Emotions in Depression: What Do We Really Know?

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Abstract

Major depressive disorder is among the most common and costly of all mental health conditions, and in the last 20 years, emotional dysfunction has been increasingly seen as central to depression. Accordingly, research on emotions in depression has proceeded with fury. The urgency of the work has tempted investigators to issue premature declarations and to sometimes overlook theoretical and methodological challenges entailed in studying emotion. I report on what we have learned thus far about how depression influences emotional reactivity and emotion regulation, and also carefully demarcate the vast terrain of what we do not yet know. Ironically, an attitude of humility may enable the field to achieve the ambitious but elusive goal of developing a rich, contextually specific account of depression-related changes in emotional reactivity and regulation. Such an account is a precondition for using knowledge about emotion to intervene more effectively to reduce depression's worldwide burden.

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INTRODUCTION

The more I learn, the more I realize how much I don't know. —Albert Einstein

Depression is an urgent societal problem. Major depressive disorder (MDD) is already among the most common and costly of all mental health conditions (Kessler et al. 2003), and it is projected to become the world's most burdensome disease in the coming decades (Lépine & Briley 2011). Logically, it will be difficult to contain depression's personal and societal toll without understanding what resides at its core.

Intuitively, emotion must be a core aspect of depression. From the sufferer's perspective, the most painful aspects of the condition are uncontrollable feelings—of sadness, of emptiness, and of anxiety. Painful feelings drive people into treatment, or much worse, toward acts of self-harm, including suicide. Feelings are also at the diagnostic core of depression. The leading diagnostic manuals define depression as a mood condition: Depression cannot be diagnosed without some evidence of a persistent mood disturbance, such as excessive sadness and/or a greatly reduced experience of pleasure (Am. Psychiatr. Assoc. 2013).

MDD: major depressive disorder

Emotions: acute reactions to threats or opportunities that manifest as loosely coordinated changes in experience, behavior, or physiology

Moods: diffuse, slow-moving feeling states that typically lack an obvious environmental precipitant

Intuitions about emotion only take us so far. Scientists define emotion in a precise way: The concept of emotion refers to acute reactions to threats or opportunities that manifest as loosely coordinated changes in experience, behavior, or physiology (Gross & Thompson 2007). From a scientific perspective, it is not obvious how depression affects emotion. This is because depression appears to involve a primary problem in a different area, with mood, or prevailing affective tone. Mood, unlike emotion, is usually conceived of as a diffuse, slow-moving feeling state (e.g., Watson 2000). With these definitions in mind, the real question becomes: How do the persistent mood changes of depression influence ongoing emotional reactions to environmental stimuli? Fortunately, over the last 15 years, work drawing from affective science has begun to address this challenging issue, specifying both the contexts and the response systems in which depressed people exhibit aberrant emotional reactivity (Rottenberg 2005, Rottenberg & Bylsma 2014).

Over this period, scientists have conceived of several ways that emotions might be important to depression. One set of issues concerns the functional significance of emotional changes

for depression. This is a natural issue because it is commonly claimed that emotional reactions have important implications for adaptation and human functioning (Keltner & Kring 1998, Levenson 1999) and because depression is an episodic condition, with half or more of affected people experiencing multiple episodes (Monroe & Harkness 2011). The functional significance of emotion animates research questions, such as, how do changes in emotion relate to the waxing and waning of depression episodes? Might such changes in emotion be an antecedent risk factor that precedes episodes of depression? Or, alternatively, might such changes in emotion be a transitory correlate of the depression state that resolves upon remission? Or, finally, could such changes in emotion be an enduring consequence of depression that remains after depression (e.g., a scar)?

Finally, researchers have run with the insight that emotional reactions do not simply happen but that people try to change or alter their emotional impulses, distinguishing the emotion generation process from that of emotion regulation (Gross & Thompson 2007, Rottenberg & Gross 2003). Emotion regulation refers to people's attempts to control which emotions they have and how these emotions manifest (Gross 1998). The keen interest in emotion regulation has been especially strong in the clinical domain, where the hope has been that researchers might identify specific patterns of poor management of emotion that contribute to psychopathology, including depression (Kring & Sloan 2009, Rottenberg & Johnson 2007, Sheppes et al. 2015). Therefore, a major issue in the field of depression has been to consider how emotion regulation is altered by depression. This issue has obvious clinical application: Presumably, if we can determine how depression disrupts emotion regulation, the regulatory problem may be correctible with a therapy or intervention. In fact, explicit emotion-based therapies have been proposed for depression and are being evaluated (Campbell-Sills & Barlow 2007, Kovacs et al. 2006).

The Challenges of Studying Emotion

Given the urgency of figuring out how depression influences emotion, scientists have often barreled ahead. Although this enthusiasm is natural, it has led to the issue of premature and sometimes confusing claims. Gross & Jazaieri (2014, p. 387) keenly observe, "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."

Our urge for quick answers has collided with the fact that studying emotion is hard. From the origins of the field to recent times, researchers have despaired over the messiness of the emotion construct, even going so far as to question its very existence (e.g., Barrett 2006, Russell 2003). This is partly because emotions are multifaceted. Emotions involve changes in several different response domains, including cognition, behavior, and central and peripheral physiology. Notoriously, activity across these emotion response domains is only loosely coupled. When we observe that a person watching a horror movie reports fear but does not display any facial behavior indicating fear, or the reverse pattern of displaying but not reporting fear, we realize that an apparently simple judgment about whether fear is present is nonobvious (e.g., Mauss et al. 2005). Further complicating the issue, within each given response domain, there is no single gold standard metric for researchers to pull off the rack to stand in for emotion (Mauss & Robinson 2009, Sloan & Kring 2007). Instead, there are a variety of metrics. For instance, a researcher focusing on behavior could justify utilizing observer coding of facial expression, reaction times, or behavioral choices as emotion proxies. At a bare minimum, these complexities suggest that defining how depression affects emotion requires (*a*) assessing multiple domains and responses and (*b*) interpreting a complex pattern/profile of differences rather than a uniform and obvious signature.

Similarly, the area of emotion regulation provides an attractive, but illusive, target. There are many ways that emotion can be regulated (or dysregulated). These emotion-regulation strategies may be automatic and nonconscious or carefully planned. Furthermore, these emotion-regulation strategies are deployed in a dynamic fashion and are repeatedly adjusted as situations unfold (Gross & Thompson 2007). Because of the dynamic, often context-specific way that emotion is regulated, it can be quite challenging to simply assess emotion regulation and to confirm that regulatory efforts have occurred, let alone to document that such efforts have failed.

A stiff challenge in figuring out how depression influences emotional reactivity and emotion regulation is adequately representing the contextual factors that may influence both (Aldao 2013). In principle, there are an infinite number of contextual factors (e.g., cognitive, interpersonal, and biological) that may modify emotion and its regulation. For example, there may be important situational variations in how emotion regulation operates at home, at work, at play, with friends, alone, or in the laboratory. If so, we must represent these situational contexts to have an adequate account of how depression affects emotion regulation. For instance, it would be especially useful to corroborate observations across two classic research designs—laboratory experiments and field studies—which represent contrasting assessment contexts. Laboratory experiments, ideally, achieve high(er) internal validity by assessing emotion through standardized stimulus presentation and control over the environment, whereas field studies have strong external validity and should generalize better to everyday life contexts (e.g., Bylsma et al. 2011, Ebner-Priemer & Trull 2009). As described in the section titled Emotional Reactivity in Depression: Taking Stock and Moving Forward, corroboration of observations across designs and examination of emotion across multiple assessment contexts have been rare in depression research.

The Goals of This Review

There are tensions between wishing to quickly uncover how depression affects emotion and being mindful of obstacles to definitive conclusions. This review attempts to navigate these tensions. My goal is to provide a snapshot of the field that separates what we know from what we do not yet know. I take advantage of the latest developments in affective science, an often explicitly translational enterprise that seeks to bridge basic research on normative emotion functioning with applied research on clinical disorders (Kring & Sloan 2009, Rottenberg & Johnson 2007), including both theoretical advances and improved means to elicit and assess emotion inside and outside the laboratory (Coan & Allen 2007, Gross 2007). Consistent with affective science, I examine depression-related changes in emotion across multiple systems and contexts. Data focus primarily upon emotional experience, emotional behavior, and autonomic physiology (heart rate, electrodermal activity) (Lang et al. 1993) because these three systems have been historically important to the study of emotion and because space does not permit detailed consideration of all emotion-relevant systems. Similarly, although emotional reactivity and emotion regulation are the main foci, I foray into related areas (emotional memory, emotion knowledge, emotional intelligence, and attitudes toward emotion).

This article has two themes: (*a*) in limited arenas we have achieved some solid knowledge, and (*b*) outside of these arenas, depression has been difficult to characterize. In sorting out what we know from what we do not know, I dwell at times on seemingly conflicting findings and resist the temptation to sweep discrepancies under the rug. In fact, I explain how seemingly odd results can motivate key directions for future research, offering a consideration of specific strategies to expand the zone of what we really know about emotional reactivity and emotional regulation in depression. Ultimately, a clearer map of the ways in which emotional reactivity and emotional

regulation go awry is not merely an academic exercise but is necessary to harness the data of emotion toward better interventions for depression.

EMOTIONAL REACTIVITY IN DEPRESSION: WHAT WE KNOW

Emotion context insensitivity (ECI): an emotion that is insensitive to changes in emotional context

When I began to examine negative emotional reactivity in depression 20 years ago, affective science was relatively new, and the body of available work was rather modest. Moreover, relatively few theories were expressly directed at it. Based upon the robust clinical fact of anhedonia (Am. Psychiatr. Assoc. 2013, Costello 1972; for a review, see Pizzagalli 2014), one could predict that depression would reduce reactivity to positive emotion elicitors. However, expectations were not so clear concerning how depression would alter reactivity to negative emotion elicitors. From general emotion theory, one could adduce an intuitive matching prediction about mood-emotion interactions, such that clinically depressed moods would potentiate reactivity to negative emotional stimuli, on the premise that a background affective state primes reactivity to an acute foreground stimulus of matching valence (e.g., anxiety primes fear at a strange noise) (Rosenberg 1998). Similarly, cognitive theories of depression could be adapted to make similar priming predictions (Beck 1967). If depressed people are guided by negative cognitive structures called schema, which guide preferential processing to negative stimuli, then when negative stimuli are encountered in the environment, these negative schema should presumably enhance the accompanying emotional reactions.

Contra such expectations, a series of investigations starting from my time as a graduate student at Stanford in the early 1990s found no evidence of enhanced reactivity to negative material. Rather, a first experiment revealed that clinically depressed persons exhibited less differential reactivity to sad material and reported smaller increases in sadness than healthy controls when responses from the neutral and sad films were compared (Rottenberg et al. 2002b). A second study focused on the observable behavior of crying in response to a sad film. Given the common view among clinical commentators that depressed persons cry readily and intensely, this is one situation where we expected to see clear evidence of mood potentiation (Am. Psychiatr. Assoc. 2013; for a review, see Vingerhoets et al. 2007). Instead, there were no differences in the rate of observable crying behavior between depressed and nondepressed persons. Moreover, depressed criers were less reactive than healthy criers, showed smaller crying-related increases in their sadness and less evidence of autonomic arousal (Rottenberg et al. 2002a). Finally, in an important extension, we found reduced reactivity to sad stimuli in MDD even when the sad stimuli were constructed to be high in personal relevance (Rottenberg et al. 2005). These unexpected results were a first lesson in the need to be open-minded when studying depression and emotion.

Were these experiments anomalous? By 2008, enough laboratory work had accumulated to allow a meta-analysis of emotional reactivity in depression and to allow estimation of effect sizes. Indeed, consistent with our initial experiments, results from this meta-analysis of 19 laboratory studies revealed a pattern that we termed emotion context insensitivity (ECI) (Bylsma et al. 2008) in which MDD was characterized by reduced emotional reactivity to both positively and negatively valenced stimuli, with the reduction larger for positive stimuli ($d = -.53$) than for negative stimuli ($d = -.25$). Importantly, although many of our initial results were in the domain of emotion experience, the meta-analysis results revealed similar depression-related differences in emotional reactivity across three major emotion-response systems (self-reported experience, expressive behavior, and peripheral physiology).

Led by these data, we have offered our account of ECI to explain why MDD might involve a generalized loss of context-appropriate emotional reactivity to both positive and negative elicitors (Rottenberg & Bylsma 2014, Rottenberg & Cowden Hindash 2015). ECI was grounded in the evolutionary functions of mood as regulating effort and energy expenditure (Nesse 2000). By

this account, the evolved function of strong negative mood states, such as those seen in clinical depression, is to diminish action in risky, dangerous situations, where behavior is not paying off. Ordinarily, emotions function to alert us to important environmental changes and motivate us to deal with these changes (Scherer 2009). However, clinically depressed mood states override emotion's normal dynamic function, invoking broad reductions in motivated action, including reductions in emotional reactivity to ongoing environmental stimuli (Rottenberg et al. 2005); promoting an internal cognitive focus (Andrews & Thomson 2009); and diminishing the ability for incentives to alter behavior (Treadway et al. 2012).

Although ECI may be a modern term that helps scientists address aspects of depression and emotion, it should be underscored that the underlying phenomenon has been with humanity for ages. For many centuries, people struggling with depression have complained that their emotional world is undifferentiated, flat, dull, and empty (Jackson 1986). Likewise, a multitude of behavioral observations from inpatient settings document that depression can diminish motivated activity to the point of immobility, even catatonia in severe cases (Starkstein et al. 1996). Beyond the field of emotion, a number of commentators have characterized depression as a syndrome marked by inflexibility and stereotypy in cognition, behavior, and physiology (for a review, see Kashdan & Rottenberg 2010). ECI is in keeping with such characterizations.

Since the meta-analysis, additional observations have emerged to make us more confident that the ECI pattern should be grouped into the "what we know" category. Several independently studied phenomena, traveling by different names, fit with ECI. For example, work by Kuppens and colleagues (2010) has focused on the construct of emotional inertia. Emotional inertia pertains to the degree to which emotions exhibit carryover, or resistance to change, and it is operationalized via autocorrelation modeling in which the current emotional state is predicted from the previous emotional state. Reporting and displaying a high degree of carryover across temporal contexts can be seen as consistent with ECI. Empirically, emotional inertia has repeatedly been found to be elevated among people who have symptoms of depression (Brose et al. 2015; Koval et al. 2012, 2013; Kuppens et al. 2010) and is negatively correlated with measures of well-being across many studies (Houben et al. 2015). Moreover, high inertia at one point in time also predicts greater depression at a future time point (Kuppens et al. 2012).

A second ECI-related construct is emotion differentiation. Emotion differentiation refers to the extent to which different emotional responses are intercorrelated. For example, if one negative emotion, such as anger, is reported at a given moment, will other negative emotions be concurrently reported? Depressed persons have been shown to exhibit less emotion differentiation than controls, reporting a higher intercorrelation among separate negative emotions at any given time point (Demiralp et al. 2012). Thus, low emotional differentiation provides another vantage point onto the flattened emotional landscape of ECI.

A third related construct is emotion network density. Emotion network density regards the extent to which a person's future emotional responses would be predictable from other previous emotional responses in related categories. For example, does a person's level of current sadness predict his or her future levels of anger or fear? Depressed persons have been found to exhibit greater emotion network density than controls, particularly for negative emotional states (Pe et al. 2015). Again, the higher degree of predictability of negative emotions across time and context would appear to be consonant with ECI.

Finally, our meta-analysis did not address central nervous system proxies of emotion. Fortunately, since our meta-analyses, additional convergent evidence has emerged in that response domain. For example, work focusing on event-related potentials (ERP) recorded from an electroencephalogram during behavioral tasks has found that, during the processing of a variety of emotional stimuli, persons with MDD or elevated depressive symptoms, exhibit reductions in two

ERP components, specifically, the late positive potential (a measure of sustained processing of motivationally salient stimuli) and reward positivity (an index of reactivity to receipt of reward) (see Proudfit et al. 2015). Reductions in these ERP components have been interpreted as reflecting broad emotional disengagement and deficits in reward processing.

The ECI pattern also raises other questions about mood-emotion interactions in depression. How do we square reduced phasic reactivity to negative emotion elicitors with the chronic elevation in negative mood that is so characteristic of depression? One crucial set of recent findings indicates that depressed persons tend to show poor recovery from negative emotion elicitors across a variety of response systems (Salomon et al. 2009, Siegle et al. 2002). This poor recovery after a negative emotional challenge may reflect the operation of a number of processes, such as negative repetitive thought (Segerstrom et al. 2000) and/or weakened homeostatic mechanisms (Rottenberg et al. 2003), all of which render depressed persons liable to chronically elevated negative affect across contexts (Rottenberg et al. 2002b). In sum, the phenomenon of ECI in depression supports drawing a distinction between moods and emotions, challenges the assumption that moods invariably potentiate like valenced emotions, and suggests that moods and emotions may be governed by different processes (Rottenberg 2005).

For all the study of ECI, it is important to underscore that there remains much about it that we still do not fully understand. For example, the ECI pattern may be reliable, but it is not always observed, and we do not have a strong sense of what determines its boundary conditions. Second, the relationship between ECI and the clinical course of depression remains unclear. Although some early studies have been conducted, and it is plausible that ECI in various response systems could be a risk factor for future depression (e.g., Peeters et al. 2010), it cannot yet be determined whether ECI represents an antecedent risk factor for depression, a state-like feature that resolves with episodes (e.g., Salomon et al. 2013), or a scar, a consequence of depression. Therefore, the possible functional significance of ECI and ECI's status as a risk factor for depression still require clarification. Finally, inflexibility is increasingly seen as a central aspect of depression (Kashdan & Rottenberg 2010). However, research has yet to sort out the connections between ECI and other manifestations of inflexibility, including cognitive patterns such as rumination (Koval et al. 2012), physiological patterns such as diminished cardiac vagal control (Rottenberg 2007), or behavioral patterns such as avoidance (Trew 2011). Such integrative work is needed to build and test larger theoretical models of depression.

EMOTIONAL REACTIVITY IN DEPRESSION: WHAT WE DO NOT KNOW

What we do not know about emotional reactivity in depression is substantial. For some issues, there are initial findings that the field seeks to build upon. In other areas, our knowledge is minimal. One area with promising initial findings regards how depression affects emotional reactivity outside of the laboratory. This body of work in clinical samples is small and somewhat heterogeneous. However, one replicated finding is that depressed persons exhibit greater affective instability, when instability is measured as the magnitude of point-to-point variations in affect with assessment points ~90 min apart (Peeters et al. 2006; Thompson et al. 2011, 2012). Importantly, a recent meta-analysis confirmed that, across a range of clinical and nonclinical samples, affective instability, particularly for negative affective states, was associated with depression symptoms (Houben et al. 2015). Interestingly, there is also preliminary evidence that affective instability outside the lab predicts a worse depression course (Wichers et al. 2010) and that affective instability goes down with successful depression treatment (Silk et al. 2011). On the surface, these findings appear at odds with ECI.

Mood-brightening effect: depressed persons report larger decreases in negative affect after positively appraised life events than do nondepressed people

A second curious recurring finding in emotion studies regards reactivity to everyday life's pleasant events. In several investigations, depressed persons exhibit larger decreases in negative affect after a positively appraised real-life event than do nondepressed people (Peeters et al. 2003, Thompson et al. 2012). This mood-brightening effect is striking because it is counterintuitive and is not predicted by any major theory of emotional functioning in depression. Although it should be granted that pleasant events occur relatively infrequently in depressed peoples' lives and do not completely relieve dysphoria, mood brightening has now been found in participants with major and minor forms of depression, as well as across different methods for assessing affective experience (Bylsma et al. 2011). Assuming that depression-related mood brightening is reliable, it suggests another boundary condition for ECI.

There are several areas where we lack systematic knowledge. One area concerns potential variables that might moderate the effects of depression on emotional reactivity. Our meta-analyses revealed considerable heterogeneity among effect sizes (Bylsma et al. 2008), which suggested the presence of moderators. However, it was not possible for us to test for moderation of any variable because reporting practices for several potential variables were too haphazard to constitute a viable analysis. More generally, the field of depression has not focused on moderators of emotion for both theoretical and practical reasons. Most studies of diagnosed samples are designed to examine depression as a whole. Depression is typically analyzed as a between group variable, and partly because of the expense and labor involved to create clinical samples, most studies are not powered to test moderators. At present, the field has only minimally evaluated potential moderators from demographic variables, such as gender or race, to clinical variables, such as depression severity, episode history, or subtype. At present, we have only glimmerings from individual studies that greater episode severity or number may be implicated in reduced emotional reactivity (Allen et al. 1999, Forbes et al. 2005, Vaidyanathan et al. 2014).

Another set of gaps regards how diagnostic comorbidity might influence emotional reactivity in depression. Given that depression typically co-occurs with other clinical problems, this gap is notable. Yet remarkably few studies of emotional reactivity consider samples of pure noncomorbid depression. Certainly, creating such pure samples is difficult and labor intensive. Even if it were possible to constitute such samples, they could be faulted for being artificial and unrepresentative of how depression usually manifests. Furthermore, studies of emotion in depression have not generally employed a psychiatric control group, which is another potential means to isolate the effects of depression (Shankman et al. 2013). Thus, it is difficult to characterize whether effects observed in the emotion literature are unique to depression or might be carried by co-occurring conditions. Interestingly, some work has examined patterns of comorbidity to indirectly assess the unique impact of depression on emotional reactivity. Specifically, emotional reactivity in anxiety that co-presents with depression has been compared to emotional reactivity in anxiety alone (Taylor-Clift et al. 2011, Yoon & Joormann 2012). Across studies, an anxiety/depression group exhibited the ECI pattern that is elsewhere characteristic of depression, whereas the anxiety alone group did not exhibit ECI. These data suggest that the presence of depression might systematically alter the reactivity characteristics of other disorders (Taylor-Clift et al. 2011, Yoon & Joormann 2012).

Finally, there is a major gap concerning the effects of psychological or pharmacological treatment on emotional reactivity in our three systems of response. Specifically, we lack comprehensive longitudinal studies that test reactivity to emotion-generative stimuli pre- and posttreatment, which could elucidate both how emotional reactivity covaries with the depressed state and what changes in emotional reactivity are introduced via treatment. This gap is unfortunate because we know that treatments have effects in the response domains of interest. For example, antidepressants alter resting autonomic state (e.g., Balogh et al. 1993). By contrast, many neuroimaging studies have used designs in which participants engage in some form of emotion processing at multiple

time points, including pre- and posttreatment. Although this literature defies any easy summary given the complexities of mapping specific brain areas to emotional reactivity, most commentators interpret this body of neuroimaging findings to indicate treatment-related normalization of brain areas associated with emotional reactivity (for example, psychotherapy improving reward-related brain function) (see Dichter et al. 2009; also see meta-analysis by Delaveau et al. 2011). In a rare pre and post design using the startle response, depressed persons who viewed affective pictures before and after antidepressant treatment continued to evidence persistent depression-related differences in the emotion-modulated startle reflex after the treatment (reduced emotion-modulated startled relative to controls) (Dichter et al. 2004). By contrast, Wichers and colleagues (2009) examined self-reported negative affective responses to everyday life stressors using ecological momentary assessment before and after antidepressant treatment and found treatment effects: Six weeks of antidepressant treatment reduced stress sensitivity.

EMOTIONAL REACTIVITY IN DEPRESSION: TAKING STOCK AND MOVING FORWARD

At this stage of our knowledge, there are a number of puzzles and tensions regarding existing findings about emotional reactivity in depression. These issues invite informed speculation to guide further work. One issue concerns when ECI is (and is not) observed. For one, the ECI pattern appears more likely and robust in laboratory contexts rather than in everyday life contexts. Laboratory contexts may be sensitive to ECI for several reasons. Laboratory contexts typically value standardization and feature normative, generic emotional stimuli, such as pictures or short films, rather than individualized, idiographic stimuli (Rottenberg et al. 2005). What laboratory studies may be telling us is that depression systematically reduces reactivity to generic emotion elicitors (i.e., stimuli that affect the average nondepressed person). This interpretation leaves open the possibility that depressed persons retain reactivity to situations with idiosyncratic meaning, which is almost the very definition of everyday life (Barlow & Nock 2009). Alternatively, affective instability in the everyday lives of depressed persons could also reflect systematic differences in the situations that depressed people encounter, such as more unpredictable exposures to negative emotion eliciting situations (e.g., verbal abuse by a workplace supervisor). Recently, Houben and colleagues (2015) argued that instability and inertia are not necessarily incompatible because of the different ways the two measures are derived; larger shifts in affect (instability) can be superimposed upon higher carryover from point-to-point inertia.

Similarly, it is unclear whether the mood-brightening effect in field studies reflects a true discrepancy from laboratory findings of ECI or whether it reflects differences between laboratory and ambulatory assessments of emotion. Again, unlike laboratory studies, which generally use objectively created positive stimuli, field studies do not use objective emotional stimuli. In field studies, positive (or negative) events are defined by the participant's appraisal of said events (i.e., events that were judged by the participant to be pleasant are coded as positive events). What the mood-brightening effect may be telling us is that, in those relatively infrequent times that depressed people perceive pleasant events to be occurring, they can be reactive to said events. This explanation seems credible in light of the work implicating appraisal factors in mood brightening (e.g., everyday life pleasant events interrupt rumination) (Takano et al. 2013). A related consideration is that depressed persons' observed reactivity to everyday life's pleasant events could be due to the relative rarity of such events (a contrast effect). Moreover, scientists might also observe enhanced positive reactivity if depressed persons had higher thresholds to appraise life events as pleasant, meaning that the life events appraised by depressed people as positive were objectively more impactful than the life events so appraised by nondepressed people. To begin to sort out

the role of subjective appraisals versus objective event characteristics, future field studies could examine emotional reactivity to staged standardized positive events (e.g., a compliment by a stranger). One could predict that depressed people would be less reactive than nondepressed persons to standardized everyday life positive events, similar to the observations seen with normative stimuli in laboratory designs.

Another key issue regarding when ECI is (and is not observed) concerns the timescale of the assessments. In the laboratory, emotional reactivity is almost invariably assessed in the short term of seconds or minutes; importantly this is a timescale we traditionally associate with emotions (Lang et al. 1993). In this shorter-term window, ECI-like effects are often observed. By contrast in field studies, the assessment windows are typically much longer, ranging from hours to even days, and over these longer windows depressed persons sometimes exhibit affective instability. Conceivably, the differences between laboratory versus field study results could reflect the operation of processes operating at different timescales. Affective instability outside the laboratory could be reflecting processes that are observable only at longer windows, such as disturbances in circadian rhythms (Peeters et al. 2006) or chaotic interpersonal behaviors (Russell et al. 2007). In this respect, affective instability and inertia could be seen as not as fundamentally incompatible but as different sides of the same coin. A recent commentary from the perspective of dynamical systems theory suggested that we might consider the juxtaposition of instability and inertia as a kind of punctuated ECI in which the overall system is fragile and in which minor perturbations can trigger a critical transition, which lurches the system to another stable state that is resistant to change (e.g., a long lasting elevation in negative affect) (Wichers et al. 2015). Clearly, research that systematically examines emotional functioning in depression at different levels of temporal resolution is needed to evaluate this possibility.

The gaps in research on emotional reactivity in depression are many. In this Section, I have offered informed speculation concerning what may reside in some of the gaps. The next steps are, of course, to perform the empirical tests. Among the most pressing needs is a stronger representation of how contextual factors influence emotional reactivity in depression. Such contextual factors include systematic examination of situational factors, such as home versus work and solitary versus social (Bylsma et al. 2011). One challenge in this work is practical, accumulating sufficient numbers of observations of emotional reactivity in enough people across enough contexts to enable systematic contrasts. In other fields, close attention to situational contexts has long been routine, as in studies of cardiovascular functioning assessing reactivity in both laboratory and ambulatory contexts to understand the exact conditions under which exaggerated reactivity might harm the heart (Kamarck et al. 2003) or work in schizophrenia documenting the effects of varying social contexts in triggering psychotic symptoms (Myin-Germeys et al. 2001). Suffice it to say, the field of depression needs more intensive multimethod studies assessing emotional functioning across multiple contexts (see Heller et al. 2015 for a model study).

Second, there is also need for improved theory concerning emotional reactivity in depression. Emotion is a major domain of functioning akin to biology, development, social functioning, etc. Yet when compared to these other domains, emotion-based theories of depression are less well developed. In the cognitive domain, by contrast, there are multiple mature theories of depression that elaborate upon ultimate origins, identify responsible proximal mechanisms, and generate specific predictions (e.g., Abramson et al. 1989, Beck 1967). Importantly, these cognitive theories have spawned a powerful therapy, which is widely viewed as a partial proof of the theory (Beck 2005). Unfortunately, work on emotional reactivity in depression has been slower to receive strong theoretical guidance, despite the intense theoretical ferment within the study of emotion. For instance, ECI gradually arose as a theoretical account from a series of unexpected empirical observations. As theories of emotional reactivity in depression mature, it is important for them to identify the

mechanisms responsible for depression-related differences in emotion. To yield clinical payoffs, we must feed forward each advance in understanding of emotion-based mechanisms to the design of more effective interventions.

EMOTION REGULATION IN DEPRESSION: WHAT WE KNOW

Although the generation of an emotional response is certainly related to efforts to regulate that response, scientists have increasingly found it useful to distinguish emotion regulation from emotional reactivity (Gross & Thompson 2007). Depression researchers may find the regulation-reactivity distinction helpful for delineating pathways into mood disturbance. An outcome like chronically low mood could be affected via emotion-regulation routes (a person's response to low mood is to withdraw from rewards) and/or via emotion-generation routes (a person faces the slow, wasting death of a loved one). Thus, researchers interested in emotion regulation in depression have been motivated to identify specific emotion-regulation problems that could contribute to its onset, maintenance, or recurrence (Hofmann et al. 2012). Like emotion generation, the domain of emotion regulation is complex and multifaceted. A number of potential emotion-regulatory problems might contribute to depression, including an inadequate repertory of emotion-regulatory strategies, selection of the wrong emotion-regulation strategies, poor implementation of emotion-regulation strategies, or problematic emotion-regulatory goals (Bonanno & Burton 2013, Kashdan & Rottenberg 2010).

One point of departure is the perspective of the depressed and depression-vulnerable themselves. A perceived inability to control moods and emotions is among the chief clinical complaints of depressed people, including reporting constant sadness, feeling chronically numb and cut off from emotions, and/or not understanding feelings. Likewise on questionnaire measures, depression is associated with reporting large, uncontrollable responses to stress (high neuroticism) (Morris et al. 2009), appraising emotions as threatening (Campbell-Sills et al. 2006), and having lower expectations of self-regulatory success (Brockmeyer et al. 2012, Ehring et al. 2008).

On self-report inventories of emotion-regulation strategies, depressed persons also endorse using strategies that are different from those used by healthy persons. At the broadest level, vulnerability to depression has been associated with a greater endorsement of emotion-regulation strategies that experts consider maladaptive (Kovacs et al. 2009). Interestingly, depression was not associated with reduced endorsement of adaptive emotion-regulation strategies (Kovacs et al. 2009), suggesting that vulnerable persons are at least aware of potentially useful emotion-regulation strategies (Aldao et al. 2010).

Research on individual emotion-regulation strategies has clustered on a few strategies that might be depressogenic. The most studied maladaptive emotion-regulation strategy is rumination: Rumination is known to be associated with prolonged and deepened sad mood in laboratory and field studies (Nolen-Hoeksema et al. 2008). A large volume of investigations finds associations between rumination and depression symptoms or diagnosis (Nolen-Hoeksema et al. 2008). There is also evidence that rumination is observed outside of depression episodes. Ehring et al. (2008), for example, found that formerly depressed participants reported more rumination but did not differ from never-depressed participants in their reported use of reappraisal. Interestingly, although there is a clear association between rumination and depression, depressed people are not necessarily aware of rumination's deleterious effects on mood. Indeed, vulnerable people typically believe that they receive benefits, such as increased insight, from using this strategy.

Another more-studied emotion-regulation strategy is reappraisal, a cognitive form of emotion regulation, which is considered to be adaptive by most researchers (Gross 1998). Studies with clinical and nonclinical samples associate depression with less frequent habitual use of

reappraisal (D'Avanzato et al. 2013, Garnefski & Kraaij 2006, Joormann & Gotlib 2010). Other emotion-regulation strategies, such as acceptance, avoidance, and suppression, have also been evaluated to a more modest degree, and at least some evidence indicates that depression is associated with lower endorsement of acceptance (Shallcross et al. 2010), greater endorsement of avoidance (Tull et al. 2004), and greater endorsement of suppression (D'Avanzato et al. 2013).

Only a subset of emotion-regulation strategies has been examined with respect to depression. On the one hand, it seems premature to focus on a small number of emotion-regulation strategies, given the immense pool of potentially relevant emotion-regulation strategies in the larger domain. On the other hand, a recent meta-analysis examining the relationship between several emotion-regulation strategies and psychopathology provides some support for focusing on some strategies more than others. Based on the magnitude of the average effect sizes, Aldao and colleagues (2010) concluded that rumination, suppression, avoidance, and problem solving were more strongly related to mental health than were acceptance and reappraisal. Furthermore, in connection with depression and anxiety, larger effect sizes were observed for rumination and avoidance than for acceptance and reappraisal (Aldao et al. 2010). Of course, effect sizes do not speak completely for themselves, but it is notable that major therapeutic approaches, such as acceptance-based treatments and cognitive-behavioral therapy (Beck 2005, Hayes et al. 1999), were associated with strategies with smaller observed effect sizes (acceptance and reappraisal) (Aldao et al. 2010).

Although this work with self-report instruments is an important first step, self-report measures provide only one vantage point on to emotion regulation—one that has important limitations. Self-reports of emotion regulation are convenient, but such reports may not be veridical for several reasons. One concern is that people with depression may endorse poor or faulty emotion regulation because they are generally biased toward negative self-evaluation and tend to report themselves unfavorably over many dimensions (Coyne & Gotlib 1983). Error is also a concern because many aspects of emotion regulation are automatic and may occur outside of conscious awareness (Gross & Thompson 2007). Thus, humans, irrespective of psychopathology, may often lack sufficient insight to accurately self-report on their emotion-regulation strategies (Robinson & Clore 2002). Moreover, transitory factors, such as mood state, likely introduce bias and error into self-assessments of emotion regulation. Finally, in at least some cases, there is the issue of circularity in that the content of items drawn from measures of emotion regulation overlap considerably with the symptoms of depression (e.g., worry and anxiety, rumination and depression) (Treynor et al. 2003). The limitations of self-report assessments regarding emotion regulation are considerable.

It is often the case that depression researchers ultimately want to make inferences about emotion-regulatory skills, working under the assumption that psychopathology involves a reduced ability to successfully implement emotion-regulation strategies. Emotion-regulatory skills are no doubt an important aspect of human functioning and an appealing intervention target (Berking et al. 2008b). The small literature employing self-report measures of emotion-regulatory ability does indeed sometimes find that people who appraise their emotion-regulatory skill level as low are more likely to develop depression symptoms (Berking et al. 2014; but see Berking et al. 2008a). However, the jury is still out as to whether self-report measures of emotion-regulation ability truly tap into performance, and so the burden of proof must rest on those making the claim. Assuming that self-reports of emotion-regulatory ability measure ability would be akin to assuming that self-reports of intelligence measure intelligence (and to dispensing with an IQ test). By the same token, self-reports of emotion-regulation strategies cannot provide useful information about emotion-regulation skills. A person could consistently choose poor emotion-regulation strategies and yet have good general skill at implementing emotion-regulatory strategies, and vice versa. For these reasons, laboratory performance-based tests are the preferred means to assess emotion-regulatory skill in depression.

EMOTION REGULATION IN DEPRESSION: WHAT WE DO NOT KNOW

To state the conclusion at the outset, the field has not gotten far in demonstrating that depression is associated with worse emotion-regulatory skill. Depression-related differences on laboratory-based tests of emotion-regulation skill are modest and inconsistent. Indeed, it is relatively uncommon for depressed persons to perform more poorly than controls on performance-based emotion-regulation tasks (Greening et al. 2014), and when differences are observed, the effect sizes are modest (Kovacs et al. 2015) or are observed only for portions of a protocol (Joormann et al. 2007). The closest thing to a consistent finding is support in two of three studies testing the hypothesis that depressed people are less able to use positive autobiographical memories to alleviate sadness than nondepressed people (Joormann et al. 2007, Kovacs et al. 2015; but see Werner-Seidler & Moulds 2012). There are numerous published null findings in which depressed and nondepressed persons were instructed to repair sadness with cognitive reappraisal or distraction, with no differences observed between controls and depressed or remitted MDD participants in both neuroimaging (Beauregard et al. 2006, Erk et al. 2010, Kanske et al. 2012, Perlman et al. 2012) and nonimaging studies (Ehring et al. 2010, Joormann et al. 2007). Given the prevailing bias against publication of null results, it is likely that additional negative unpublished studies have been performed (Easterbrook et al. 1991). Finally, and also inconsistent with a skill deficit, in at least one study depressed people evidenced a greater improvement in their negative affective state using distraction than did nondepressed people (Smoski et al. 2014).

If depressed people perceive their emotion-regulation skills to be inferior, why is there so little laboratory evidence of emotion-regulation skill deficits? One explanation is that depressed people may subjectively perceive their emotion-regulation abilities as being worse than they actually are and that, despite expectations of poor performance, depressed people are not actually worse in their emotion-regulatory abilities. Consistent with this possibility, one investigation found that, even as depressed persons reported that they perceived it as more difficult to suppress their sadness, they achieved the same emotion-regulation outcome, reporting comparable reductions in sadness as did control participants (Beauregard et al. 2006). Alternatively, depressed persons may demonstrate less emotion-regulation skill outside of the laboratory, consistent with their skill perceptions, but in the laboratory, they can adequately use functional strategies when explicitly instructed. Consistent with this explanation, Ehring et al. (2010) reported that, even though formerly depressed participants were less likely to endorse using reappraisal in everyday life, these participants did not differ from the control group in their ability to effectively use reappraisal instructions in the laboratory. Potentially, an interesting parallel comes from studies of cognition, where the usually widespread memory problems in depression are not observed under specific laboratory conditions that control attention (Hertel 1994).

It also remains possible that laboratory studies have not detected significant emotion-regulation skill deficits in depression owing to assessment limitations. This cannot be ruled out because only a mere fraction of the many emotions, emotion-regulation strategies, and regulatory contexts have been tested in laboratory studies. Most investigations, by far, have focused on the regulation of sadness. A rare exception that examined anger found no depression-related differences across three emotion-regulation strategies (reappraisal, acceptance, no instruction) (Ellis et al. 2013). The bottom line is that there are several viable explanations for the weakness of experimental evidence for depression-related impairment of emotion-regulatory skills, and further work is required to determine which of these explanations is correct.

A final caveat concerns caution in interpreting depression-related differences in emotion-regulatory performance when these are observed. The laboratory allows control of many factors, but control of motivational factors is difficult, and typically motivational factors have been ignored.

This is a thorny issue because most emotion-regulation strategies leave little in the way of behavioral residue, and thus, there is no way to monitor how hard a person is working to implement the strategy (is the participant trying to reappraise?) independent from the outcome itself (was negative emotion reduced?). For this reason, it is difficult to confirm that depression-related differences on laboratory tasks reflect skill differences rather than motivational differences. Importantly, a recent investigation uncovered behavioral evidence that depressed people were less motivated to downregulate sadness than nondepressed people (Millgram et al. 2015). If this finding is borne out in future work, researchers will have to find ways to construct their skill-based emotion-regulation tasks to allow parsing of skill deficits from motivational deficits.

EMOTION REGULATION IN DEPRESSION: TAKING STOCK AND MOVING FORWARD

As scientists pursue emotion regulation in depression, there has been a yawning gap between what has been reached for and what has been grasped. It has become routine to state that depression is a disorder of emotion regulation, yet the field still lacks a rigorous, comprehensive description of what exactly is dysregulated in this condition. On the positive side, some points of departure have been established for creating such a description: Depressed people (*a*) report using maladaptive emotion-regulation strategies to a considerable extent, (*b*) perceive themselves to be inferior in their ability to self-regulate emotion, and (*c*) show consistent differences on a handful of specific maladaptive emotion-regulation strategies. For a few of these strategies, such as rumination, knowledge is more extensive, and research has established a meaningful nomological network. Joormann and coworkers (Joormann 2010, Joormann & Gotlib 2010), for example, have conducted an impressive research program considering the cognitive processes underlying rumination, tying rumination to depression-related executive functioning deficits and specifically to difficulties in removing irrelevant negative material from working memory. Furthermore, with select strategies like rumination, work has illuminated how and why this emotion-regulation strategy might be depressogenic. For example, the late Susan Nolen-Hoeksema and colleagues (2008) explained how rumination may foster depression by undercutting and displacing more active emotion-regulation strategies, such as problem solving.

To advance beyond these points of departure, four directions are critical. First, we need to move away from the historic focus on the utilization of individual emotion-regulation strategies toward a more dynamic view of self-regulation (Aldao et al. 2015, Sheppes et al. 2015, Trull et al. 2015). Not only is it difficult to explain a large portion of depression risk as a function of individual emotion-regulation strategies, but there is growing evidence for the idea that individual emotion-regulation strategies have varied consequences rather than uniform ones. Recent reviews of the importance of context have argued that strategies considered to be adaptive are not necessarily good, nor are maladaptive strategies necessarily bad (Aldao 2013, Bonanno & Burton 2013). Empirically, cognitive reappraisal does not have invariant positive effects (Troy et al. 2013), but it may help or hurt depending on the situation. Likewise, whether one suppresses or expresses facially expressive behavior appears less important than the ability to flexibly express or suppress emotional expression as demanded by the situational context (Bonanno et al. 2004). In other words, whether or not an emotion-regulatory strategy leads to a good outcome depends on it being deployed flexibly in the right place, at the right time, and in the right way (Aldao et al. 2015).

This more dynamic approach to emotion regulation views self-regulation as a sequence of environmental transactions (Aldao et al. 2015, Sheppes et al. 2015), asking a different question at each stage of the sequence. Initially, what is the range of emotion-regulation strategies that a person brings to the situation? Presumably, limitations in a person's repertory of

emotion-regulation strategies could increase liability to depression (Dixon-Gordon et al. 2015b). How does a person choose which strategy to use in a specific situation? Depression could involve breakdowns in how people perceive situations or how situations are matched to available regulatory responses. How does a person monitor the success of an initial regulatory response and modify the response if it is not effective? Depression, conceivably, may diminish people's ability to modulate regulatory efforts. And, going full circle, how does the success or failure of prior regulatory efforts shape how a person self-regulates in the next situation? Depression may involve problems in learning from prior regulatory efforts. Although an emphasis on emotion-regulation flexibility is relatively new in depression, it is promising because greater regulatory flexibility has already been shown to predict better adaptation (Bonanno et al. 2004, Coifman & Bonanno 2010, Waugh et al. 2011) and because evidence elsewhere shows that depression involves deficits in psychological flexibility (e.g., Kashdan & Rottenberg 2010).

Affect valuations: differences in the values that people attach to different affective states

With this in mind, a second key direction for future work on emotional regulation in depression is to identify the regulatory contexts that are most important to the disorder. This is a challenge because a large number of contexts are potentially relevant in the transactional approach, and only some of these can be highlighted here (for a review, see Aldao 2013). One key context for regulation is affective. In the emotional context, both the intensity and type of emotion are what typically precipitate a need for emotion regulation, and the exact nature of the emotional context may also shape which regulation strategies are selected (Dixon-Gordon et al. 2015a, Sheppes et al. 2011). At high intensities of negative emotion, for example, people are more likely to choose avoidance as an emotion-regulation strategy (Sheppes 2014), a strategy particularly germane to depression (Trew 2011). A second context for emotion regulation is the other emotion-regulation strategies that are available in a person's repertory. One investigation found that adaptive emotion strategies were only protective against psychiatric symptoms for people who tended to also endorse using maladaptive strategies (Aldao & Nolen-Hoeksema 2012a). This finding was interpreted to mean that the availability of adaptive strategies can buffer the deleterious effects of maladaptive regulation strategies (i.e., a compensatory process) (Aldao & Nolen-Hoeksema 2012a). Finally, different situations can be presumed to pull for different forms of emotion regulation. To date, remarkably little is known thus far concerning how fluctuating situational contexts might alter spontaneous generation of emotion-regulation strategies (Aldao & Nolen-Hoeksema 2012b). Therefore, examining how emotion regulation might shift (or might be stable) across situational contexts, such as across field settings and laboratory assessments (Yaroslavsky et al. 2016), is a major area of opportunity for depression studies.

A third key future direction for the study of emotion regulation in depression is to consider emotion-regulatory goals. Historically, researchers have not focused on emotion-regulatory goals in depression, perhaps assuming that depressed persons are as motivated to maximize pleasure and minimize pain as are nondepressed persons. However, this assumption may be unwise as we learn more about systematic individual differences and the value people attach to different affective states, and that these differences in affect valuation are linked to depression (Millgram et al. 2015, Tsai et al. 2006). Intuitively, one might expect that stronger valuations of positive affective states protect against depression, yet empirical indications are for the inverse relationship (Tsai et al. 2006). In fact, higher valuations placed on happiness have been associated with a depression diagnosis and its symptoms (Ford et al. 2014) as well as diminished well-being (Mauss et al. 2011). Such findings suggest that emotion-regulatory goals may be different from other goals, like mastering the piano, in which a stronger motivation to pursue the goal generates behaviors that increase the probability of achieving the goal. Thus, paradoxically, diminishing unduly high affective valuations may prove to have therapeutic value for depression.

Finally, most laboratory and field studies examining emotion-regulatory outcomes have focused on short-term affective outcomes (i.e., reduced negative affect, increased positive affect). It is important for future work on emotion regulation in depression to broaden outcome measures beyond the short-term feeling state. This is not only because emotion-regulation motives go beyond the pursuit of pleasure and the avoidance of pain (Tamir 2009) but also because nonhedonic outcomes may be just as critical for understanding adaptation and risk for psychopathology. For instance, it is important to detail how specific forms of emotion regulation might be predictive of downstream behaviors that maintain psychopathology (Christensen & Aldao 2015). For example, avoidance behavior is associated with depression and helps maintain it (Jacobson et al. 2001). Thus, it would be useful to examine what emotion-regulatory strategies are predictive of subsequent behavioral avoidance. It would be similarly helpful to investigate cases in which emotion-regulatory strategies predict behaviors that disrupt depression. For example, an investigation of two different forms of rumination (concrete versus abstract) found that depressed participants who engaged in the concrete form of rumination exhibited improvement in their subsequent problem-solving behavior (Watkins & Moulds 2005).

CONCLUDING COMMENTS: TOWARD INTERVENTION

This article has focused upon distinguishing between what we know and do not know about emotions in depression. A close inspection of the current state of the field indicates that there is much that we still do not know, and there is considerable cause for humility. Humility is not a moral stance, however. This humility is ultimately practical. As we apply the theories and methods of affective science to depression, being mindful of the challenges of studying emotion is more likely to result in work that stands the test of time. Starting off from our confident departure points is the only sound way to begin to create a detailed, contextually specific map of how emotional reactivity and emotional regulation go awry in depression. Realistically, such a map is the best means to harness the data from emotion to develop more powerful interventions, which ultimately curtail the worldwide burden of depression.

At a moment where the ground is shaking in the world of diagnosis and intervention, a firm place to stand is needed now more than ever. A detailed map of how and where emotion goes awry in mood disorders is sorely needed, whether one wishes to target traditional single diagnoses, like depression for intervention, or supports a move toward transdiagnostic diagnostic targets, such as those represented by the Research Domain Criteria initiative (Insel et al. 2010). A sound map is needed whether the goal is to optimize interventions that explicitly target emotion regulation (Campbell-Sills & Barlow 2007, Kovacs et al. 2006), to reconfigure traditional interventions like cognitive behavioral therapy so they better target emotion regulation (Berking et al. 2013), or to devise entirely new interventions that alter emotional reactivity or regulation in this devastating mental health condition.

SUMMARY POINTS

1. In the last 20 years, emotional dysfunction has been increasingly seen as central to depression.
2. The urgency of documenting emotional deficits in depression has tempted investigators to issue premature declarations and to sometimes overlook theoretical and methodological challenges entailed in studying emotion.

3. Although ECI is a key finding in depression, the field still needs a stronger representation of how contextual factors influence emotional reactivity.
4. Vulnerability to depression has been associated with a greater endorsement of several emotion-regulation strategies that experts consider maladaptive.
5. Depression-related differences on laboratory-based tests of emotion-regulation skills are surprisingly modest and inconsistent.
6. The field will profit from application of dynamic models of emotion regulation to depression.
7. A detailed, contextually specific map of how emotional reactivity and emotional regulation go awry in depression is ultimately needed to develop more powerful interventions to curtail the worldwide burden of depression.

FUTURE ISSUES

1. To better understand ECI in depression, additional research is needed to ascertain its boundary conditions, relationship to the clinical course of depression, and connections with other manifestations of inflexibility.
2. Systematic research is needed on potential variables that might moderate the effects of depression on emotional reactivity.
3. Work on emotional reactivity in depression would profit from inclusion of a stronger representation of situational factors.
4. Future work using dynamic models of emotion regulation could clarify exactly how depression is a condition of inflexible emotion regulation.
5. Future work on emotional regulation in depression could identify the regulatory contexts that are most critical to the disorder.
6. Work on emotion regulation in depression should broaden the outcome measures that are used to judge regulatory success or failure beyond short-term feeling states.
7. Additional work is needed to understand emotion-regulatory goals in depression.

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