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Affect labeling enhances exposure effectiveness for public speaking anxiety

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ABSTRACT

Exposure is an effective treatment for anxiety but many patients do not respond fully. Affect labeling (labeling emotional experience) attenuates emotional responding. The current project examined whether affect labeling enhances exposure effectiveness in participants with public speaking anxiety. Participants were randomized to exposure with or without affect labeling. Physiological arousal and self-reported fear were assessed before and after exposure and compared between groups. Consistent with hypotheses, participants assigned to Affect Labeling, especially those who used more labels during exposure, showed greater reduction in physiological activation than Control participants. No effect was found for self-report measures. Also, greater emotion regulation deficits at baseline predicted more benefit in physiological arousal from exposure combined with affect labeling than exposure alone. The current research provides evidence that behavioral strategies that target prefrontal-amygdala circuitry can improve treatment effectiveness for anxiety and these effects are particularly pronounced for patients with the greatest deficits in emotion regulation.

Although behavioral treatments for anxiety disorders are highly effective in reducing symptoms of anxiety (Butler, Chapman, Forman, & Beck, 2006; Hofmann & Smits, 2008; Tolin, 2010), many patients do not improve, drop out of treatment, or relapse (Arch & Craske, 2009; Clark et al., 2006; Davidson et al., 2004). Given the need to improve treatments, the goal of the current project is to translate neuroscience research to enhancing the effectiveness of exposure therapy for public speaking anxiety. Specifically, this project compared the effectiveness of exposure alone to exposure plus affect labeling (i.e. putting feelings into words) for individuals with public speaking anxiety.

Public speaking anxiety, a form of social phobia, is one of the most common psychological problems in the United States with prevalence estimates ranging from 11% to 30% of the population (Pollard & Henderson, 1988; Stein, Walker, & Forde, 1996; Wittchen, Stein, & Kessler, 1999). Current treatments for public speaking anxiety combine traditional exposure (e.g., repeated trials of public speaking) with cognitive restructuring in which patients are taught to think about the feared situation neutrally or positively rather than negatively (Heimberg, 2002; Hofmann & Smits, 2008; Hope, Heimberg, Juster, & Turk, 2000; Rapee & Heimberg, 1997). Such treatments aim to reduce anticipatory anxiety, anxiety during speaking, and rumination about the speech after it is over (Clark & Wells, 1995). Although exposure alone appears to be an effective treatment for social anxiety disorder (Feske & Chambless, 1995), to our knowledge, no researchers have used laboratory studies to assess whether adding verbalization (such as cognitive restructuring) to exposure enhances its effects on fear reduction.

Neuroscience research can inform our understanding of anxiety and exposure therapy, and studies on fear learning and anxiety pinpoint the amygdala as central to fear acquisition and responding (Davis, 1992). Activation of prefrontal regions and the strength of connectivity between the prefrontal cortex (PFC) and the amygdala, are essential to successful fear extinction. For example, electrical stimulation of the medial PFC led to reductions of conditioned fear responding in rats (Milad & Quirk, 2002). Greater ventromedial PFC activity is associated with better extinction of conditioned fear in humans (Delgado, Nearing, LeDoux, & Phelps, 2008; Milad et al., 2005; Phelps, Delgado, Nearing, & LeDoux, 2004). Assuming that extinction is a central mechanism of exposure therapy (Craske et al., 2008; Craske, Liao, Brown, & Vervliet, 2012), PFC down-regulation of amygdala may contribute to successful exposure therapy, and strategies that augment such downregulatory pathways may augment outcomes from exposure therapy. In addition,

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evidence suggests that patients with social anxiety disorder have weaker connectivity between the medial orbitofrontal PFC and the amygdala compared to healthy controls (Hahn et al., 2011). Therefore, treatments that strengthen connectivity between prefrontal regions and the amygdala may prove particularly beneficial in the treatment of social anxiety.

Disruption theory of language and emotion (Lieberman, 2003, 2011) posits that labeling one's emotional state can disrupt the experience of that emotional state. However, because intent to reduce distress is not explicit, affect labeling has been conceptualized as an incidental emotion regulation strategy (Burklund, Creswell, Irwin, & Lieberman, 2014), which differs from intentional strategies such as cognitive restructuring or emotional suppression. A number of neuroimaging studies have demonstrated that labeling one's emotional experience activates areas of the PFC, and reduces activation in the amygdala (Gorno-Tempini et al., 2001; Hariri, Bookheimer, & Mazziotta, 2000; Hariri, Mattay, Tessitore, Fera, & Weinberger, 2003; Narumoto et al., 2000). The right ventrolateral PFC is consistently activated during affect labeling (Cunningham, Johnson, Chris, Gore, & Banaji, 2003; Lieberman et al., 2007; Narumoto et al., 2000), and it is presumed that this region downregulates amygdala activation. The principle of neural plasticity states that repetition of a process can increase efficiency and efficacy of that process through changes in neuron function, chemical profile, and structure (Anderson, 2010; Kandel & Schwartz, 1982). Therefore, repeated affect labeling may enhance connectivity in PFC-amygdala pathways in turn improving patients' ability to regulate emotional responses. Additionally, there may be a dose response relationship between the quantity of affect labeling trials and the degree of enhanced connectivity.

In accord with this notion, two studies have demonstrated that affect labeling enhances the effectiveness of exposure. Tabibnia, Lieberman, and Craske (2008) examined the effect of repeated exposure to evocative images with and without negative affective labels. In study 1 with healthy controls, repeated presentation of emotionally evocative images paired with an affect label resulted in greater attenuation of skin conductance responding and heart rate deceleration upon re-presentation of the images without a label at one-week re-test. In study 2, the findings were replicated for skin conductance response in spider-fearful subjects who were exposed to spider images paired with negative labels compared to no labels or neutral labels. Kircanski, Lieberman, and Craske (2012) compared the effects of exposure to a live spider without linguistic processing, with affect labeling, with reappraisal, and with distraction, in spider fearful subjects. At one-week re-test the group that completed exposure with affect-labeling had lower skin conductance responses while viewing a spider and moved closer to the spider compared to the reappraisal and exposure alone groups. In addition, those who used the greatest number of anxiety- and fear-related words during affect labeling showed the greatest reductions in skin conductance responding and moved closest to the spider.

Another consideration we examine in the current study is whether the matching of treatments to individuals may improve therapy outcomes. Two possibilities have been evaluated: individuals with a deficit are more likely to benefit from treatments that target that deficit (compensation), and individuals with a strength will benefit most from a treatment that matches that strength (capitalization; Rude & Rehm, 1991). Recent studies on depression and suicidality treatment have found support for both capitalization (Cheavens, Strunk, Lazarus, & Goldstein, 2012) and compensation (Wingate, Van Orden, JoinerJr., Williams, & David, 2005). Studies examining amygdala activation (McClure et al., 2007), emotional reactivity to evocative images (Niles, Mesri, Burklund, Lieberman, & Craske, 2013), and heart rate variability (Davies, Niles, Pittig, Arch, & Craske, 2015) as predictors of treatment outcome for anxiety patients, support a compensation model, with superior outcomes for patients with greater reactivity at baseline. We aimed to evaluate whether affect labeling would most benefit those with a deficit or with a strength in affect labeling at baseline. The extent to which affect labeling at baseline reduces distress serves as an indicator of incidental emotion regulation capacity, and can be used to determine whether participants with strengths (capitalization) or with deficits (compensation) in emotion regulation benefit more from an intervention augmented with implicit emotion regulation training (i.e., affect labeling).

The current study had three aims. The first aim was to assess whether affect labeling enhanced the effectiveness of exposure compared to exposure alone. We hypothesized that participants instructed to use affect labeling during exposure would show greater attenuation of fear in anticipation of and recovery from public speaking compared to those who completed exposure alone. The second aim was to assess whether the number of anxiety- or fear-related words used during affect labeling predicted greater attenuation of fear responding at re-test. We hypothesized that participants who used more anxiety or fear related words compared to other negative emotion words would show the greatest fear reduction at re-test. The third aim was to assess whether individual differences in incidental emotion regulation (i.e., the extent to which affect labeling reduced distress in a pre-testing session) moderated response to exposure with affect labeling versus exposure alone. Given mixed findings in the literature, these analyses were mainly exploratory, and we made no a priori predictions.

1. Method

1.1. Design

This study used a 2 (Group) × 3 (Time) mixed design with public speaking fearful participants. Groups included exposure combined with affect labeling (AL), and exposure alone (Control). Time included assessment time-points at baseline (Time 1), following exposure (Time 2) and at 1-week follow-up (Time 3).

1.2. Participants

One hundred two participants (AL = 52; Control = 50) were recruited to participate. Two participants assigned to the Control group were not included in analyses: one participant received the incorrect study protocol due to experimenter error and another fell asleep during the experiment. Therefore, the final sample included in analyses was 100. See Fig. 1 for a consort diagram of flow through study procedures. Participants had a mean age of 25 (SD = 9.1), 80% were female, 92% were students, and 37% spoke English as a second language. The ethnic breakdown of the sample was 55% Asian, 16% Hispanic, 14% Caucasian, 6% African American, and 9% other.

Eligible participants reported a 6 or higher on anxiety and a 5 or higher on avoidance of public speaking on a 0 to 8 scale. The prompts for anxiety and avoidance respectively were “How anxious would you feel giving a formal speech before a live audience?” and “How likely would you be to avoid taking a class that required an oral presentation?” Zero indicated no anxiety/never avoid, and 8 indicated extreme anxiety/always avoid. This two question survey has been used to recruit public speaking fearful participants in previous studies (Culver, Stoyanova, & Craske, 2012; Tsao & Craske, 2000). Participants were over 18 years of age, fluent in English, free of heart, neurological, or respiratory conditions, hearing impairment, physician recommendation to avoid stressful situations, current treatment for public speaking anxiety, or psychotropic medication prescription for an emotional problem. Participants