Published in final edited form as:
Curr Psychiatry Rep. 2012 June ; 14(3): 182–187. doi:10.1007/s11920-012-0262-2.

Emotion Regulation and Anxiety Disorders

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Abstract

A growing body of research suggests that the construct of emotion regulation is important for understanding the onset, maintenance, and treatment of anxiety disorders. In this review, we provide a selective overview of this emerging field and highlight the major sources of evidence. First, evidence suggests that the construct of emotion regulation can be differentiated from the construct of emotion. Second, there is a large and consistent body of research demonstrating that emotion regulation strategies can modulate emotional responding, and this finding is observed in both behavioral and neuroimaging studies. Third, measures of emotion regulation explain incremental variance in measures of anxiety disorder symptoms not accounted for by measures of negative affect. Although the research implicating emotion regulation in the anxiety disorders is promising, future research will be necessary to further clarify causal mechanisms explaining how emotion regulation confers vulnerability for anxiety disorders and to improve the clarity and consistency of definitions of emotion regulation.

Keywords

Emotion Regulation; Anxiety

Introduction

Emotion regulation has been theorized to be a construct *distinct from anxiety* that incrementally explains the onset and maintenance of anxiety disorders [1]. From this perspective, anxiety disorders cannot be conceptualized simply as a problem of too much anxiety; instead, one's strategy and capacity to modulate one's emotions are essential towards understanding the etiology, maintenance, and treatment of anxiety disorders. Here, we provide a selective review of the relevant literature regarding the major sources of evidence for the emerging view of emotion regulation in anxiety disorders: 1) emotion and emotion regulation are distinct, but related, processes, 2) emotion regulation strategies modulate emotional responding, and 3) measures of emotion regulation explain incremental variance in measures of anxiety disorder symptoms.

Differentiating Emotion from Emotion Regulation

Fear is typically defined as an organism's defensive response that motivates the detection, escape, and avoidance of possible sources of danger [2], and common observable indicators typically include three response domains: overt behavior, physiology, and cognitive domains [3]. One limitation of defining fear solely from these observable domains is the possibility of

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creating a tautology; for example, a person avoids because they are afraid, and we know they are afraid because they avoid. In this regard, the neurobiolology of fear has been clarified in past 20 years using both human and animal models [4–5], allowing for a delineation of neural mechanisms mediating the generation of fear that motivates the observable domains of fear responding. This neural network of regions includes the amygdala, orbitofrontal cortex, and rostral anterior cingulate [4–6].

The amygdala, an almond shaped structure in the limbic cortex, is a key node within this network involved in detecting biologically salient stimuli (e.g., threat), via projections from the thalamus, and in motivating physiological, behavioral, and cognitive changes to respond to the detected threat, via projections to the hypothalamus, visual cortex, and prefrontal [4–5]. For example, amygdala lesions in humans are associated with decreased skin conductance towards fear-conditioned stimuli [7–8], despite being able to verbally report the stimulus-shock contingency [8], and impaired detection of threat [9]. Similarly, amygdala activity in response to threat measured during fMRI correlates with attentional biases towards threat among anxious populations [10]. In rodents, lesions of the central nucleus of the amygdala block three common behavioral indicators of conditioned fear: the freezing response [11], fear-potentiated startle [12], and response suppression [13]. Accordingly, the well-characterized neural circuitry mediating the detection of threat and motivation of fear-relevant responding allows for the differentiation of the source of motivation (i.e., fear grounded in a well-defined neural network) from the responses motivated (i.e., behavioral, physiological, and cognitive responses).

By contrast, emotion regulation is a multidimensional construct that broadly refers to a heterogeneous set of actions designed to modulate “which emotions we have, when we have them, and how we experience and express them” [14]. Given the emphasis on function (i.e., to modulate emotional responding), response topographies associated with emotion regulation take on many forms, such as re-appraisal, distraction, avoidance, escape, suppression, and the use of substances to enhance or blunt emotional experience. The process model of emotion regulation [15] suggests that emotion regulation strategies and their effects can have different consequences depending on the time during which they are employed. For example, prior to encountering an emotion-eliciting stimulus, an individual can engage in situation selection (e.g., refusing to give a public speech), situation modification (e.g., telling friends you would prefer not to about the recent death of your mother), attentional deployment (e.g., doing a crossword puzzle while in the waiting room at a doctor’s office), and cognitive change (e.g., re-interpreting the meaning of a situation, such as viewing a romantic date as an opportunity to learn about somebody new instead of as an opportunity to be negatively evaluated). Individuals can also attempt to modify the experience of emotion *after* the emotion has been activated. Possible response topographies in this domain can include suppression (i.e., attempting to block the behavioral and/or experiential aspects of an emotion) and acceptance (i.e., practicing awareness of the emotion-based sensations without attempting to alter them) [16–17].

Originally, research on emotion regulation focused on explicit/strategic response topographies in which the individual purposefully engages in a specific strategy to modulate emotional responding [17]. However, emotion regulation attempts can also be automatic/implicit, and this area has become a popular recent topic [18–19]. Implicit emotion regulation has recently been defined as “any process that operates without the need for conscious supervision or explicit intentions, and which is aimed at modifying the quality, intensity, or duration of emotional response” [18]. Given the ubiquitous nature of emotion experiences that often need to be modulated, implicit emotion regulation offers a needed compliment to explicit emotion regulation by being less dependent on cognitive resource availability. Specific response topographies that encompass implicit emotion regulation

include emotional conflict adaptation (e.g., after becoming distracted by an emotionally provocative cue one might implicitly increase attentional resources directed towards the current task), affect labeling (e.g., labeling an upcoming speech as 'dreadful' can unintentionally increase anxiety during it), and through emotion regulation goals and value (e.g., if one carries the belief that anxiety is dangerous, the experience of anxiety can be potentiated) [19].

Given the immense heterogeneity within the emotion regulation construct, it is not surprising that there are not well-defined neurobiological networks that clearly mediate emotion regulation. On the one hand, there has been a surge in neuroimaging research in the last decade focused on identifying neural correlates of emotion regulation, and this research has consistently identified regions within the prefrontal cortex (PFC), (e.g., dorsal lateral PFC, dorsal and ventral medial PFC, ventral lateral PFC, and subgenual, rostral, and dorsal anterior cingulate) as critical in both explicit and implicit attempts to regulate emotions [20–21]. On the other hand, the PFC is a vastly functionally heterogeneous structure; thus, it seems nearly impossible not to find significant activation somewhere in the PFC while engaging in an emotion regulatory process. For example, one review [20] indicated consistent activations located within the broad PFC across 16 neuroimaging studies of emotion regulation, but noted marked variability in the specific anatomical site within the PFC of activations correlated with emotion regulation (e.g., left versus right, dorsal versus ventral, etc). Moreover, a related problem is that not all anatomical sites implicated in emotion regulation have direct projections to key nodes within the emotion processing network, such as the amygdala [22], which begs the question of their involvement in emotion regulation specifically versus some domain general cognitive process (e.g., attention; effortful control).

However, one recent advance towards more clearly delineating the specific neural networks mediating emotion regulation was revealed through a meta-analysis of extant neuroimaging studies of emotion regulation [23]. These authors compared neural activation during cognitive re-appraisal studies, fear extinction studies, and placebo control studies under the premise that each of these methodologically varying conditions involves the modulation of emotional responding. Accordingly, activations due to specific peculiarities of one task should cancel out across the conditions, whereas common activations are largely attributable to emotion regulation processes. They observed one site activated across studies and across conditions: the ventral medial PFC. The meta-analysis also observed consistently decreased activation of the left amygdala across the conditions, suggesting validity of the interpretation that the task conditions share emotion regulation properties. Similarly, Schiller and Delgado (2010) [21] investigated common neural activations across three studies examining emotion regulation (using cognitive reappraisal), fear extinction, and fear reversal (i.e., a task in which the CS+ and CS- switch) and also found a common increase in vmPFC activation across tasks. The robust activation of this site is significant because it maps onto known anatomical pathways (e.g., the vmPFC has a direct projection to the amygdala) [22] and known neurobiological mechanisms (e.g., the rodent homologue of the vmPFC is necessary for fear extinction) [24]. While this research suggests a key region for emotion regulation, the human brain operates as a distributed network of information processing; thus, the vmPFC likely does not mediate emotion regulation independently and more research is needed to map out more clearly the regions working in concert with the vmPFC to modulate emotional responding.

Emotion Regulation Processes Can Modulate Emotional Responding

Behavioral Studies

The basic methodology for testing whether emotion regulation processes can modulate emotion responding involves experimentally manipulating the use of an emotion regulation strategy (via instructions and training) and examining changes in emotional responding during emotional provocation. In regards to studies of explicit emotion regulation strategies, use of this basic methodology has demonstrated that 1) expressive suppression (i.e., instructing participants to display no overt facial expressive signs of emotion) generally leads to increased sympathetic arousal during emotional provocation [17], 2) cognitive re-appraisal (i.e., appraising the emotional cue as less dangerous/more positive) generally leads to decreased self-reported negative affect [17, 25] and decreased startle probe potentiation during emotional provocation [26], 3) emotion suppression (i.e., instructions to suppress the experience of an emotion) can increase negative affect during emotion provocation among anxious individuals [27], and 4) emotional acceptance can decrease self-reported negative affect [27] and physiological responding [25] during emotional provocation. This body of research provides consistent experimental evidence that explicit emotion regulation strategies modulate emotional responding.

There are considerable methodological challenges for experimentally manipulating implicit emotion regulation, because giving explicit instructions to use a strategy necessarily induces an explicit/strategic component that confounds the intended implicitness of the manipulation [18]. One of the most established bodies of research that provides support that implicit emotion regulation techniques modulate emotional responding involves habitual training of attentional allocation [18–19, 28]. The concept behind this approach is that training a habitual bias either towards or away from threat stimuli induces the implicit the use of this strategy upon a new emotional provocation. In this paradigm, participants are engaged in training sessions during which they complete computerized tasks with hundreds of trials. The task typically presents two cues, one emotional and one neutral, on the screen at a time, and a probe then appears in the location previously occupied by one of the cues. The participant's task is to respond to the probe as quickly as possible. To train an implicit attentional bias away from threat, the training task manipulates the location of the probe to always appear in the location previously occupied by the neutral cue; thus, the participant is trained to avoid the emotional cue and attend to the neutral cue. Studies using this approach have found that the attention biases towards either neutral or emotion cues can be trained, and that these trained biases can accordingly increase (for biases towards threat) or decrease (for biases away from threat) emotional responding during subsequent emotional provocation as well as generally decrease symptoms of anxiety [29–30]. This body of research suggests that implicit emotion regulation strategies can also modulate emotional responding.

Neural Level of Analysis

Neuroimaging studies have adapted the basic behavioral methodology used for studying explicit emotion regulation for delivery in the scanner. This research has demonstrated 1) that re-appraisal is associated with increased PFC activity, reduced amygdala activity, and inverse relationships between PFC and amygdala activity [31–32], and 2) that instructions to enhance emotional experience via negative-re-appraisal are similarly associated with increased PFC activity and increased amygdala activity [32–33]. While a limitation of these studies is that they use pictorial stimuli that may not provide an ecologically valid model of anxiety disorder-relevant processes, studies [34] have found similar neural activations during fear-condition paradigms which may be a more ecologically valid paradigm for

modeling anxiety disorders. These studies provide consistent neuroimaging evidence that emotion regulation strategies modulate emotional responding.

The neural correlates of one implicit emotion regulation strategy, emotional conflict adaptation, have been relatively well-studied, albeit by only one research group. This paradigm capitalizes on the well-studied observation that cognitive control during a high conflict trial is enhanced if the preceding trial was also high conflict; that is, one 'adapts' to the previous high conflict trial by increasing cognitive control resources to the task, which improves performance on the subsequent high conflict trial [35]. In emotional conflict adaptation tasks, emotional conflict (i.e., emotional congruency between target and distracter) is manipulated on the current trial (high versus low) and on the previous trial (high versus low), and performance on the current trial is assessed as a function of this 2 × 2 manipulation. Two studies have found that adaptation to emotional conflict on the prior trial is mediated by activity of rostral ACC and accompanied by reductions in amygdala activity [6, 36]. Further, two recent neuroimaging studies using this task have demonstrated that generalized anxiety disorder is associated with less rostral ACC activity during conflict adaptation and less of an inhibitory effect of the rostral ACC on amygdala activity [37–38]. These studies support the hypothesis that implicit emotion regulation augments emotional responding.

Measures of Emotion Regulation Explain Incremental Variance in Anxiety Disorder Symptoms

There is growing evidence suggesting that maladaptive patterns of emotion regulation characterize individuals with anxiety disorders, particularly those with generalized anxiety disorder (GAD). The emotion dysregulation model [39] posits that GAD is marked by experiencing emotions quickly, easily, and with high intensity. Emotional reactivity in GAD makes emotions difficult to regulate which is further complicated by difficulty identifying and understanding emotions. Preliminary research has reported supportive evidence for this emotion dysregulation view of GAD. For example, Mennin and colleagues [40] found that analogue and clinical GAD samples exhibited difficulties understanding emotions, negative reactivity to emotions, and an inability to self-soothe following the experience of a negative emotion in comparison to healthy control participants. Such emotion regulation difficulties also predicted GAD status when controlling for worry, anxiety, and depressive symptom severity. A more recent study also found that emotion regulation difficulties predicted GAD above and beyond the experience of non-clinical panic attacks and panic disorder [41]. In fact, the shared relationship of emotion regulation difficulties with both panic disorder and GAD may partially explain the association between these disorders.

Engagement in maladaptive emotion regulation strategies may also uniquely contribute to the development of panic disorder. For example, Tull, Rodman, and Roemer [42] found that experiential avoidance, emotional non-acceptance, and lack of emotional clarity was associated with the fear of bodily sensations among those with a recent history of uncued panic attacks above and beyond other panic-relevant variables. Research has also shown that those with a recent history of uncued panic attacks report using more emotionally avoidant regulation strategies during exposure to positive and negative emotion-eliciting film clips, despite comparable levels of distress and physiological arousal [43]. Although Anxiety sensitivity (AS) has been identified as a specific risk factor for the development of panic disorder, there is evidence suggesting that whether or not AS leads to the development of panic disorder may depend, at least in part, on how emotion is regulated. Consistent with this notion, Kashdan, Zvolensky, and McLeish [44] found that among those high in AS, anxious arousal and worry were heightened in the presence of less acceptance of emotional distress; anxious arousal, worry, and agoraphobic cognitions were heightened when fewer

resources were available to properly modulate affect; and agoraphobic cognitions were heightened in the presence of high emotion expressiveness.

Emotion regulation difficulties may also contribute to the development of posttraumatic stress disorder (PTSD). Indeed, expressive suppression has been found to be associated with PTSD symptoms in a trauma-exposed community sample [45]. PTSD symptom severity has also been found to be associated with lack of emotional acceptance, limited access to effective emotion regulation strategies, and lack of emotional clarity [46]. In addition, such difficulties in emotion regulation were associated with PTSD symptom severity even when controlling for negative affect. Similarly, Cloitre, Miranda, Stovall-McClough, and Han [47] found that emotion regulation and interpersonal problems were both significant predictors and together made contributions to functional impairment equal to that of PTSD symptoms among women with a history of childhood abuse. Participants' improved capacity to regulate negative emotion during exposure-based treatment was also found to mediate the association between therapeutic alliance established early in treatment and PTSD symptoms at posttreatment [48]. This finding suggests that emotion regulation difficulties may also have implications for the treatment of PTSD. Given that emotion regulation difficulties may also partially explain the high rates of PTSD among those seeking treatment for substance use disorders, targeting emotion regulation difficulties during treatment may also lead to changes in conditions that are commonly comorbid with PTSD. Indeed, emotion-focused coping has been found to mediate the relationship between PTSD symptom severity and negative situational drug use [49].

Although the available literature suggests that emotion regulation difficulties is uniquely associated with anxiety disorder symptoms in adults and children, a mechanism that may account for the association remains unclear. One hypothesis in this regard is that emotion regulation strategy confers vulnerability to anxiety disorders by potentiating the effect of negative affect. Consistent with this hypothesis, research has found the predicted interactions between measures emotion regulation and measures of negative affect in predicting severity of anxiety disorder symptoms [44]. These data converge in suggesting that emotion regulation may potentiate the contribution of emotional reactivity towards anxiety disorder symptoms.

Conclusions

This selective review highlighted the major sources of evidence for the emerging view that one’s strategy and capacity to regulate emotion is an important determinant in the onset and maintenance of anxiety disorders; however, this literature is not without limitations. For example, is it one’s capacity for emotion regulation, one’s selection of emotion regulation strategies, or both, that most potently explains vulnerability for anxiety disorders? Further, the multidimensional nature of the emotion regulation construct makes it difficult to clearly define and differentiate from emotion. In addition to the identification of causal mechanisms, future longitudinal research is also needed to clarify the direction of effects. That is, does emotion regulation lead to the development of anxiety disorders or does anxiety disorders lead to the development of maladaptive emotion regulation strategies? McLaughlin and colleagues [50] did find that emotion dysregulation predicted increases in anxiety symptoms over seven months after controlling for baseline symptoms. By contrast, anxiety symptoms did not predict increases in emotion dysregulation after controlling for baseline emotion dysregulation. Additional longitudinal research along these lines will prove vital in further clarifying the direction of the relationship between emotion regulation and anxiety disorder symptoms. Future research is needed to further clarify how this emerging field will aid in our understanding and treatment of anxiety disorders.

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