlow, 1991; Kring & Bachorowski, 1999). We are confident that additional efforts—guided by the best that affective science has to offer—will yield a taxonomy of emotional disturbances that is based on causal analyses of these underlying processes. Before this goal can be achieved, however, three challenges must be addressed.

A first challenge is to articulate when a pattern of emotional disturbance is, in fact, present. Contextual factors such as age, gender, social situation, and culture all complicate the interpretation of whether an emotional response is situationally appropriate. For example, what constitutes excessive sadness? When a person cries over spilt milk is he or she displaying excessive sadness? A two-year-old probably is not, a seven-year-old may be, and an adult probably is (and may have a clinical disorder). An adult male crier would presumably be *easier* than a female crier to classify as excessively sad, given the gender differences in adult crying typically observed in western contexts (Rottenberg, Gross, et al., 2002). In some social situations, such as the funeral of a loved one, prolonged crying may be normative for both genders (and noncrying a sign of deviance). Even at a funeral, however, there is clear cultural variation in the norms for how long, how deep, and how severe the expression of grief is to be. In short, defining what constitutes “excessive sadness,” or any other postulated emotional disturbance, is a deceptively difficult problem that requires researchers to integrate a considerable amount of contextual information into their formulations.

Once an emotional disturbance has been identified, a

second challenge is to analyze it in terms of its component processes. As noted above, the challenge here is that emotion generative and regulatory processes are often tightly intertwined. In fact, rarely is the generation of an emotional response in an adult *not* accompanied by some form of regulation. That is, emotion generative and regulatory processes *co-determine* which emotions we have, when we have them, how intensely we have them, and how long they last. Fortunately, experimental methodologies now exist to help disentangle the relative contribution of generative and regulatory processes in any given emotion outcome, as well as to isolate the biological substrates of each of these component processes (e.g., functional magnetic resonance imaging; see Ochsner, Bunge, Gross, & Gabrieli, in press). As these procedures are increasingly applied to psychiatric populations, we will learn the similarities and differences between disorders in their effects on these component processes, knowledge that is likely to be very useful in refining psychiatric categories and sharpening clinical interventions.

Finally, once a disturbance in emotion generation and/or regulation has been broken down into its component processes, a third challenge is to locate this disturbance within a meaningful overarching framework. Because emotion is multi-faceted, many such frameworks are possible. For example, a taxonomy of emotional disturbance might be organized around each of the major emotion response systems: cognition, experience; physiology (both central and peripheral), or behavior. Moreover, emotions can be analyzed either at the level of broad dimensions (positive, negative) or at the level of discrete emotions (happiness, fear). Therefore, taxonomies of emotional disturbance might be organized around either dimensional or discrete categorizations. Finally, because there are so many points in the emotion-generative process where emotions can go wrong, taxonomists need to decide how emotional disturbances should fall within temporal frames that can vary from milliseconds to hours or days.

Despite these challenges, we believe this taxonomic venture has a bright future. Affective science and clinical science already have many points of contact. We are hopeful that this contact will develop into a happy, long-term marriage. As our basic knowledge about emotion grows, it will be increasingly essential for clinical scientists and practitioners to become familiar with affective science. After all, this body of work provides a rich, empirically

grounded conception of emotion and a set of tools for (1) thinking about (and thus diagnosing) emotional disorders, and (2) identifying specific points in the emotion generative-regulatory process to target for intervention. We expect that the advent of causally based taxonomies of emotional disorders will give clinicians a useful supplement to *Diagnostic and Statistical Manual of Mental Disorders* (4th ed.), which augments the assessment and treatment of various forms of disorder. In our opinion, affective science stands to gain as much as clinical science from this marriage. Just as the understanding of normal brain function has been informed by the study of brain lesions, affective scientists have a profound opportunity to learn about the normal operation of emotion by studying emotion when it goes wrong. Disordered emotional states, like no other phenomena, expose the complexity—and the fragility—of human emotion regulation. In the same way that the insights of affective science will be an asset for clinical scientists, so too will the accumulated mass of clinical observations and data provide a gold mine for affective scientists that will occupy them profitably for many years to come.

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