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Emotion Regulation and the Anxiety Disorders: An Integrative Review

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Abstract

The construct of emotion regulation has been increasingly investigated in the last decade, and this work has important implications for advancing anxiety disorder theory. This paper reviews research demonstrating that: 1) emotion (i.e., fear and anxiety) and emotion regulation are distinct, non-redundant, constructs that can be differentiated at the conceptual, behavioral, and neural levels of analysis; 2) emotion regulation can augment or diminish fear, depending on the emotion regulation strategy employed; and 3) measures of emotion regulation explain incremental variance in anxiety disorder symptoms above and beyond the variance explained by measures of emotional reactivity. The authors propose a model by which emotion regulation may function in the etiology of anxiety disorders. The paper concludes with suggestions for future research.

Keywords

Emotion Regulation; Anxiety; Fear; Coping; GAD

The history of anxiety disorder research exemplifies how an emphasis on empirical research can facilitate theoretical and practical developments. The purpose of this paper is to explore how data derived from emotion regulation studies might similarly advance existing anxiety disorder theory. This paper will review several lines of work showing that: 1) emotion (i.e., fear and anxiety) and emotion regulation can be differentiated at the conceptual, behavioral, and neural level of analyses; 2) emotion regulation attempts can augment and diminish emotional responding; and 3) measures of emotion regulation explain incremental variance in anxiety disorder symptomatology. We then follow with a theoretical model derived from this literature showing how emotion regulation may function in the development and maintenance of anxiety disorders, and cast that model in terms of several testable predictions. The paper concludes with recommendations for future research that may facilitate theoretical developments by elucidating the manner in which emotion regulation functions in the etiology and maintenance of anxiety disorders.

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Differentiating Emotion from Emotion Regulation

Defining Emotion

A necessary step in demonstrating that emotion regulation incrementally adds to existing anxiety disorder theory is demonstrating that emotion regulation is not redundant with existing emotion constructs. In the context of anxiety disorders, fear and anxiety are the primary relevant emotions from which emotion regulation needs to be distinguished. 'Fear' refers to an emotional system motivating defensive behaviors elicited from an immediate specific threat cue; 'anxiety' refers to an emotional system motivating defensive behaviors elicited from an impending non-specific threat cue (e.g., predatory imminence model; Fanselow & Lester, 1988; Quinn & Fanselow, 2006). For the purposes of the present paper, fear and anxiety will be treated as unitary instead of as separate constructs, but it is important to note that future research is necessary to test whether emotion regulation differentially interacts with anxiety versus fear. At the conceptual level of analysis, fear is defined as an organism's defensive response that motivates the detection, escape, and avoidance of possible sources of danger (e.g., Barlow, 2002; Öhman, 2000; Öhman & Mineka, 2001). At the phenomenological level of analysis, fear is typically defined in three domains: overt behavior, physiology, and verbal-cognitive domains (Lang, 1968; Lipp, 2006; Zinbarg, 1998). These lower-order phenomenological indicators support the conceptual definition of fear. For example, enhanced threat detection characterized by visual awareness and bodily orientation operates as a first line protective function for the individual. Acute sympathetic arousal enhances blood flow and oxygen to large muscle groups to facilitate response mobilization. Escape and avoidance action tendencies serve a protective function by creating physical distance between the individual and the source of threat.

Despite observations that these behavioral, physiological, and verbal-cognitive indicators characterize fear, these indicators tend to be weakly intercorrelated (Hodgson & Rachman, 1974; Lang, 1968; Rachman, 1978; Rachman & Hodgson, 1974; Zinbarg, 1998). One explanation for this 'loose coupling' is that the indicators are multiply determined. The intensity of fear elicitation, for example, may mediate the degree of association. Marks and colleagues (1971) found that skin conductance and heart rate changes significantly correlated during exposure to a highly threatening stimulus, whereas no correlation was noted during exposure to less threatening stimuli. Another determinant of the fear indicators may be contextual demands. Miller and Bernstein (1972) found moderate correlations between avoidance and subjectively reported anxiety, heart rate, and respiration during an exposure task in which individuals were told they could avoid when they become too uncomfortable. When participants were told to control their urge to avoid, there was no correlation between avoidance and any of the other indicators (see Hodgson & Rachman, 1974). Accordingly, fear may be a determinant of the indicators, but contextual factors may also determine whether the indicators occur and to what degree they intercorrelate.

Zinbarg (1998) argued that the loose coupling of the three response systems could be explained by a hierarchical model of fear in that: 1) the three response domains are lower-order indicators of a unitary higher-order fear construct, and 2) the lower-order indicators are multiply determined. From this perspective, fear is not reducible to any of the observable indicators, either singularly or in combination; rather, fear is a unitary higher-order construct that downwardly determines variance in the lower-order indicators. Furthermore, factors other than fear can determine variance in the lower-order indicators. As we will describe, emotion regulation may be one of the key determinants responsible for lower-order factor variability. Thus, weak correlations between indicators of fear would be expected because the response domains are only indicators of a higher-order fear construct and they can be determined by multiple factors other than fear.

Zinbarg (1998) speculated that future neurobehavioral research would further clarify the boundaries of the unitary higher-order fear construct. Consistent with this prediction, a wealth of data demonstrates that the amygdala, a neural structure in the medial temporal lobes of the brain, is a critical structure mediating behavioral, cognitive, and physiological indicators of fear (Kim & Jung, 2006; LeDoux, 2000; Myers & Davis, 2007; Quinn & Fanselow, 2006). Lesions of the central nucleus of the amygdala in rodents block three common behavioral indicators of conditioned fear: fear-potentiated startle (Campeau & Davis, 1995; Walker & Davis, 1997), the freezing response (Zimmerman, Rabinak, McLachlan, & Maren, 2007), and response suppression (Lee, Dickinson, & Everitt, 2005). Human patients with amygdala lesions fail to demonstrate increased skin conductance towards fear-conditioned stimuli (Bechara et al., 1995, 1999), despite being able to verbally report the stimulus-shock contingency (Bechara et al., 1995). Similarly, human patients with amygdala lesions demonstrate impaired skin conductance responses while passively viewing emotional images (Glascher & Adolphs, 2003) as well as impaired detection of threat (Anderson & Phelps, 2001). Amygdala activation during fMRI correlates with attentional biases towards threat among anxious populations (Monk et al., 2004; van den Heuvel et al., 2005), further suggesting a role of the amygdala in determining cognitive indicators of fear.

These data demonstrate a robust relation between the amygdala and behavioral, physiological, and cognitive indicators of fear, thereby supporting Zinbarg's (1998) hierarchical model of fear. Moreover, the data suggest an important temporal sequencing: amygdala processing occurs first, which subsequently motivates physiological, behavioral, and cognitive responses. The lower-order indicators of fear may be outputs of a unitary fear system, which may be neurologically centered around the amygdala. Given the relatively weak correlations between the lower-order indicators and the observations that contextual demands influence the correlations between the indicators (Hodgson & Rachman, 1974; Zinbarg, 1998), it is likely that other factors (e.g., emotion regulation) may moderate the degree to which the fear system motivates changes in the lower-order outputs. This analysis differentiates the source of motivation (i.e., fear grounded in the amygdala) from the responses motivated (i.e., behavioral, physiological, and cognitive responses), which helps reduce tautological assessments of fear (e.g., people avoid because they are afraid, we know they are afraid because they avoid).

Defining Emotion Regulation

Emotion regulation in the current paper refers to a heterogeneous set of actions that are designed to influence "which emotions we have, when we have them, and how we experience and express them" (Gross, 2002, p. 282). Emotion regulatory behavior can manifest in many obvious and subtle ways and include: re-appraisal, distraction, avoidance, escape, suppression, emotion and problem-focused coping, and use of substances to enhance or blunt emotional experience. Each of these strategies subsumes numerous actions that can be applied to both positive and negative emotional states (Parrott, 1993). Most of them, however, can be characterized by actions that aim to alter the form, frequency, duration, or situational occurrence of events that may precede an emotional response as well as the events that may follow an emotional response (Gross, 1998a). This process model of emotion regulation (Gross, 2007) suggests that emotion regulation strategies and their effects can have different consequences depending on the time during which they are employed.

Specific response topographies an individual may employ prior to encountering the emotion-eliciting stimuli include situation selection (e.g., detouring to avoid driving over a bridge), situation modification (e.g., telling friends you would prefer not to talk about an impending interview if the topic gets brought up), attentional deployment (e.g., distracting children while preparing for an injection), 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). These different response possibilities all share the similar theme of altering the experience of an emotion before the occurrence of the emotion. Individuals can also engage in emotion regulation attempts to alter the experience of an emotion *after* the emotion has been activated. Specific topographies may include suppression (i.e., inhibiting the behavioral and/or experiential aspects of an emotion) and acceptance (i.e., noticing the emotion-based sensations without attempting to alter them; Feldner et al., 2003; 2006; Gross, 1996; Gross & Levenson, 1993; Levitt et al., 2004). Regardless of the timing at which regulation strategies are employed, it is important to note that regulatory processes may be relatively automatic or habitual occurring in or outside of awareness (e.g., selective attention; see Mauss et al., 2007), whereas others may be more purposeful or deliberate (e.g., escape, blame, suppression).

Similar to fear/anxiety, emotion regulation can be conceptualized as a higher-order construct downwardly influencing lower-order indicators. Cognitive-affective neuroscience has consistently demonstrated that higher-order cortical structures, particularly the prefrontal cortex (PFC), its subunits [e.g., medial prefrontal cortex (mPFC), orbitofrontal cortex, (OFC)] and related structures [e.g., anterior cingulate cortex (ACC)], mediate attempts to regulate emotions (Davidson, Fox, & Kalin, 2007; Ochsner & Gross, 2008; 2005; Quirk, 2007; Quirk & Beer, 2006). It is important to note that linking emotion regulation with the PFC is not necessarily 'neural phrenology' (cf., Uttal, 2001). Rather, advances in neuroscience illuminate that 1) emotion regulation and fear are mediated by different neural regions, and 2) the role of the PFC in emotion regulation is in theoretical alignment with the general function of the PFC. Miller and Cohen's (2001) integrated theory of PFC function posits that this region broadly maintains goal directed behavior by controlling the activity of other brain structures.

> "In this respect, the function of the PFC can be likened to that of a switch operator in a system of railroad tracks. We can think of the brain as a set of tracks (pathways) connecting various origins (e.g., stimuli) to destinations (responses). The goal is to get the trains (activity carrying information) at each origin to their proper destination as efficiently as possible, avoiding any collisions. When the track is clear (i.e., a train can get from its origin to destination without risk of running into any others), then no intervention is needed (i.e., the behavior can be carried out automatically and will not rely on the PFC). However, if two trains must cross the same bit of track, then some coordination is needed to guide them safely to their destinations… In the brain, this is achieved by biasing the influence that patterns of PFC activity have on the flow of activity in other parts of the brain…" (Miller & Cohen, 2001, p. 184).

This analogy to a switch operator is consistent with the hypothesized role of the PFC in emotion regulation. Emotion motivates behavior, but in some contexts, emotional behavior is inappropriate. The PFC's role in emotion regulation may be in inhibiting the bottom-up influences of emotion so that individuals can continue to engage in context-appropriate goal-directed behavior.

Two main lines of research provide evidence that the PFC and functionally-related structures mediate emotion regulation processes. First, it is becoming increasingly recognized that the PFC is critically involved in down-regulating neural fear structures (e.g., amygdala) during fear extinction learning (Myers & Davis, 2007; Quirk, 2006; Sotres-Bayon et al., 2004, 2006). For example, Morgan and colleagues (1993) found that PFC lesions in rodents had no effect on fear acquisition, but resulted in relatively prolonged fear responding to the conditioned stimulus during extinction training. Second, a wealth of data demonstrates PFC activation during functional magnetic resonance imaging (fMRI) studies in which individuals are asked to reappraise, suppress, augment, and diminish their emotional responses (see Bishop, 2007; Oschner & Gross, 2005, 2008). For instance, Oschner and colleagues (2004) identified increased PFC activity during both up-regulation (via instructions to imagine a scene as

happening to a loved one) and down-regulation (via re-appraisal) of emotion. These data demonstrate that higher-order cognitive processes in which individuals attempt to alter their emotional experience/expression recruit neural regions centered around the PFC. Accordingly, emotion regulation may be neurologically centered around activity of the PFC and related structures.

One challenge within the emotion regulation literature is that emotion regulation may encompass a broad range of response topographies. If emotion regulation is too broadly defined as any attempt to alter the experience of emotion, it is difficult to see how anything a person does could *not* be indicative of emotion regulation. Similar arguments have been made elsewhere in the literature that emotion and emotion regulation are so closely coupled that distinguishing between the two is exceedingly difficult (Campos, Frankel, & Camras, 2004). Given this important conceptual limitation in this growing field, we limit our focus to two specific emotion regulation topographies that appear to be best operationalized in the available emotion regulation literature: re-appraisal and suppression. However, we acknowledge the limitation that narrowing our focus to these two forms of emotion regulation limits the generalizability of the findings and conclusions drawn herein.

Summary

Fear/anxiety and emotion regulation can be differentiated at the conceptual, neural, and behavioral (i.e., phenomenological expressions) levels of analyses. Fear/anxiety is an organism's defensive response towards potential sources of danger, involves physiological, overt-behavioral, and cognitive indicators, and is neurologically-oriented around the amygdala. Emotion regulation refers to attempts to alter an emotional experience, can involve specific overt-behavioral and cognitive strategies, and is neurologically-oriented around PFC and functionally-related neural structures. Accordingly, fear/anxiety and emotion regulation are distinct and non-redundant constructs.

Emotion Regulation Processes Can Augment and Diminish Emotional Responding

Cognitive-Behavioral Levels of Analysis

Gross and Levenson (1993) compared individuals told to suppress expressive emotional behavior while watching neutral and disgusting films to individuals told to just watch the films. Results generally demonstrated no differences between the groups in self-reported emotion, but greater sympathetic arousal in the suppression group compared to the no-instruction group. Similarly, Gross and Levenson (1997) found that behavioral suppression during a sad film led to greater sympathetic arousal compared to no-instruction, and the groups again did not differ in self-reported sadness. Gross (1998b) then added a re-appraisal condition to his previous experimental design, in which participants were told to view a surgery film in a detached manner. Results demonstrated that participants in the re-appraisal condition experienced less negative emotion while watching the film compared to both the behavioral suppression and control groups, whereas the behavioral suppression group did not show differences in self-reported emotion and did show elevated sympathetic arousal compared to both other groups. Finally, two studies (Jackson et al., 2000; Lissek et al., 2006) have found that re-appraisal while viewing negative images or receiving electric shocks led to less startle probe potentiation compared to instructions to maintain the emotion or up-regulate the emotion (i.e., via imagining the scene as more personally relevant or the shocks as more painful). Though these studies differ in the type of emotion eliciting stimulus employed, the data collectively demonstrate that behavioral suppression does not necessarily reduce negative affect, but instead tends to increase arousal and startle. Re-appraisal, on the other hand, appears to be effective in reducing

negative affect and startle. These data suggest that emotion regulation techniques can affect emotional responding.

While Gross's (19931997) methodologies used behavioral suppression as a form of emotion regulation, other studies have investigated whether *emotion* suppression (i.e., instructions to suppress the experience of an emotion) impacts affective outcomes. Feldner and colleagues (2003) found that individuals scoring high in cognitive avoidance instructed to suppress reported more anxiety during a laboratory-based carbon dioxide (CO_2) enriched-air biological challenge compared to participants low in baseline levels of cognitive avoidance instructed to suppress. However, instructions to suppress generally resulted in decreased heart rate responding relative to instructions to observe the emotional sensations. Feldner and colleagues (2006) later found that individuals instructed to suppress demonstrated slower physiological recovery subsequent to a CO_2 biological challenge compared to the observe group. Similarly, other research has found that individuals either high in anxiety sensitivity (Eifert & Heffner, 2003) or diagnosed with panic disorder (Levitt et al., 2004) demonstrated less self-reported negative emotion during a CO_2 challenge when instructed to accept negative feelings compared to instructions to suppress or a no-instruction control. Finally, Campbell-Sills and colleagues (2006) extended this work among a mixed anxiety and mood disordered sample and found that acceptance led to lower heart rate while viewing a negative film relative to suppression, and acceptance showed less negative affect during recovery after the film compared to suppression. Though these studies differ in emotion eliciting stimulus and sample characteristics, they converge in demonstrating differential effects of emotional suppression and acceptance.

Two studies have investigated the effect of enhancing emotions on the startle response. Participants were asked to enhance their emotions while viewing negative pictures or receiving electric shocks (Jackson et al., 2000; Lissek et al., 2006). Post-experiment questionnaires demonstrated that participants generally used a negative re-appraisal technique while enhancing their emotion (e.g., imagining the scene happening to a loved one or themselves, focusing on the worst possible outcome). Results demonstrated that negative re-appraisal led to increased startle during startle probes relative to control instructions.

The effect of emotion regulation techniques on the experience of emotion demonstrate 1) emotion regulation techniques affect subjective, physiological, and behavioral indicators of emotion and 2) depending on the emotion regulation techniques employed, emotional responding can either be increased or decreased. It appears re-appraisal generally leads to less self-reported negative affect, less physiological reactivity, and less startle. Behavioral suppression leads to increased physiological reactivity, but may not influence self-reported negative affect. Early evidence suggests emotion suppression may decrease physiological arousal relative to an observation condition during emotion elicitation but increase such arousal subsequent to the offset of the emotion evocation. Emotion suppression also generally appears to increase negative affect relative to re-appraisal, but when considered within the context of other risk factors linked to anxiety (i.e., anxiety sensitivity), the effects are not consistent (Feldner et al., 2006). Finally, negative re-appraisal appears to enhance startle during negative emotion elicitation.

The articles cited above employ a similar experimental paradigm, and it is important to note relevant limitations to this paradigm. The paradigm can only examine *acute* emotional consequences of *acutely applied* emotion regulation techniques. Accordingly, these studies cannot address the long-term consequences of habitual use of a given emotion regulation strategy. This limitation is particularly important, as it is difficult to conclude that emotion regulation functions in the etiology or maintenance of anxiety disorders without this data. A second limitation of this body of research is that the studies all differ in emotion elicitation paradigms, comparison conditions, and sample types. For example, the work by Gross (e.g.,

Gross et al., 1993; 1998b) used mainly a disgust elicitor as the negative mood inducing stimulus, whereas Feldner and colleagues' work (Feldner et al., 2004; 2006) used biological challenge procedures as the eliciting stimulus. Moreover, the Feldner et al. studies mainly tested emotional suppression, whereas the Gross studies tested behavioral suppression and reappraisal. Thus, the studies differ on at least two important domains. There is general convergence in basic findings across the studies, which provides preliminary evidence that emotion regulation may unitarily modulate different negative emotions (e.g., fear versus disgust versus anxiety), however it is vital for future research to continue to directly test whether the emotion regulation techniques have similar effects across negative mood states. Nonetheless, the following section generally corroborates these basic findings at the neural level of analysis.

Neural Level of Analysis

There has been a surge of recent neuroimaging studies investigating the neural correlates and consequences of re-appraisal and suppression (see Ochsner & Gross, 2008). Ochsner and colleagues (2002) presented participants with negative and neutral photos during fMRI scanning and asked participants to either attend to their feelings without trying to alter them, or to re-appraise the images so that they no longer experienced negative feelings. Results demonstrated that re-appraisal led to less subjectively reported negative affect while viewing negative pictures relative to the attend condition. Re-appraisal was associated with greater activity in PFC regions relative to the attend condition. ACC activity during re-appraisal was correlated with a reduction in negative affect ($r = .81$). Re-appraisal was associated with less amygdala activity relative to the attend condition. Left ventral PFC was negatively correlated with amygdala activity during re-appraisal ($r = -.68$). Accordingly, re-appraisal is associated with greater PFC activity and less amygdala activity, and importantly, the magnitude of PFC activity negatively predicts the magnitude of amygdala activity. These neuroimaging data strongly complement cognitive-behavioral data in suggesting that emotion regulation strategies can affect the degree of fear experienced. Several other studies also demonstrate that re-appraisal is associated with increased PFC activity, reduced amygdala activity, and significant negative correlations between PFC and amygdala activity (e.g., Banks et al., 2006; Hariri et al., 2003; Ochsner et al., 2004; Phan et al., 2005; Urry et al., 2006). Collectively, these data demonstrate 1) a strong relation between PFC and re-appraisal techniques, 2) reduced amygdala activity during re-appraisal, and 3) greater PFC recruitment during re-appraisal tends to predict less amygdala activity and negative affect.

Additional studies have found that instructions to enhance emotional experience via negative-re-appraisal are similarly associated with increased PFC activity and *increased* amygdala activity. Ochsner and colleagues (2004) asked participants to watch, re-appraise, or negative-reappraise their feelings while watching negative pictures. These investigators found that self-reported negative affect increased linearly across re-appraise, watch, and negative-reappraise instructions. Importantly, these authors also found increased PFC activity in the reappraise and negative-reappraise conditions, and that amygdala activity increased linearly from instructions to re-appraise, watch, and negative-reappraise. Other investigators (Eippert et al., 2007; Goldin et al., 2008; Kim & Harmann, 2007; Urry et al., 2006) have replicated these basic findings and demonstrate that both re-appraise and negative-reappraise instructions led to increased PFC activity, re-appraise led to less amygdala activity relative to watch instructions, while negative-reappraise led to greater amygdala activity relative to watch instructions or reappraise instructions.

The above evidence demonstrating inverse relations between PFC and amygdala activity is limited to correlational designs and it cannot be determined if increased PFC activity causes decreased amygdala activity, or if decreased amygdala activity somehow causes increased PFC

activity. Electrical stimulation and lesion studies in rodents and non-human primates, however, do provide some evidence that PFC activity causally affect amygdala output. Electrical stimulation of the mPFC in rodents following a fear-conditioning paradigm reduces neural responding in the lateral nucleus (Rosenkranz et al., 2003) and central nucleus (Quirk et al., 2003) of the amygdala. Kalin and colleagues (2007) found that OFC lesions in monkeys led to less fear-related behaviors (e.g., freezing) in response to a human profile and a snake relative to monkeys without OFC lesions. Morgan and LeDoux (1999) found that PFC lesions in rodents reduced fear towards fear-conditioned stimuli, but did not affect fear learning. LaCroix and colleagues (2000) found that depending on the particular PFC region lesioned in rodents, freezing towards anxiety-provoking situations or fear-provoking situations were reduced. These data collectively provide experimental evidence that regions of the PFC causally affect emotion processes.

Another limitation of the neuroimaging studies cited above is that they are largely limited to using pictorial stimuli to induce negative affect. Pictorial stimuli may not be an externally valid experimental analogue of the day-to-day stimuli that elicit negative affect in individuals generally, or anxiety disordered individuals specifically. Moreover, the specific negative emotions that are induced may vary from study to study (e.g., fear versus disgust versus sadness), and it is not clear that the types of negative emotions regulated in neuroimaging studies map on to the types of negative emotions that individuals with anxiety disorders may be experiencing or attempting to regulate. Recent research may help address these limitations.

Milad and colleagues (2007) and Phelps and colleagues (2004) engaged participants in a fear-conditioning paradigm, in which the unconditioned stimulus (US) was a mild but painful shock, during fMRI testing. Results demonstrated PFC activity during extinction trials, and that greater activation of the PFC during extinction led to greater reduction in skin conductance elicited by the CS (r = .66, Milad et al., 2007; r = .75, Phelps et al., 2004). Delgado and colleagues (2008) extended these fear-conditioning findings to an emotion regulation paradigm. These authors embedded instructions to attend (e.g., think of your feelings) or regulate (e.g., think of something calming in nature) their emotions into the same fear-conditioning paradigm used by Phelps and colleagues (2004), with the exception of omitting extinction trials. Behavioral data demonstrated that instructions to regulate led to decreased skin conductance in response to the CS relative to instructions to attend. fMRI data demonstrated that greater PFC activity during instructions to regulate led to reduced skin conductance responses to the CS. Amygdala activity towards the CS was also reduced during instructions to regulate. These three studies (Delgado et al., 2008; Milad et al., 2007; Phelps et al., 2004) suggest that the findings from prior studies (see Ochsner & Gross, 2005, 2008) generalize to a fear-conditioning paradigm, which may be a more ecologically valid paradigm for modeling anxiety disorders.

Summary

The data reviewed above suggest that emotion regulation techniques can augment or diminish emotional responding, depending on the technique employed. This effect appears to hold at both the cognitive-behavioral and neural levels of analysis. Moreover, the neuroimaging studies demonstrate that fear and emotion regulation engage distinct neural regions, and that neural regions mediating emotion regulation are negatively correlated with neural regions mediating fear during re-appraisal. Importantly, these effects are found in fear-conditioning paradigms that may provide a more ecologically valid model of anxiety disorders (Delgado et al., 2008; Phelps et al., 2004). The data also further demonstrate the distinctness of the fear/anxiety and emotion regulation constructs. Therefore, emotion regulation appears to be a distinct construct that may causally influence fear/anxiety expression.

Traditional lower-order indicators of fear (i.e., overt-behavior, verbal-cognitive processes, and physiology) are loosely coupled and multiply determined (Hodgson & Rachman, 1974; Zinbarg, 1998). Neurobehavioral research demonstrates that the amygdala may mediate behavioral, cognitive, and physiological indicators of fear (e.g., Davis & Whalen, 2001; Myers & Davis, 2007). Data also demonstrate that emotion regulation techniques affect these same behavioral, verbal-cognitive, and physiological indicators. Moreover, neural regions mediating emotion regulation are strongly negatively correlated with amygdala activity during re-appraisal. Accordingly, it is possible that emotion regulation may determine significant variance among the three commonly-employed indicators of fear. These data appear to address previous concerns over the loose coupling of fear indicators. For example, Rachman (1978) asked "How should we describe a person who feels calm while approaching a feared situation but, when the responses are measured, displays clear physiological disturbances? An autonomic coward?" (p. 243). The current conceptualization would suggest that emotion regulation can modulate the output of fear and maintain goal-directed behavior, and thus desynchrony is expected under some circumstances. If the key dependent variables in anxiety disorder research are broadly construed as these lower-order indicators, then it appears that theory needs to account for the influence of both fear *and* emotion regulation strategies.

Finally, though specific emotion regulation topographies have been linked with specific emotional consequences, it is unlikely that any emotion regulation technique is universally 'good' or 'bad' across contexts. Future research is needed to clarify the boundary conditions under which the different topographies effectively, and appropriately, up and down regulate emotional responding.

Emotion Regulation Explains Incremental Variance in Anxiety Disorder Symptoms

Theory as well as experimental and clinical evidence suggest that anxiety disordered populations, especially persons with generalized anxiety disorder (GAD), appear to be characterized by dysfunctional emotion regulation strategies (cf. Amstadter, 2008). According to the emotion dysregulation model (Mennin et al., 2004), GAD is marked by experiencing emotions quickly, easily, and with high intensity. This emotional reactivity makes emotions difficult to regulate and is further complicated by the difficulty with identifying and understanding emotions that characterizes those with GAD. Preliminary evidence for the emotion dysregulation model has been found in the literature, though this research is limited to mainly self-report measures. For example, Salters-Pedneault and colleagues (2006) found that deficits in emotional clarity, the acceptance of emotions, the ability to engage in goal-directed behaviors when distressed, the ability to control impulsive behaviors when distressed, and access to effective regulation strategies (all constructs measured via the Difficulties in Emotion Regulation Scale, Gratz & Roemer, 2004) were significantly related to analogue GAD status.

Research in this area has defined emotion regulation as ways of responding to one's emotions (regardless of the nature or quality of these emotions), with difficulties in emotion regulation consisting of dysfunctional (e.g., inappropriate, inflexible, maladaptive) responses to emotions. As such, the current conceptualization of emotion regulation distinguishes learned difficulties in emotion regulation from temperamental emotional vulnerabilities (Gratz & Roemer, 2004). Recent research also indicates that self-report measures of emotion regulation predict anxiety disorder symptoms when controlling for measures of emotion reactivity and temperamental emotional vulnerabilities. For example, Mennin and colleagues (2005) 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. Furthermore, such emotion

regulation difficulties were predictive of GAD status even when controlling for worry, anxiety, and depressive symptom severity. A more recent study also found that emotion regulation difficulties reliably predicted GAD above and beyond the experience of non-clinical panic attacks and panic disorder (Tull, Stipelman, Salters-Pedneault, & Gratz, in press).

Other studies have also implicated maladaptive emotion regulation in panic disorder, though some of this research is limited to self-report measures. For example, Tull, Rodman, and Roemer (2008) found that the fear of bodily sensations predicted experiential avoidance, emotional non-acceptance, and lack of emotional clarity above and beyond other panic-relevant variables in a sample of 91 individuals with a recent history (past year) of uncued panic attacks. Experimental extensions also show that despite comparable levels of distress and physiological arousal, participants 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 (Tull & Roemer, 2007). Anxiety sensitivity (AS) has been identified as a core risk factor for the development of panic disorder (Schmidt et al., 1999). However, recent work suggests that whether or not AS ultimately leads to the development of panic disorder may depend, at least in part, on how individuals regulate their emotions. Consistent with this notion, Kashdan, Zvolensky, and McLeish (2008) 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. Similarly, the experimental work by Feldner and colleagues (2006) further suggests that the affective consequences of emotional suppression in the context of a laboratory-based biological challenge procedure depend, at least in part, on levels of AS.

Emotion regulation may also have incremental utility in explaining recovery from posttraumatic stress. For example, expressive suppression has been found to be associated with posttraumatic stress disorder (PTSD) symptoms in a trauma-exposed community sample (Moore, Zoellner, & Mollenholt, 2008). A recent study also found that PTSD symptom severity was associated with lack of emotional acceptance, difficulty engaging in goal-directed behavior when upset, impulse-control difficulties, limited access to effective emotion regulation strategies, and lack of emotional clarity (Tull, Barrett, McMillan, & Roemer, 2007). 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 (2005) 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.

Emotion regulation difficulties may also have implications for the treatment of PTSD. For example, it has been shown that participants' improved capacity to regulate negative mood states during exposure-based treatment mediated the relationship between therapeutic alliance established early in treatment and PTSD symptoms at posttreatment (Cloitre, Stovall-McClough, Miranda, & Chemtob, 2004). Emotion regulation difficulties may also partially explain the high rates of PTSD among those seeking treatment for substance use disorders. Indeed, emotion-focused coping has been found to mediate the relationship between PTSD symptom severity and negative situational drug use (Staiger, Melville, Hides, Kambouropoulos, & Lubman, in press).

Evidence is accumulating that people with panic spectrum problems and relatively elevated levels of posttraumatic stress symptoms utilize substances (e.g., alcohol, nicotine) to regulate negative emotions to a greater degree than those without such problems (Bibb & Chambless, 1986; Zvolensky, Gonzalez, Bonn-Miller, Bernstein, & Goodwin, 2008). Importantly, use of

these types of substances and related processes (e.g., withdrawal from them) has been implicated in the development and maintenance of such conditions (Breslau & Klein, 1999; Cox, Norton, Dorward, & Fergusson, 1989; van der Velden, Kleber, & Koenen, 2008). Thus, while substance use and related factors are thought to have direct effects on the development and maintenance of these conditions, the emotion regulation functions of these substances are critical in understanding why many people with anxiety psychopathology use substances and subsequently develop anxiety problems.

Recent evidence suggests that the relation between emotion regulation and some anxiety disorder symptoms is not necessarily direct. Cisler and colleagues (2009) found that a measure of emotion regulation [i.e., the Difficulties in Emotion Regulation Scale (DERS); Gratz & Roemer, 2004] did not independently predict self-reported spider fear or contamination-related OCD when negative affect and disgust propensity were controlled. However, emotion regulation interacted with disgust propensity to predict both spider fear and contamination-related OCD. Kashdan and colleagues (2008) found that non-acceptance of emotions and limited access to emotion regulation strategies moderated the effect of anxiety sensitivity on anxious arousal, worry, and agoraphobic cognitions. Kashdan and Steger (2006) similarly found that social anxiety and expressive suppression interacted to predict low positive emotion and low positive events. These data converge in suggesting that emotion regulation may potentiate the contribution of emotional reactivity (e.g., anxiety sensitivity, social anxiety, disgust) towards anxiety disorder symptoms. Accordingly, emotion regulation may function in anxiety disorders as a moderator of the relation between emotion reactivity and anxiety disorder symptoms.

Lastly, research is also demonstrating that self-reported emotion regulation explains incremental variance in anxiety disorder symptoms among children and adolescents. Suveg and Zeman (2004) found that children diagnosed with anxiety disorders self-reported less efficacy in controlling negative emotions relative to control children, and mother's of children diagnosed with anxiety disorders perceived their children as more emotionally inflexible. One recent study extends this literature to a more ecologically valid computerized assessment of emotional reactivity and emotion regulation. Carthy and colleagues (2010, this issue) presented a variety of ambiguous scenarios to children diagnosed with anxiety disorders and children without anxiety disorders. In one block, children read the scenarios, rated how negative they would feel in the scenario, and described what they would do to calm down in that scenario. In a second block, re-appraisal was explained to the children before again presenting the ambiguous scenarios, asking the children to re-appraise the scenario, and asked the children to rate whether re-appraisal would help improve their feelings. Results demonstrated greater self-reported negative emotionality in response to the tasks among the anxious compared to the non-anxious children. Moreover, anxious children reported using more avoidance and help seeking behaviors and less re-appraisal in response to the scenarios and perceived re-appraisal as a less effective emotion regulation strategy compared to non-anxious children. While these studies are limited to verbal-report indices, they suggest that emotion regulation deficits characterize children with anxiety disorders.

One specific area in the child and adolescent literature where the importance of emotion regulation is emerging is in the study of anxiety control beliefs. Anxiety control beliefs refer to one's perceived ability to control negative emotional and bodily reactions (Rapee et al., 1996), and this construct is considered central to understanding the development of anxiety disorders (Barlow, 2002). Anxiety control beliefs are related to emotion regulation, such that both constructs pertain to actual or perceived ability to influence one's emotional reactions (Weems & Silverman, 2006). Weems and colleagues (2003) found that children and adolescents diagnosed with anxiety disorders had significantly lower perceived ability to control anxious reactions compared to control children and adolescents. Further, anxiety

control beliefs significantly discriminated between youth with and without anxiety diagnoses when controlling for both self-reported anxiety and locus of control. Weems and colleagues (2007) later found that anxiety control beliefs predicted unique variance in both child-reported and parent-reported anxiety symptoms after controlling for both anxiety sensitivity and cognitive errors. Ginsburg and colleagues (2004) similarly found that anxiety control beliefs predicted unique variance in panic disorder symptoms among adolescents after controlling for anxiety sensitivity. While these data are limited to self-report indices of anxiety control beliefs, Hogendoorn and colleagues (2008) recently found that self-reported anxiety control beliefs and indirectly measured anxiety control beliefs (measured via an adaptation of the Implicit Association Task) both predicted children's self-reported anxiety. Finally, recent evidence suggests that cognitive-behavioral treatment for childhood anxiety disorders may work through increasing anxiety control beliefs. Muris and colleagues (2009) found that increases in perceived ability to control anxious responding from pre-post treatment significantly predicted reductions in social phobia and GAD symptoms from pre-post treatment when also controlling for changes in negative automatic thoughts. These data further suggest that anxiety control beliefs, and emotion regulation generally, are important in understanding childhood and adolescent anxiety disorders.

The studies reviewed in this section primarily use correlational designs with self-report indices. It is important to note the relevant limitations of this basic design. First, the correlational nature of the data cannot rule out the alternative hypothesis that elevated symptoms of anxiety disorders causes deficits in emotion regulation, as opposed to vice versa. This concern can be assuaged by examining data from studies using an experimental design and finding that emotion regulation techniques can cause changes in emotional responding (e.g., Gross et al., 1993; 1997 e.g., Gross et al., 1998); however, it has not yet been empirically demonstrated that long-term use of particular regulation strategies causes long-term increases in anxiety disorder symptoms. Second, there are numerous constructs posited among the varying self-report measures of emotion-regulation. There has been a paucity of psychometric research examining the structure of emotion regulation, thus it is currently unclear whether these varying constructs are unique versus overlapping versus redundant. For example, the Difficulties in Emotion Regulation Scale (Gratz & Roemer, 2004) has an 'emotional awareness' and an 'emotional clarity' subscale that was supported by an exploratory factor analysis, but it may be the case that these two scales are tapping the same construct (e.g., 'emotional understanding'). Similarly, it is assumed for the purposes of the present paper that the different emotion regulation scales used across the studies are measuring the same 'emotion regulation' construct and that results can be directly compared across studies. It will be important for future research to empirically demonstrate that the Acceptance and Action Questionnaire (Hayes et al., 2004), Anxiety Control Questionnaire, Difficulty in Emotion Regulation Scale (Gratz & Roemer, 2004), Emotion Regulation Questionnaire (Gross & John, 2004), etc. all correlate with each other and demonstrate similar relations with measures of anxiety disorder symptoms. It also remains to be seen whether self-report measures of emotion regulation actually predict habitual use of the intended emotion regulation strategies. Finally, this section focused broadly on the evidence suggesting that emotion regulation explains incremental variance in anxiety disorder symptoms, but future research is necessary to determine whether emotion regulation differentially modulates symptoms of the varying anxiety disorders (e.g., is emotion regulation as relevant for understanding OCD as it is for understanding GAD?).

Summary

Data reviewed in this section suggest that maladaptive patterns of emotion regulation characterize individuals with anxiety disorders. Moreover, difficulties in emotion regulation remain significantly related to anxiety disorder symptoms even when emotional reactivity constructs, such as general anxiety and depression, are controlled (Ginsburg et al., 2004; Menin

et al., 2005; Tull et al., 2007; Weems et al., 2003; 2005). Emotion regulation may also amplify the effect of emotional reactivity on anxiety disorder symptoms (Cisler et al., in press; Kashdan et al., 2008; Kashdan & Steger, 2006) or may involve behaviors that directly increase risk of problem development (e.g., Breslau & Klein, 1999; van der Velden et al., 2008). Accordingly, these data compliment experimental research (e.g., Gross, 1998b) demonstrating that emotion regulation techniques can augment or diminish fear/anxiety and related psychopathology.

Theoretical Model

Based on the available evidence, it is possible to construct a testable model by which emotion regulation may function in the development and maintenance of anxiety disorders. Prior theory posits that dispositional factors, such as inhibited temperament and/or a tendency to respond with negative affect, may potentiate the effect of later specific learning pathways on the development of anxiety disorders (Barlow, 2002; Mineka & Zinbarg, 1996, 2006). For example, an individual high in negative affect would be more likely to develop a phobia of dogs after being bitten by a dog relative to an individual low in negative affect who is bitten by a dog. The two main pieces of these models (i.e., dispositional factors; specific learning pathways) both explain emotional reactivity; that is, they explain possible processes that result in an individual's heightened emotional reactivity either generally (i.e., negative affect) or towards specific stimuli (i.e., specific learning pathways).

Consistent with these well-supported theories of anxiety disorder development, we propose that emotion regulation functions in the development of anxiety disorders during the post-conditioning phase (i.e., after initial fear/anxiety learning). Emotion regulation may not be involved in the initial acquisition of either general fear/anxiety or fear/anxiety towards specific cues. This is consistent with neurobehavioral research demonstrating that the PFC may not be involved in fear learning, but is involved in fear extinction (e.g., Morgan et al., 2003; Myers & Davis, 2007; Phelps et al., 2004; Quirk et al., 2000). We posit that individual differences in patterns of emotion regulation moderate the consequences of fear-conditioning processes. This suggestion is derived from the research reviewed above demonstrating that 1) emotion regulation technique moderates emotional responding in laboratory-based experiments, and 2) maladaptive patterns of emotion regulation explain incremental variance in anxiety disorder symptoms above and beyond measures of negative affect. The following derivative model proposes two temporally distinct processes by which emotion regulation may lead to the development and maintenance of anxiety disorders.

The first process (see Figure 1) pertains to the effect of emotion regulation strategies on 'online fear,' that is, the immediate degree of fear experienced during a single encounter with the conditioned stimuli. This is consistent with research reviewed above demonstrating that emotion regulation techniques can causally influence acute emotional responding. Emotion regulation strategies involving suppression (either behavioral or emotional) or negative reappraisal are predicted to augment (i.e., moderate) the behavioral, physiological, and cognitive outputs of the fear response upon re-encountering the conditioned stimuli. Heightened online fear outputs during re-encounters with the fear cues are proposed to have two immediate consequences. First, heightened online fear when re-encountering conditioned stimuli may then function as 're-conditioning' events, whereby negative expectancies are reinforced and the individual learns again that the fear cues are indeed signals for danger. Second, heightened online fear may strongly motivate avoidance, thus preventing an opportunity for inhibitory learning (cf., Craske et al., 2008;Foa & Kozak, 1986) during which the individual would learn that despite increased fear, the conditioned stimulus is actually not a signal for danger. It is further proposed that these two immediate consequences are routes by which a single event (i.e., re-encountering a conditioned stimulus) contributes to the maintenance, as opposed to the weakening, of fear. Pre-existing (i.e., pre-conditioning)

individual differences in patterns of emotion regulation will likely predict the short-term use of emotion regulation strategies postconditioning, and thus will also predict the maintenance or weakening of fear during post-conditioning encounters with the conditioning stimuli.

The second process (see Figure 2) proposed in this model pertains to the long-term consequences of emotion regulation strategies. This suggestion is consistent with research reviewed above demonstrating that individuals with anxiety disorders appear to be characterized by relatively stable maladaptive patterns of emotion regulation. Heightened online fear as described in the first process is not problematic *per se*. That is, a single distressing re-encounter with a conditioned stimulus would not be a sufficient criterion for any anxiety disorder. In contrast, if emotion regulation strategies involving suppression and negative re-appraisal are used chronically and in an inflexible manner, then there may be two main consequences. First, this pattern of responding would result in increased online fear occurring across encounters with conditioned stimuli and accordingly motivate excessive avoidance of these situations, thus maintaining the fear across time. Second, chronic avoidance as a result of these strategies would result in the functional impairment that typically defines anxiety disorders, such as impaired work performance and social relationships, and chronically enhanced online fear may result in the impairing degree of distress typically reported in individuals suffering from these disorders. Again, pre-existing individual differences in emotion regulation will likely predict the long-term use of emotion regulation strategies post conditioning, and thus will likely predict the onset of disorder.

This model is derived from the research reviewed above and it will be important for future research to test the predictions suggested in the model in an a priori manner.

Future Directions

The body of research discussed above demonstrates that emotion regulation techniques can augment or diminish fear at both the cognitive-behavioral and neural levels of analysis. Additionally, measures of emotion regulation explain incremental variance in anxiety disorder symptoms above and beyond measures of emotional reactivity. These data suggest that theories of anxiety disorders should not only account for how heightened anxiety/fear is acquired, but should also account for how individuals regulate these emotions. This body of research is new, and in keeping with the tradition of an empirical approach to understanding anxiety disorders, only more data will determine the ultimate utility of incorporating emotion regulation into theories of anxiety disorders. Empirically examining the following considerations may facilitate this process.

1. How do different emotion regulation strategies affect emotion, and do they differentially affect different emotions? Gross's (1998a, 2001a, 2007) model of emotion regulation specifies several different emotion regulation topographies. Only two of these (i.e., re-appraisal and suppression) have received substantial empirical attention. It remains to be seen whether other types of emotion regulation techniques affect emotion in manners similar to re-appraisal and suppression. Additionally, it remains to be seen whether the emotion regulation strategies are more or less effective depending on the emotion (e.g., disgust versus fear) regulated. In a related vein, most research has focused on conscious emotion regulation, but emerging work is demonstrating that emotion regulation often happens automatically (Mauss et al., 2007). It will be necessary to test whether automatically versus strategically applied emotion regulation attempts have similar emotional consequences. Future research along these lines may help determine just how far-reaching, or limited, are the implications of emotion regulation for anxiety disorder development and maintenance.

2. Examine the temporal consequences of the different emotion regulation strategies. The body of data reviewed above largely suggests that during a short-period of time in the context of a laboratory experiment, re-appraisal is an effective way to reduce emotional responding and suppression and negative re-appraisal is an ineffective way to reduce emotional responding. There is surprisingly little data regarding the long-term use of these strategies. On the one hand, some research (Gross & John, 2003; Moore et al., 2008; also see John & Gross, 2004 for a review) found that habitual use of re-appraisal, as indexed via a self-report measure of emotion regulation, is positively correlated with the experience of positive emotion, better social outcomes, and greater well-being, and negatively correlated with the experience of negative emotion. Habitual use of expressive suppression, however, is negatively correlated with the experience of positive emotion, social outcomes, and well-being, and positively correlated with the experience of negative emotion. This line of evidence suggests that the short-term consequences of these strategies found in the laboratory settings map onto the long-term consequences of using these strategies in the real world. On the other hand, some theories (Acceptance and Commitment Therapy; Hayes et al., 1999) would predict that it is not the emotion regulation strategy itself that predicts poor outcome, but whether emotion regulation strategies are used inflexibly and insensitively across contexts that predicts poor outcome. In line with this theory, Bonanno and colleagues (2004) found that the ability to alternate between enhancing emotional expression and suppressing emotional expression in a laboratory setting predicted greater decreases in distress ratings of New York college students up to 2 years following the September 11th terrorist attacks. Accordingly, it may not be how often an individual uses one strategy that is important, but how flexible an individual is in using multiple strategies to regulate emotions that predicts outcome. More experimental evidence is needed investigating the short- and long-term consequences of different emotion regulation strategies and the flexibility to alternate between different strategies.

3. Do measures of emotion regulation prior to the development of anxiety-related psychopathology add incremental predictive utility to measures of anxiety-relevant conditioning in the prospective prediction of anxiety disorder development? Addressing this question will help determine whether emotion regulation facilitates the prediction and explanation of anxiety disorder development. Accordingly, this line of research is essential in testing the theoretical utility of incorporating emotion regulation.

4. Does targeting emotion regulation in treatment lead to increased efficacy? Examining if targeting emotion regulation in treatment (cf., Berking et al., 2008) leads to increased efficacy will be necessary for establishing emotion regulation in theories of anxiety maintenance. Controlled investigations will be an essential step here, and may allow for a test of 1) whether treatments that explicitly target emotion regulation strategies produce better outcomes compared to treatments that do not, and 2) whether a treatment's efficacy is mediated by reductions in emotion regulation. Research along these lines will have important implications for both theory and clinical practice.

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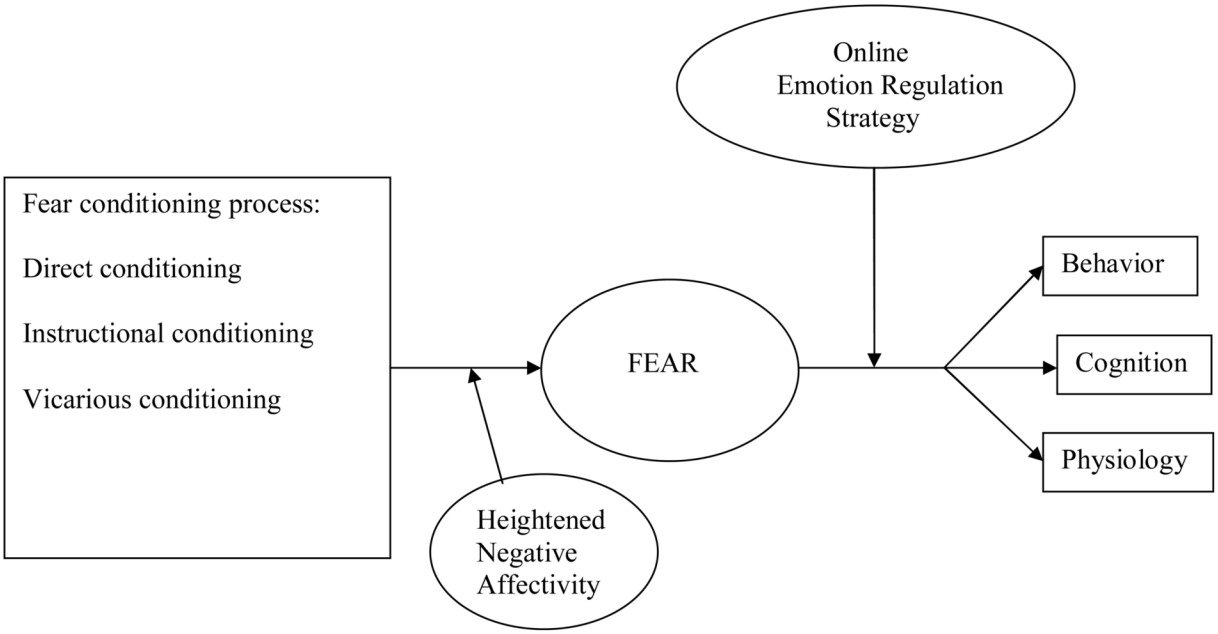

Figure 1.
Emotion regulation strategies may increase the expression of online fear upon re-encountering conditioned stimuli in a given moment.

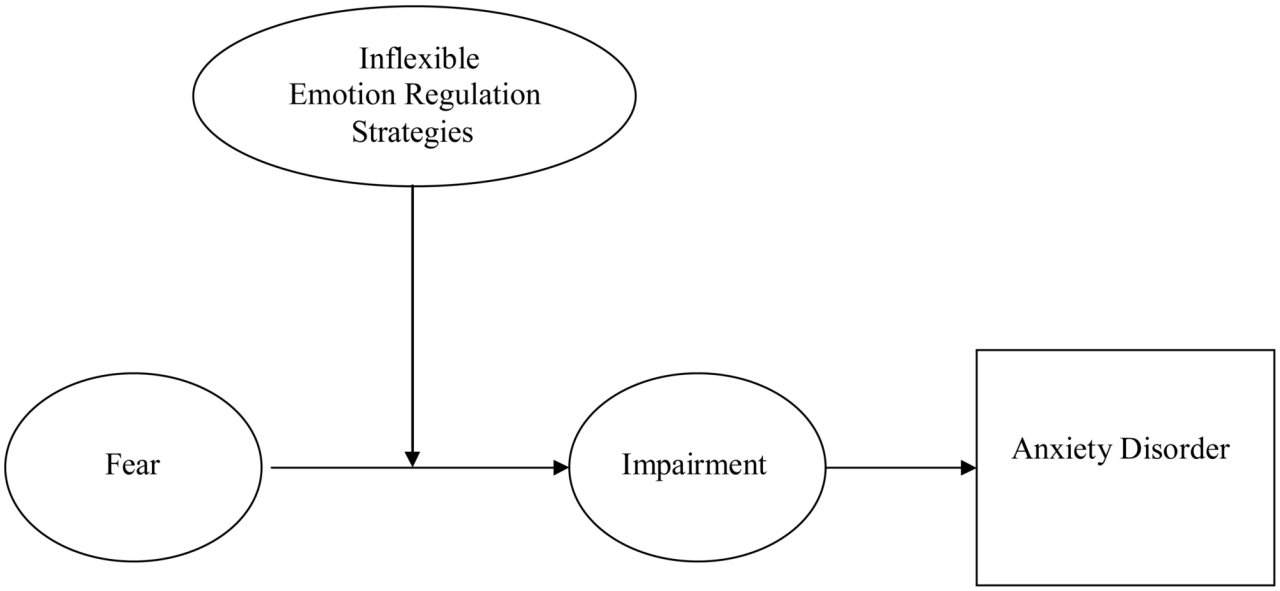

Figure 2.
Inflexible use of emotion regulation strategies over time may lead to the impairment that typically defines an anxiety disorder.