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How Putting Feelings into Words Reduces Our Emotional Experiences: Understanding Mechanisms of Affect Labeling

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How Putting Feelings into Words Reduces Our Emotional Experiences: Understanding Mechanisms of Affect Labeling

A dissertation submitted in partial satisfaction of the requirements for the degree Doctor of Philosophy in Psychology

by

Jared Torre

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ABSTRACT OF THE DISSERTATION

How Putting Feelings into Words
Reduces our Emotional Experiences:
Understanding Mechanisms of Affect Labeling

by

Jared Torre
Doctor of Philosophy in Psychology
University of California, Los Angeles, 2016
Professor Matthew D. Lieberman, Chair

The act of putting feelings into words, or 'affect labeling', can attenuate our negative experiences. Unlike explicit emotion regulation techniques, affect labeling may not even feel like a regulatory process as it occurs. Nevertheless, research investigating affect labeling has found it produces a pattern of effects similar to those seen during explicit emotion regulation, suggesting affect labeling is a form of implicit emotion regulation. However, the mechanisms driving the processes behind affect labeling remain poorly understood and, despite rising interest in converting affect labeling paradigms into clinical interventions, many questions remain about the best way to implement affect labeling in a laboratory setting. This dissertation is the culmination of research that addresses several open questions about affect labeling and suggests improvements for the paradigm moving forward.

The dissertation of Jared Torre is approved.

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University of California, Los Angeles

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To Paul and Marla

For their unwavering support and endless patience

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Presentations

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Torre, J. B., Inagaki, T. K., Mulvenna, C. M., Haltom, K. E. B., Stanton, A. L., & Lieberman, M. D. (April 2012). Common Neural Mechanism for Emotion Regulation and Motor Inhibition. Poster presented at 4th Annual Social & Affective Neuroscience Society Annual Conference: New York, New York.

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Background

When we think about emotion regulation, we likely think of a process that requires effort, whether physical or mental, that 'removes' us in some way from the cause of our emotion. We might avert our eyes from a gruesome car crash or even try convincing ourselves it isn't as bad as it looks. Successful emotion regulation might more easily be thought of as an escape from something that elicits an emotional response in us, eliminating our feelings by avoiding the eliciting stimulus. We probably would not think that focusing on our feelings without trying to change them could achieve the same effect. In fact, emerging evidence in the study of emotion regulation has begun to build a case depicting a surprising kind of emotion regulation: putting feelings into words, an act called 'affect labeling', can itself be a form of implicit emotion regulation. This notion about the benefits of talking about our feelings has existed in various forms including therapy (Esterling, L'Abate, Murray, & Pennebaker, 1999; Greenberg, 2002) and research on expressive writing (Pennebaker & Beall, 1986; Pennebaker, 1993). Only over the past decade has affect labeling been focused upon specifically as a potential form of emotion regulation and tested within the lab.

Talking about our feelings or, in some cases, using emotional language to describe the things that upset us, does not necessarily feel like an exercise in emotion regulation. So how can we know if it is? One way is to see if it actually regulates emotions. When an individual experiences an emotion, it elicits loosely connected responses across experiential, physiological, and behavioral domains (Levenson, 2003; Mauss, Levenson, McCarter, Wilhelm, & Gross, 2005). Emotion regulation is often conceptually defined as a manipulation of the quality, duration, or intensity of an emotion (Gross & Thompson, 2007; Gross, 1998b; Koole & Rothermund, 2011). It stands to reason that any process which modulates these primary

channels of output for an emotion should be considered a kind of emotion regulation. To demonstrate, we turn first to a characterization of the well-studied form of emotion regulation, 'reappraisal'.

Reappraisal is the reinterpretation of an emotionally evocative stimulus in order to alter its emotional impact (Gross, 1998a). In the examples of emotion regulation provided earlier, convincing ourselves a car accident looks worse than it was is an attempt at reappraisal; we have initially appraised the wreck as potentially lethal or injurious to those involved and have reappraised the observed damage as (hopefully) cosmetic. In line with the domains of emotion effects listed above, engagement of reappraisal can alter subjective experience of an emotion as measured through self-report (Blechert, Sheppes, Di Tella, Williams, & Gross, 2012; Kalisch et al., 2005; McRae, Ciesielski, & Gross, 2012; McRae, Jacobs, Ray, John, & Gross, 2012; Ray, McRae, Ochsner, & Gross, 2010), autonomic arousal (Eippert et al., 2007; Kalisch et al., 2005; Kim & Hamann, 2012; McRae, Ciesielski, et al., 2012; Ray et al., 2010; Urry, van Reekum, Johnstone, & Davidson, 2009), and emotion-related behaviors such as overt physical expression of emotion (Gross, 1998a, 2002), reduced risk-taking (Park & Lee, 2011), and reaction times during interpersonal evaluation (Blechert et al., 2012). In the neural domain, several meta-analyses of neural activations during reappraisal (Buhle et al., 2014; Diekhof, Geier, Falkai, & Gruber, 2011; Frank et al., 2014; Kalisch, 2009; Kohn et al., 2014) each identified the following prefrontal regions often associated with cognitive control as more active during emotion regulation via reappraisal: ventrolateral prefrontal cortex (vlPFC), dorsolateral prefrontal cortex (dlPFC), supplementary motor area (SMA), and anterior cingulate cortex (ACC). Meta-analyses that looked at deactivations due to reappraisal also found significant reductions in amygdala activation, a region associated with emotion-generation. Some evidence points specifically to

the inhibitory role of vlPFC in reappraisal (Golkar et al., 2012; Ochsner, Silvers, & Buhle, 2012), emotion regulation (Berkman & Lieberman, 2009; Hooker & Knight, 2006), and self-control more broadly (Cohen, Berkman, & Lieberman, 2012; Cohen & Lieberman, 2010; Tabibnia et al., 2011, 2014) as well as the inverse relationship between vlPFC activity and amygdala activity during reappraisal (Banks, Eddy, Angstadt, Nathan, & Phan, 2007; Ochsner, Bunge, Gross, & Gabrieli, 2002). Finally, appropriate and effective implementation of emotion regulation is often considered adaptive and reappraisal has been linked to positive long-term benefits on health (Hopp, Troy, & Mauss, 2011; McRae, Jacobs, et al., 2012), especially when compared to other less adaptive strategies like suppression (Gross & John, 2003). If affect labeling can demonstrate a similar profile of effects in the same experiential, autonomic, neural, and behavioral domains, then it too should be considered as a form of emotion regulation.

Emotion Regulation Effects of Affect Labeling

Neural

Activity within the limbic system, the amygdala in particular, is often observed as an indication of emotion generation (Lindquist, Wager, Kober, Bliss-Moreau, & Barrett, 2012; Mechias, Etkin, & Kalisch, 2010; Phan, Wager, Taylor, & Liberzon, 2002; Wager, Phan, Liberzon, & Taylor, 2003). According to meta-analytic accounts, reappraisal typically elicits increased activity within a network of prefrontal regions (vlPFC, dlPFC, SMA, and ACC) and decreased limbic activity in the amygdala. As mentioned earlier, the vlPFC in particular is heavily implicated as a major contributor to the deactivation of amygdala activity (Golkar et al., 2012; Ochsner et al., 2012) due in part because of its role in a wide variety of self-control processes (Berkman & Lieberman, 2009; Cohen et al., 2012; Cohen & Lieberman, 2010;

Tabibnia et al., 2014) and also because of the observed inverse connectivity between vlPFC and amygdala during reappraisal (Banks et al., 2007; Ochsner et al., 2002).

Affect labeling, when studied in the lab, typically uses the following task paradigm: participants are shown emotionally evocative images, one at a time, with a selection of affect labels underneath. They are instructed beforehand to select one of the emotion words using a response box or keyboard to indicate the correct or most appropriate label that describes the image depicted. In some studies, emotionally evocative images of faces conveying an emotional expression are used (negative expressions such as anger, fear, etc.) and participants are instructed to select the label that best describes the emotional expression depicted (e.g. 'angry', 'scared', etc.; see Figure 1.1A). These trials are often compared against control conditions that require participants to instead either 1) choose the appropriately gendered name (e.g., 'Helen' or 'Steve') presented that matches the gender of the expressive face instead of the affect ('gender label') or 2) match the emotion of the expressive face with the corresponding face below expressing the same emotion ('affect match'). In other studies, highly aversive images of scenes depicting gore, acts of aggression, crying individuals, and so on, are used. Affect labeling trials using images of this type of this type are typically contrasted against passive observation of similarly aversive images (see Figure 1.1B). Importantly, viewing either type of stimulus (expressive faces or aversive scenes) has been shown to induce amygdala activity (Britton, Taylor, Sudheimer, & Liberzon, 2006). Affect labeling elicits increased vlPFC and decreased amygdala activity compared to gender labeling (Burklund, Craske, Taylor, & Lieberman, 2015; Lieberman et al., 2007; S. E. Taylor, Eisenberger, Saxbe, Lehman, & Lieberman, 2006), affect matching (Hariri, Bookheimer, & Mazziotta, 2000; Payer, Baicy, Lieberman, & London, 2012; Payer, Lieberman, & London, 2011) or passive viewing of expressive faces (S. F. Taylor, Phan, Decker, &

Liberzon, 2003), and compared to passive observation of aversive scenes (Burklund, Creswell, Irwin, & Lieberman, 2014). In fact, a meta-analysis of amygdala activity across a variety of tasks reported labeling emotions present within an evocative stimulus yields significantly decreased odds of amygdala activity relative to passively viewing those stimuli (Costafreda, Brammer, David, & Fu, 2008). Moreover, incidental processing of affect, as opposed to explicit processing through labeling, was neither more nor less likely to produce signal in the amygdala than passive viewing. One study recently reported patients with brain lesions were significantly impaired in their ability to track the emotional state of a film character using a dial with labels from 'extremely negative' to 'extremely positive' to the extent they had a damaged right vlPFC (Goodkind et al., 2012) suggesting the necessary involvement of vlPFC in the affect labeling process.

Many studies have additionally reported negative connectivity between vlPFC and amygdala such that as activity in vlPFC increases during affect labeling amygdala activity decreases, suggesting the two regions are in communication during affect labeling (Foland et al., 2008; Hariri et al., 2000; Lieberman et al., 2007; Payer et al., 2012, 2011; S. E. Taylor et al., 2006). Importantly, dynamic causal modeling was recently used to identify the directionality of this relationship in affect labeling; rather than decreased amygdala activity driving the relationship, increased output from vlPFC (and not other prefrontal regions) was identified as the cause of decreased amygdala activity during affect labeling further suggesting the role of vlPFC in the down-regulation of amygdala responsiveness (Torrisi, Lieberman, Bookheimer, & Altshuler, 2013).

Much like reappraisal, affect labeling is dependent upon the activation of prefrontal control regions and the diminished activity of emotion-generation regions, in particular the

amygdala. Regardless of the stimulus type (e.g., faces or aversive scenes) and regardless of the matched control condition (e.g., gender labeling, affect matching, or viewing aversive stimuli), activation within these prefrontal regions, vlPFC in particular, is associated with successful implementation of affect labeling and inhibition of the amygdala.

Experiential

The simplest way to measure the emotional experience of an individual is to ask them. Though we are not directly accessing the emotional state, through subjects' self-reports we can still acquire a measurable understanding of the experience (Barrett, 2004). It is admittedly difficult to fully justify the use self-reported emotional experience as a primary outcome of interest when the essence of what you are studying is the verbalization of emotional experience; it is unknown how using self-report as an outcome to compare affect labeling with a control condition is itself an affect labeling manipulation that contaminates the effects. Despite this difficulty, many studies have demonstrated effects on self-report: when participants apply affect labeling to emotionally charged stimuli, they tend to report diminished levels of affect compared to conditions that do not engage affect labeling related processing during stimulus presentation.

In a series of four studies (Lieberman, Inagaki, Tabibnia, & Crockett, 2011), participants were shown emotionally evocative images and, in addition to a simple 'view' control condition, were instructed to engage in either affect labeling (study 1), affect labeling and reappraisal (studies 2 and 4), or affect labeling and distraction (study 3). During the affect labeling condition, participants would choose between two emotionally charged words below the evocative image. Although both words shared the same valence as the image (e.g., negative words for a negative image), only one word presented was related to the emotional content of the scene depicted. For example, an image depicting a sickly man in a hospital bed with a painful

expression would appear alongside the words ‘anguish’ and ‘bomb’ with the former being the correct affect label to choose in this instance (for another example, see Figure 1.1B). Negatively valenced and arousing images from the International Affective Picture System (IAPS; Lang, Bradley, & Cuthbert, 2008) were used in the first three studies and participants rated the distress they felt on a 7-point scale after either viewing or regulating in each trial. In the fourth study, positive IAPS images were used and participants instead rated how ‘pleasant’ they felt after each trial. Regardless of valence, results from each study demonstrated affect labeling significantly reduced affect reported by participants after each trial compared to viewing the stimuli without regulation. Interestingly, distress reduction from affect labeling was positively correlated within participants with distress reduction from reappraisal, suggesting a common underlying mechanism driving the affect attenuation across these processes.

These regulatory effects of affect labeling on self-report have been replicated in several other studies. In an fMRI study, Burklund et al. (2014) also used negative IAPS images and measured the effects of affect labeling, reappraising, and viewing these stimuli on self-reported distress. In this case, affect labels presented beneath images described simple emotion states (e.g., ‘angry’, ‘anxious’, etc.) and subjects were asked to choose the label that best represented their own emotional reaction to the image presented. Each trial was also presented with the option ‘other’ to avoid constraining participants to the two labels provided. As before, self-reported distress showed significant reductions for both affect labeling and reappraisal conditions compared to viewing aversive images and, as before, these reductions showed significant correlation across the two conditions.

An interesting line of work on affect labeling investigates effects on affective responses to stimuli and resultant increases in self-reported physical symptoms that stem from viewing

highly aversive images (Constantinou, Bogaerts, Van Diest, & Van den Bergh, 2013). Affect labeling, in this case choosing provided labels that best described the emotion depicted within the image presented, significantly reduced 1) experienced negative valence and arousal from aversive images, 2) experienced positive valence within pleasant images, and 3) self-reported physical symptoms after negative images (Constantinou et al., 2015; Constantinou, Van Den Houte, Bogaerts, Van Diest, & Van den Bergh, 2014).

In another fMRI study (S. F. Taylor et al., 2003), participants were instructed to either attend to emotionally salient images or to rate the stimuli as 'pleasant', 'neutral', or 'unpleasant'. After each condition, participants rated how much of certain emotions they felt at the time and rated significantly decreased amounts of feeling 'sad' after the 'rating' condition than the 'attend' condition. However, no differences were observed across conditions in this study for ratings of being 'disgusted', 'shocked', 'upset', or 'disturbed'.

Though this may not always be the case (c.f. Matejka et al., 2013), within the domain of subjective experience of emotion affect labeling can diminish feelings of both positive and negative affect and that these reductions within individuals correlate strongly with similar reductions from reappraisal.

Autonomic

Although there is no consensus on precisely what profiles of specific emotions look like in the autonomic domain, it is generally accepted that the experience of emotional events produces a measurable autonomic signal (Ekman, Levenson, & Friesen, 1983; Kragel & LaBar, 2014; Kreibig, 2010; Levenson, 2003) that is tethered to other measures of emotional reactance (Daubenmier, Hayden, Chang, & Epel, 2014; Heller, Lapate, Mayer, & Davidson, 2014; Mauss

et al., 2005; Yang et al., 2007) and can be altered via emotion regulation processes (Gross, 2015).

In several cases, affect labeling produces an immediate reduction of autonomic responses to an emotional event. For instance, when comparing the application of subjective affect labels (i.e. words describing one's own emotional state) against objective affect labels (i.e. words describing the eliciting stimulus) to aversive images, skin conductance responses showed more reduction during the application of objective affect labels (McRae, Taitano, & Lane, 2010). In another study, after emotional induction, participants who were asked to report on their currently felt anger, compared to participants who were not instructed to self-report, demonstrated an autonomic profile demonstrating reduced emotional reactivity including decreased heart rate, decreased cardiac output, and increased total peripheral resistance (Kassam & Mendes, 2013) which is suggestive of movement away from a state of anger (Mendes, Major, McCoy, & Blascovich, 2008). Moreover, compared to stating facts about an experience, verbalizing emotional experience decreased skin conductance responses and voice pitch, indicating lower arousal (Matejka et al., 2013).

In other cases, autonomic effects of affect labeling are not immediately evident but exhibit a longer-term delayed effect, particularly in clinical applications of affect labeling. In one such instance, participants viewed an aversive film and were instructed to talk about either their emotional reactions or the film's sequence of events. Participants demonstrated increased physiological responses (lower skin temperature and higher skin conductance) despite no change in self-reported emotional reactance while talking about their emotional experience, but showed reduced physiological responses and increased self-reported positive affect 48 hours later when

viewing the film a second time compared to participants who were only instructed to talk about the film's sequence of events (Mendolia & Kleck, 1993).

Turning to clinical applications of affect labeling, both healthy individuals and those with a moderate to severe fear of spiders were shown images of spiders and demonstrated decreased skin conductance eight days later when shown these same stimuli but only when images were initially presented with negative word labels (Tabibnia, Lieberman, & Craske, 2008). Interestingly, for the individuals with a fear of spiders, this affect labeling effect generalized to novel images of spiders. Similarly, patients with clinically diagnosed arachnophobia who engaged in affect labeling during an initial session with a live, caged tarantula in the room with them demonstrated greater decreases in skin conductance response during a second session one week later compared to patients who engaged in distraction, reappraisal, or mere exposure alone (Kircanski, Lieberman, & Craske, 2012). Importantly, for patients assigned to the affect labeling condition, the more negative affect words used the greater the reduction in skin conductance as well as the more steps in the exposure therapy achieved (e.g., moving closer to the spider, opening the cage, touching the spider directly, etc.) during the second session one week later. Finally, in a study investigating the effects of affect labeling in patients with public speaking anxiety (Niles, Craske, Lieberman, & Hur, 2015), combining affect labeling with exposure produced greater reductions in skin conductance responses over the course of the eight day procedure compared to exposure alone. Interestingly, patients who used more affect labels had greater reductions in skin conductance responses and fewer non-specific skin conductance responses during anticipation of giving a speech during the final session. Further, patients who demonstrated larger deficits in affect labeling at baseline (smaller decreases in self-reported

distress in a laboratory affect labeling task) demonstrated greater reductions in skin conductance as a result of the affect labeling training.

Similar to reappraisal, we have seen that affect labeling can have diminishing effects on autonomic activity in a variety of ways. Applying affect labels to stimuli external to ourselves has a greater effect on reducing skin conductance than applying similar labels to our own internal emotional experience. Compared to either not reporting on our feelings or making statements of facts instead of our emotional experience, affect labeling has also shown immediate reduced autonomic activity in the moment. And finally, talking about or labeling our emotional experiences has shown a delayed longer-term effect on autonomic activity that can last as long as a week and perhaps longer.

Behavioral

Emotions are functional and often prepare us both mentally and physically to take certain actions (Frijda, 1986; Levenson, 1999). If emotional states are altered, then we would expect downstream behavioral effects which arise from these emotions to be altered as well. Several studies have demonstrated that engagement of affect labeling can alter the output of an emotion-related behavior. As mentioned earlier, patients with a clinical fear of spiders were more likely to proceed further along in the exposure therapy (e.g., moving physically closer to the spider) when they engaged in affect labeling compared to reappraisal, distraction, or exposure alone (Kircanski et al., 2012) and the number of affect words generated by patients correlated with increased steps taken during the exposure process. In another study already mentioned (Mendolia & Kleck, 1993), judges rated participants who talked about their emotions after watching an aversive film were rated as exhibiting less difficulty describing their reactions to film during the second viewing 48 hours later as well as displaying less tension while talking

about their emotions then did subjects who were instructed to only discuss the film's sequence of events. Subjects who were part of a pair of fMRI studies investigating affect labeling demonstrated less aggression towards others during a noxious noise task to the extent that affect labeling reduced amygdala activity (Payer et al., 2012, 2011). Adolescent girls who were part of an electronic diary study measuring their emotions were rated as demonstrating reduced levels of anxiety by their parents to the extent that they use the electronic diary to log their emotional states, and thus engage in affect labeling, more (Thomassin, Morelen, & Suveg, 2012). And finally, students who wrote about their test-related anxieties before taking a math test performed significantly better than students who either sat quietly or wrote about something unrelated for the same amount of time (Ramirez & Beilock, 2011). This effect was especially present for students with a high amount of test anxiety, likely as they had the most amount of disruptive affect to reduce through the intervention.

There are many behaviors that can be elicited or altered by our emotional states and changing these emotions ought to change the resultant behaviors as well. Though this domain of effects is not the most well-studied in the context of affect labeling, as is the case with reappraisal, changes in downstream behavioral effects as a result of engaging affect labeling have been observed in approaching an aversive stimulus, body signals displaying distress, tension, or anxiety, aggressive behaviors, and test taking performance.

Health Implications

Although not a central characteristic of emotion regulation, given the importance of successful emotion regulation in healthy living, we might expect to see a relationship between effective use of affect labeling and positive health outcomes as well as poor affect labeling ability and health deficiencies in physical and mental domains. Much like reappraisal (Chen &

Miller, 2014; Gross & John, 2003), strong affect labeling ability also has several connections to the health literature that implicate affect labeling as an important part of physical and mental health.

Nearly three decades of research on expressive writing has demonstrated that writing about emotional experiences confers significant and long-lasting physical and mental health benefits (Frattaroli, 2006; Frisina, Borod, & Lepore, 2004; Smyth, 1998). Affect labeling has been implicated as a major mechanism through which expressive writing confers these health benefits (Stanton & Low, 2012). More frequent usage of negative emotion words (i.e. affect labels) in expressive writing essays reduced physical symptoms in cancer patients as long as three-months after the intervention (Low, Stanton, & Danoff-Burg, 2006) while another study showed a greater use of emotion words during an expressive writing intervention mediated the relationship between constructive processing and decreased depressive symptoms also up to three-months later (Hoyt, Austenfeld, & Stanton, 2014).

Interestingly, chronic use of affect labeling is also related to greater health. Individuals who have a greater tendency to employ affect labeling in their daily lives show a reduced relationship between distress and morning levels of the stress-related hormone cortisol (Daubenmier et al., 2014).

Several studies have also linked poor affect labeling ability with mental health. In the neuroimaging literature, bipolar patients performing affect labeling in an fMRI scanner were shown to have decreased vlPFC (Foland-Ross et al., 2012) and increased amygdala activity compared to health controls as well as a reduction in the typical negative connectivity between vlPFC and amygdala as a function of bipolar severity (Foland et al., 2008). This pattern is nearly identical to the pattern of neural activations in bipolar patients performing reappraisal (Townsend

et al., 2013). Bipolar patients also exhibit increased N170 wave amplitude and high frequency heart rate variability during affect labeling compared to healthy controls that were reduced towards normal, healthy levels after a mindfulness based cognitive-behavior therapy (Howells, Rauch, Ives-Deliperi, Horn, & Stein, 2014). In one study of social anxiety (Burklund et al., 2015), patients and healthy controls were both administered the affect labeling paradigm with expressive faces in an MR scanner. Healthy controls demonstrated the typical profile of neural effects including increased vlPFC, decreased amygdala, and negative connectivity between the two, whereas patients with social anxiety instead showed increased amygdala activity during affect labeling and no significant connectivity between vlPFC and amygdala. In another study (S. E. Taylor et al., 2006), individuals who come from risky families, which puts them at risk for mental health issues (Repetti, Taylor, & Seeman, 2002), have demonstrated positive connectivity between vlPFC and amygdala during affect labeling. Finally, symptoms of alexithymia, an inability to identify and express one's feelings that is linked to poorer mental health (Samur et al., 2013), have been shown to negatively correlate with vlPFC activation during affect labeling (Payer et al., 2011).

Affect Labeling as Implicit Emotion Regulation

Thus far, we have seen that regardless of domain, affect labeling has demonstrated a profile of regulatory effects that very closely resembles that of more widely accepted forms of emotion regulation, reappraisal in particular. Experientially, both positive and negative self-reported affect were reduced when engaging in affect labeling compared to viewing aversive stimuli. Autonomic effects were also diminished when talking or labeling emotional states. In some cases, the effects were immediate while in others they observed a delayed time-course with

longer-term effects lasting at least one week after affect labeling was engaged. An extremely similar profile of neural regions involved in reappraisal effects are likewise involved in affect labeling: increased prefrontal control regions, in particular vlPFC, decreased amygdala activity suggesting a reduction in emotion-generative processes, and an inverse relationship between vlPFC activity and amygdala such that the vlPFC is brought online to down-regulate the amygdala. Behaviorally, evidence within very different contexts, from the laboratory to the classroom, has demonstrated that affect labeling can alter a range of emotion-related behaviors in desirable ways. Finally, affect labeling contributes to good physical and mental health and has been demonstrated as absent or deficient in poor health. Together, these findings suggest that affect labeling is indeed a form of emotion regulation (see Figure 1.2 for a conceptual summary). What makes it an implicit is how it is typically perceived as an effective emotion regulation strategy.

One of the more interesting features of affect labeling is the mistaken lay theory most people hold about how affect labeling affects distress. In work already discussed earlier investigating reductions in self-reported distress due to affect labeling (Lieberman et al., 2011), a subset of the participants were not shown aversive images and instead had 'attend' and 'affect label' trials described rather than shown to them. Participants were then asked to predict how much distress they would experience if shown the actual 'attend' and 'affect label' trials.

Participants predicted that affect labeling would produce *greater* distress than simply attending to the same image. In contrast, participants predicted that reappraisal would produce *lower* distress than affect labeling or attending to an aversive image. Importantly, this pattern of predicted distress holds even for participants who, just before becoming predictors, went through the entire affect labeling paradigm as experiencers and actually reported significantly *reduced*

distress for trials when affect labeling was engaged. Predictors had no trouble accurately predicting the direction of effects on distress for reappraisal or distraction, however, the predicted regulatory effects of affect labeling on distress were not just underestimated, they were reversed. People seem unaware of the regulatory effects of affect labeling and lay theories of strategy-dependent emotion regulation efficacy are so strong as to countermand their own experiences with it.

This impressive failure to accurately predict even the direction of regulatory effects from affect labeling has an interesting implication for the placement of affect labeling within the larger emotion regulation literature. Along with engagement of regulatory processes and an impact on emotional output, activation of a regulatory goal has been described in the past as a core feature of emotion regulation (Gross, 2014). However, as described above, individuals seem to have very powerful lay theories about how affect labeling ought to manipulate their emotional state, though it instead does the opposite, making it unlikely that individuals would have a conscious goal to engage affect labeling in order to down regulate their emotional reaction considering they strongly believe it does the opposite. The lack of a conscious goal suggests affect labeling may be a form of implicit emotion regulation.

A defining characteristic of implicit emotion regulation is that it does not require conscious supervision of explicit intention yet still alters an emotional experience (Koole & Rothermund, 2011), which we have seen fits the description of affect labeling well. Implicit forms of emotion regulation are also thought not to require effort to be engaged as is the case with habituation, fear extinction, or emotional conflict adaptation (Gyurak, Gross, & Etkin, 2011). Here, affect labeling presents as a peculiar case of implicit emotion regulation because although it does not require intent to regulate an emotional experience in order for it to be

effective, it is not effortless and requires a conscious conversion of either the internal emotional experience or the external evocative stimulus into a linguistic symbol. In this way, affect labeling might feel like an explicit process because of the effort required, but its counterintuitive effects and operation as a regulatory process without conscious awareness suggests it should be considered a form of implicit emotion regulation instead (Gyurak & Etkin, 2014).

Possible Mechanisms of Affect Labeling

Despite the many findings on affect labeling discussed earlier, the basic mechanisms enabling affect labeling and its function as a form of implicit emotion regulation remain poorly understood. In this section, we will outline a few possible candidates and discuss their merit as underlying mechanisms of affect labeling.

Distraction

A simple claim made about affect labeling could be that it operates via distraction; requiring application of language to an evocative stimulus momentarily distracts us from fully processing and engaging the stimulus as we would have otherwise and thus shows diminished effects. This account seems reasonable considering a small amount of evidence comparing affect labeling to distraction. Regulatory effects on self-reported distress of affect labeling and distraction did not differ significantly when compared directly (Lieberman et al., 2011). Additionally, successful distraction depends upon a similar profile of neural mechanisms as both reappraisal and affect labeling such as the vlPFC, SMA, and ACC and yields reduced amygdala activity as well (McRae, Hughes, et al., 2010). However, for several reasons, this account seems unlikely. Many studies have compared affect labeling to gender labeling, another condition which requires a similar amount of distraction from the stimulus by applying labels but which

does not show the same regulatory effects as affect labeling. If affect labeling did not engage regulatory processes, we might even expect to see higher activation of amygdala rather than less with the inclusion of emotion words given that they have been shown to activate the amygdala (Straube, Sauer, & Miltner, 2011). Further, in at least one instance, affect labeling was significantly more effective than distraction (Kircanski et al., 2012). If affect labeling effects were driven entirely by a distraction-related mechanism, we would not expect them to differ. Moreover, if affect labeling did operate by means of distraction, it becomes especially difficult to explain the time-delayed effects of clinical applications of affect labeling described earlier. Distraction is routinely cited as an impediment in the treatment of anxiety disorders, whereas affect labeling produces reliable long-term benefits in clinical contexts (Craske, Street, & Barlow, 1989; Grayson, Foa, & Steketee, 1982). While distraction may not be the correct characterization of affect labeling effects, perhaps some form of detachment might be a better characterization.

Self-reflection

In order to put our feelings into words, we must first identify what those feelings are, requiring an amount of self-reflection. Being aware of and observing our own experiences, especially of emotional experiences, is a primary feature of dispositional mindfulness (Baer, 2004) which has been linked to affect labeling ability. Individuals who exhibit higher levels of dispositional mindfulness also show stronger neural activations during affect labeling in several key areas including vlPFC and dlPFC as well as greater decreases in amygdala, activations which suggest a more robust and effective neural response to affect labeling as a function of dispositional mindfulness (Creswell, Way, Eisenberger, & Lieberman, 2007). Emotional introspection, without explicit processing through language, has itself been shown a neural

profile similar to what we would expect from successful emotion regulation: increased activity in vlPFC and decreased activity in amygdala (Herwig, Kaffenberger, Jäncke, & Brühl, 2010). With this evidence in mind, the important component in affect labeling could be self-reflection upon our emotions while the translation of these feelings into language only serves to initiate the introspection process or an externalized indicator that the self-reflection occurred. It might not be about language per se, but the steps required to get there.

This account begins to unravel, however, when we consider that successful regulation through affect labeling can be achieved when our focus is on an external stimulus, for example when we label the emotional expression in a face or the most aversive component within a scene. Focusing on emotions within the self and emotions within others share many common neural substrates (Ochsner et al., 2004) though not all. It is possible that affect labeling the self and others require slightly different processes in order to operate. Exploring the dichotomy between internally-focused affect labeling, when we put our own feelings into words, and externally-focused affect labeling, when we use language to identify the most aversive aspect of a stimulus, is an important avenue for future exploration of affect labeling effects.

Symbolic Conversion

Finally, affect labeling may operate as a form of emotion regulation by engaging more abstract thinking about the emotions or aversive stimuli being labeled. A number of studies have shown that when affect labels are replaced with more abstract labels, we see a similar profile of effects. In a set of studies reported earlier (Constantinou et al., 2015, 2014), participants also performed a more abstract variant of labeling by choosing to label aversive stimuli as either 'object', 'animal', 'human', 'landscape'. This 'content labeling' condition showed a similar effectiveness in reducing self-reported affect and physical symptoms as the affect labeling

condition. When participants were told to classify pictures of aversive and threatening stimuli (e.g., a snake mid-lunge or a gun pointed straight at the camera) as either 'natural' or 'artificial' in origin, vlPFC activity increased, limbic activity in the amygdala decreased (Hariri, Mattay, Tessitore, Fera, & Weinberger, 2003), and skin conductance responses decreased (Tupak et al., 2014). Another study showed that, in a paradigm very similar to the typical affect labeling paradigm, heightened amygdala activity viewing African-American faces was reduced when the label 'African-American' was applied to the images (Lieberman, Hariri, Jarcho, Eisenberger, & Bookheimer, 2005). Interestingly, abstract thinking has been linked to activity in vlPFC (Bunge, Kahn, Wallis, Miller, & Wagner, 2003) and is critical in processing the meaning of abstract words (Hoffman, Jefferies, & Lambon Ralph, 2010). Finally, abstract thinking about the causes of emotional states has also been suggested as an important component of reflecting upon feelings without increasing their negative impact and as potentially important feature distinguishing harmful rumination from helpful self-reflection (Kross, Ayduk, & Mischel, 2005). It may be the case that some types of labels other than affect labels are similarly effective at reducing affect, though we have shown that at least some others (e.g., gender labeling) are not. Symbolic conversion of the eliciting stimulus into language is an important component in affect labeling, though it is also possible that certain non-affect specific labels are also effective. It is also similarly possible that, while not specifically labeling affect, these more abstract labels do not preclude the processing of affect in a manner critical to more obvious forms of affect labeling. Much additional research is required to fully understand this distinction.

Open Questions About Affect Labeling

Reappraisal is a powerful form of emotion regulation for research because it demonstrates strong effects, tethers significantly to positive health effects, is cheap to implement, easy to train participants to perform, and until recently had no major identifiable drawbacks, especially when compared to other dominant strategies of emotion regulation (e.g., suppression; Gross, 2014). More recently, researchers have begun to uncover some of the drawbacks of reappraisal implementation such as a natural preference for other strategies at higher emotional intensities (Sheppes, 2014). As interest and research in affect labeling grow, especially within the clinical domain, and additional resources are dedicated to uncovering its effectiveness as a regulatory process, it becomes important to understand the limitations of affect labeling as an effective form of emotion regulation and acknowledge possible boundary conditions beyond which affect labeling may no longer be as effective.

How negative is too negative?

Emotion regulation strategies vary in their effectiveness as a function of the intensity of the evocative stimulus. For example, high intensity emotional stimulation may decrease the benefits of applying reappraisal compared to other strategies (Sheppes, Brady, & Samson, 2014; Sheppes & Gross, 2012) . Individuals prefer to employ distraction rather than reappraisal when emotional intensity is high, possibly because it is more effective or easier to implement in those situations (Sheppes, Scheibe, Suri, & Gross, 2011). Though as of yet untested, it is possible that affect labeling likewise is less effective or more difficult to implement when the target emotion needing regulation is highly intense.

There may be another way in which too much negativity can adversely affect successful regulatory engagement of affect labeling. In several cases, beneficial effects of affect labeling

were shown to increase with the amount of its engagement. As usage of negative affect labels increased, patients with spider phobia made greater progress in exposure therapy (Kircanski et al., 2012), medical students reported a greater reduction of depressive symptoms (Hoyt et al., 2014), and physical symptoms in cancer patients decreased after expressive writing interventions (Low et al., 2006). However in one case, use of negative words during an expressive writing intervention was shown to have a curvilinear relationship with anxiety such that moderate usage of affect labeling demonstrated the greatest reduction in anxiety symptoms while very high use of affect labeling was related to increased anxiety during a three-month follow-up (Niles, Byrne Haltom, Lieberman, Hur, & Stanton, 2015). Though more research is required, this relationship suggests there might be a down-side to the over-application of affect labeling, particularly for negative affect labels. This finding shares an interesting similarity to the literature on rumination; whereas many forms of therapy insist on the focus and/or direct confrontation of emotions (Greenberg, 2002), rumination over negative experiences can be counter-productive to progress in therapy and an indication of poor mental health (Mor & Winquist, 2002). It is possible that the harmful effects of excessive negativity are reflected within affect labeling as well.

Finally, the amount of negativity involved in the process of affect labeling may take the form of the labels themselves. Recent work has outlined the role of language in shaping our emotional experiences (Lindquist, MacCormack, & Shablack, 2015; Lindquist, Satpute, & Gendron, 2015). In this account, language acts as a context for individuals to make sense of the stimuli they are perceiving and, in the case of emotional language, may alter our emotional reactions to the stimulus by altering our perception of the stimulus itself (Barrett, Lindquist, & Gendron, 2007). We might then question if during affect labeling, some labels which provide

the context for understanding the evocative stimulus, may be more or less effective at encouraging (or disrupting) the regulatory effects of affect labeling. If only mildly negative words are used, will they be more effective at reducing felt affect to the stimulus because they reframe our understanding of the event (not unlike reappraisal)? Or would a more negative word be less effective as it generates additional affect within us that now needs to be regulated as well? Thus far, no research has directly investigated the efficacy of label intensity on the affect labeling process.

Which form of affect labeling is most effective?

As delineated earlier, there exist a number of studies investigating the effects of affect labeling that take many forms. One interesting point is that the stimulus being labeled varies between three categories: internal affect, external affect, or external objects. In many cases, participants are asked label how they themselves are feeling (Burklund et al., 2014; Kassam & Mendes, 2013; Lyubomirsky, Sousa, & Dickerhoof, 2006; Matejka et al., 2013; Mendolia & Kleck, 1993; Niles, Byrne Haltom, et al., 2015; Niles, Craske, et al., 2015). Putting our own feelings into words also has a heavy emphasis within the expressive writing literature (Frattaroli, 2006; Frisina et al., 2004). In other cases, especially within the neuroimaging literature on affect labeling, participants are instead asked to label the emotions of others by identifying their emotional expressions (Foland-Ross et al., 2012; Hariri et al., 2000, 2003; Lieberman et al., 2007; Payer et al., 2012; S. F. Taylor et al., 2003). And finally, participants are sometimes asked not to label affect *per se* but instead to use negative words to identify salient aversive objects in scenes (Kircanski et al., 2012; Lieberman et al., 2011; Tabibnia et al., 2008). Despite these three categories seeming very different, they are all considered forms of affect labeling and all exhibit the same profile of regulatory effects outlined earlier and to date extremely little work has been

done to differentiate the processes underlying each of these forms of affect labeling or even to uncover which is the most effective implementation of the paradigm (cf., McRae, Taitano, et al., 2010).

Can label source explain the inconsistent timing of effects?

Usually, we expect emotion regulation to have immediate effects. When confronted with an emotional event we want to diminish or enhance, we often prefer its alteration in the moment rather than at a later date. This is especially true of negative experiences like fear. However, sometimes, such as in a clinical context, the approach to emotion regulation requires a perspective that extends beyond the immediate situation. For example, in exposure therapy participants are exposed to an aversive stimulus so that they can diminish their negative response to it over a number of sessions.

Affect labeling has demonstrated both kinds of effects: immediate reductions in affect as well as a delayed longer-term effect. All of the neuroimaging work discussed earlier and much of the self-report findings on affect labeling show reductions in the immediate context but are only observed within a single session, whereas other studies (Kircanski et al., 2012; Mendolia & Kleck, 1993; Niles, Craske, et al., 2015; Tabibnia et al., 2008) report a delayed effect with the reductions in autonomic activity occurring days later in follow-up sessions when participants re-experience the aversive stimulus.

One feature many of these studies showing delayed effects have in common is that in most cases (cf. Tabibnia et al., 2008), the task protocol required participants to generate affect labels themselves rather than have them be provided (e.g., describing felt emotions out-loud as they are felt compared to selecting affect labels from word-choices on a screen). In fact, the only cases where affect labeling significantly *increased* self-reported affect or autonomic arousal

during the initial exposure also adopted a paradigm that required participants to self-generate and verbalize their emotional experiences (Mendolia & Kleck, 1993; Ortner, 2015). Interestingly, expressive writing, which often involves participants writing about emotions surrounding negative past events with relatively little instruction otherwise, has long-term benefits months later but, similar to the studies of affect labeling discussed in this section, participants do not report immediate effects of the affect labeling. Instead, many have reported feeling more negative affect immediately after writing sessions (Baikie & Wilhelm, 2005; Esterling et al., 1999). Self-generating labels may engage additional processes such as requiring a broader focus of self-monitoring or perhaps verbalization of internal emotional states within a monitored laboratory context increases self-consciousness, which alter the immediate but not the long-term consequences of affect labeling. It is also possible, however, that being provided affect labels generated by the experimenter instead of the participant, is a kind of interpersonal emotion regulation (Zaki & Williams, 2013). In some cases, emotional regulation can be more easily initiated with support from others (Nils & Rimé, 2012; Rimé, 2009) and to the extent that provided affect labels represent this kind of external support, this suggests a possible explanation for why providing labels could more easily induce immediate effects but self-generated labels may have a longer time-course to demonstrate effects.

Alternately, given that several of the studies with delayed effects were clinical applications of affect labeling, it is possible that this delayed effect is mostly specific to individuals with anxiety disorders. It is also possible instead that targeting phobias (i.e., deeply intense fears), it is also possible that although effects are observed eventually, immediate regulatory effects of affect labeling are not observed when the intensity of the emotional reaction is too high.

Aim of the Dissertation

Throughout this introduction, we have demonstrated the number of effects that affect labeling has on primary domains of emotional responding, a profile shared by more explicit forms of emotion regulation, reappraisal in particular. We have discussed research in which affect labeling demonstrates reduced self-reported affect, reduced autonomic activity, activation of a nearly identical profile of neural regions such as increased in prefrontal control regions (especially vlPFC) along with decreased emotion-generative activity in the amygdala, and reduced emotion-related behavioral effects. Further, we considered how, despite the effort involved in converting an emotion state or aversive stimulus into a symbol through language, the regulatory effects of affect labeling are counter-intuitive and unexpected, prohibiting it from conscious goal activation and marking the regulatory process involved in affect labeling as an implicitly activated one. Though the precise mechanisms of affect labeling are not yet fully understood, it seems clear that affect labeling should be considered a form of implicit emotion regulation.

While there are still many unanswered questions about affect labeling, this dissertation will focus on answered the three posed earlier: 1) what are the effects of label intensity on affect labeling success, 2) of the many types of affect labeling which are more or less effective at reducing emotional experience, and 3) does self-generation of affect labels enhance or disrupt the regulatory process involved in affect labeling?

Figure 1.1 – Demonstration of Typical Affect Labeling Trials

This figure demonstrates two common types of trial types in affect labeling task paradigms. A) Emotion Label: participants are instructed to choose the emotion word below that best describes the expression in the image above. B) Label Scene: participants are instructed to choose the word below that best describes the content of the image.

Figure 1.2 – Conceptual Summary of Affect Labeling Evidence

DOMAIN OF EFFECT	REAPPRAISAL	AFFECT LABELING
EXPERIENTIAL	• Decreases negative affect • Decreases positive affect	• Decreases negative affect • Decreases positive affect
AUTONOMIC	• Decreases peripheral physiology (e.g., SCR)	• Decreases peripheral physiology (e.g., SCR)
NEURAL	• Increased activity in vlPFC, dlPFC, SMA, and ACC • Decreased activity in amygdala	• Increased activity in vlPFC, dlPFC, and SMA • Decreased activity in amygdala
BEHAVIORAL	• Decreases a variety of emotion-related behaviors	• Decreases a variety of emotion-related behaviors

General Methods

For this dissertation, three tasks were administered to participants: the Label Intensity task investigating how the intensity of negative labels affects affect labeling performance, the Types of Affect Labeling task investigating which of the many forms of affect labeling is most effective, and the Number of Responses task investigating how providing or not providing labels influences the efficacy of affect labeling processes. Due to the high amount of overlap in participants, design, collection, and analysis, this chapter will cover the general methods for all studies included in this document and indicate the few cases in experimental design where approaches differ among the tasks.

Participants

Participants were healthy, right-handed individuals (N=23; 17 females, mean age = 21.48 sd = 3.89) recruited from the University of California, Los Angeles (UCLA) campus and surrounding community via flyers and emails. While all participants indicated they were at least in part non-Hispanic White, several participants also indicated a at least one additional racial background (Black/African-American: 2, Asian/Asian-American: 3, Hispanic or Latino/Latina: 3, Native American/American Indian/Native Alaskan: 2). All participants were native English speakers, had no psychiatric or neurological disorder or serious physical illness, and screened as eligible for the MR scanner (right-handed, no metal, and female participants were not pregnant). These participants were used for all three tasks described in this document and tasks were counterbalanced to avoid order effects. Three participants were excluded from all analyses in this document for technical issues during the scanning procedure (2) or for failure to follow instructions (1) yielding 20 remaining participants included in analyses. One participant was

excluded from analysis of the Label Intensity task only because of a scanner malfunction the stopped the scan prematurely yielding 19 participants included for this particular task.

Experimental Design

In each study, participants were administered a modified version of the affect labeling tasks used in a previous fMRI investigation (Lieberman et al., 2007, 2011). Tasks were presented via the Psychophysics Toolbox (Brainard, 1997; Kleiner et al., 2007) in the MATLAB environment version 7.4. Instructions were provided to subjects during a pre-scan session along with example trials to familiarize subjects with the task. Each subject confirmed understanding of the task procedures. Additionally, experimenters reminded subjects of the task instructions just prior to administration in the scanner via verbal communication as well as through a visual prompt on screen. Participants viewed the task via MR compatible LCD goggles while in-scanner responses were made via a button response box held in the subject's right hand.

Stimuli for each task were presented in a block design with each block starting with a 2.1 second instruction cue indicating which condition was about to appear. Blocks were comprised of five stimuli: four negative stimuli and one positive stimulus that appeared as either the first or last trial in each block. Positive images were included to mitigate the effects of habituation on amygdala responsiveness to the stimuli (Breiter et al., 1996; Fischer et al., 2003; Gee et al., 2015; Wright et al., 2001). For the Label Intensity task, conditions were repeated 4 times each during a single run yielding 64 negative trials (16 per condition, 4 per block) and 16 positive trials (4 per condition, 1 per block). For the Types of Affect Labeling and Number of Responses tasks, conditions were repeated 3 times during a single run yielding 48 negative trials (12 per condition, 4 per block) and 12 positive trials (3 per condition, 1 per block). Trials consisted of

simultaneous presentation of the image and accompanying labels (when applicable) for 5 seconds each and were spaced by a .3 second blank screen interstimulus interval during blocks to signify the end of a trial. Including the instruction cue and ISIs, each block lasted for 28.6 seconds. Before the first block and after the last trial in each block, a fixation cross appeared for 10 seconds to capture activity at rest. Each condition was presented in one of four pseudorandomized presentation orders during a single run that counterbalanced the order in which conditions appeared. Although the presentation orders counterbalanced the condition order across participants, within a participant the conditions were presented in a repeating order (e.g. 'ABCDABCD' for one participant but 'CDABCDAB' for another). No stimuli were repeated within or among any of the tasks. To the extent that a condition in a task had a correct response, the location of the correct answer was counterbalanced within conditions.

Label Intensity Task

The Label Intensity task was comprised of four conditions: Abstract labels, Low Intensity labels, High Intensity labels, and Gender labels. In each condition, participants were shown images from the NimStim set of facial expressions (Tottenham et al., 2009) that were either angry, fearful, or happy expressions along with two accompanying labels and were asked to perform one of two tasks depending on the condition. Expressions were counterbalanced across dimensions of emotion, gender, and whether it was an 'open' or 'closed' mouth expression. For the Abstract, Low Intensity, and High Intensity conditions, images were paired with emotion words and participants were instructed to choose via the button box the word that best describes the emotion the person on the screen is feeling and displaying. The words supplied varied as a function of their level of abstraction and intensity. For the Abstract condition, basic emotion words ('angry', 'scared', 'happy') were chosen. For the Low Intensity and High Intensity

conditions, words were chosen based on pre-tested levels of intensity suggested by the words on a scale from 1-7 from a different pool of 57 participants recruited from Amazon's Mechanical Turk service. For Low Intensity, the emotion words used were *frustrated* (mean = 4.40, sd = 1.12), *irritated* (mean = 3.86, sd = 1.33), *startled* (mean = 4.39, sd = 1.35), *worried* (mean = 4.23, sd = 1.18), *content* (mean = 2.61, sd = 1.40), and *glad* (mean = 3.68, sd = 1.62). For High Intensity, the emotion words used were *enraged* (mean = 6.81, sd = .58), *furious* (mean = 6.63, sd = .83), *terrified* (mean = 6.68, sd = .78), *horrified* (mean = 6.39, sd = 1.03), and *ecstatic* (mean = 6.26, sd = 1.28). The emotion words in the Abstract condition, *angry* (mean = 5.54, sd = 1.05), *scared* (mean = 5.37, sd = 1.10), and *happy* (mean = 4.60, sd = 1.41), each fall in the middle of their respective emotion categories between the low and high intensity words (see Figure 2.1). Emotion words were counterbalanced to appear an equal number of times with each type of expression as well as which side they appeared as a correct selection (left or right). For the Gender condition, facial expressions were paired with gender-typical names instead (e.g. Alice, Samuel, etc.) and participants were instructed to choose from the two gender-typical names the one that best matched the person on the screen. See Figure 2.2 for examples of each condition in the Label Intensity task.

Types of Affect Labeling task

The Types of Affect Labeling task was comprised of four conditions: Internal Affect labels where participants were instructed to choose from a pair of labels those that best captured their own emotional experience, External Affect labels where participants were instructed to choose from a pair of labels the one that best captured the emotional experience of the person(s) in the image, External Object labels where participants were asked to choose between a pair of labels the one that best describes the content of the image, and Observe where participants were

instructed to simply attend to the image and allow themselves and their emotions to respond naturally. In each condition, participants were shown images from the International Affective Picture System (Lang et al., 2008). Images across all conditions were matched in overall valence (Internal Affect: mean = 2.62 sd = .65; External Affect: mean = 2.43, sd = .43; External Object: mean = 2.43, sd = .51; Observe: mean = 2.49, sd = .79) and arousal (Internal Affect: mean = 5.84 sd = .87; External Affect: mean = 5.79, sd = .76; External Object = 5.83, sd = .70; Observe: mean = 6.03, sd = .66) with positive images in each condition excluded as well as when the positive images were included for valence (Internal Affect with positive: mean = 3.56 sd = 2.02; External Affect with positive: mean = 3.37, sd = 2.01; External Object with positive: mean = 3.46, sd = 2.19; Observe with positive: mean = 3.40, sd = 2.02) and arousal (Internal Affect with positive: mean = 5.53 sd = 1.01; External Affect with positive: mean = 5.51, sd = .96; External Object with positive: mean = 5.63, sd = .85; Observe with positive: mean = 5.53, sd = 1.20). No differences among any of the conditions along the dimensions of valence or arousal are statistically significant. For the Internal Affect and External Affect conditions, images were paired with basic emotion words from the list 'angry', 'scared', 'sad', 'disgusted', or 'happy'. For Internal Affect, the top two most highly rated normed emotional responses were used as the labels (Libkuman, Otani, Kern, Viger, & Novak, 2007; Mikels et al., 2005) with additional labels provided by internal rating when normed ratings were not available for images. For External Affect, labels were chosen based on apparent emotional expressions and context. For the External Object condition, one label was presented that describes the aversive content within the scene and was presented with another negative label that did not. Because in one condition (External Affect) participants were required to make judgements about the emotional state of an individual in the image, to ensure consistency across all conditions every image used contained

at least one human subject. See Figure 2.3 for examples of each condition in the Types of Affect Labeling task.

Number of Responses task

The Number of Responses task was comprised of four conditions: One Label where participants were instructed to decide if the emotion word provided best described their emotional response to the accompanying image or not, Two Labels where participants were instructed to choose between two labels the one that best captured their emotional response to the accompanying image, Free Response where participants were instructed to generate a label that best describes their emotional response to an image and press a button to indicate they had decided for that trial, and Observe where participants were instructed to simply attend to an image and allow themselves and their emotions to respond naturally. In each condition, participants were again shown images from the International Affective Picture System (Lang et al., 2008). Once again, images across all conditions were matched in overall valence (One Label: mean = 2.38 sd = .53; Two Labels: mean = 2.40, sd = .58; Free Response: mean = 2.38, sd = ..47; Observe: mean = 2.39, sd = .71) and arousal (One Label: mean = 6.01 sd = .72; Two Labels: mean = 5.95, sd = .87; Free Response = 5.94, sd = .57; Observe: mean = 6.01, sd = .56) with positive images in each condition excluded as well as when the positive images were included for valence (One Label with positive: mean = 3.33 sd = 2.05; Two Labels with positive: mean = 3.41, sd = 2.16; Free Response with positive: mean = 3.42, sd = 2.19; Observe with positive: mean = 3.36, sd = 2.13) and arousal (One Label with positive: mean = 5.52 sd = 1.23; Two Labels with positive: mean = 5.53, sd = 1.20; Free Response with positive: mean = 5.62, sd = .84; Observe with positive: mean = 5.62, sd =.97). No differences among any of the conditions along the dimensions of valence or arousal are statistically significant. For the One Label

condition, images were paired with the most highly rated normed basic emotion words from the list ‘angry’, ‘scared’, ‘sad’, ‘disgusted’, or ‘happy’ for each image while for the Two Labels condition the top two most highly rated normed emotional responses were used as the labels (Libkuman et al., 2007; Mikels et al., 2005) with additional labels provided by internal rating when normed ratings were not available for a given image. For Free Response, no labels were provided but participants were instructed before the study to mentally choose from the same words listed above in order to increase the chances that participants would perform the task correctly, to make the condition more comparable to the label conditions since they use labels from the same small pool or words, and to eliminate individual differences emotional granularity, the ability for some individuals to differentiate between their emotional states more or less than others (Smidt & Suvak, 2015). See Figure 2.4 for examples of each condition in the Number of Responses task.

Image Acquisition

Imaging data were acquired via a Siemens Prisma 3 tesla MRI scanner at the UCLA Ahmanson-Lovelace Brain Mapping Center. We acquired 2630 function T2*-weighted echo planar image volumes (EPIs; slice thickness = 2mm, no gap, interleaved slice acquisition order, 65 slices, in-plane voxel size = 2mm x 2mm, TR = 1000ms, TE = 27ms, flip angle = 70°, matrix = 104x104, FoV = 208mm, acceleration factor = 5x, phase encoding direction = A>>P, slice angle acquisition = -30° adjusted per subject to accommodate the whole brain within the FOV). The Label Intensity task was administered as a single run collecting 636 volumes for a runtime of 10 minutes and 36 seconds. A T1-weighted, magnetization prepared, rapid-acquisition, gradient echo anatomical scan (MPRAGE; slice thickness = .8mm, gap = .4mm, in-plane voxel

size = .8mm x .8mm, TR = 2300ms, TE = 2.99ms, flip angle = 7°, matrix = 256 x 256, FoV = 256mm) was also acquired.

Image Analysis

Preprocessing

Imaging data were analyzed using SPM8 (Wellcome Department of Cognitive Neurology, Institute for Neurology, London, UK). All images were first manually reoriented to align brains along a horizontal AC-PC line with the image origin at the anterior commissure; structural images were reoriented independently but functional images were reoriented using parameters from the first run's first image applied to each subsequent volume within that task. All functional images were then realigned to the first volume within the appropriate run to correct for head motion. High resolution MPRAGE structural images were co-registered to a mean EPI. MPRAGE anatomical images were then normalized using the New Segmentation algorithm within SPM8 to warp them into Montreal Neurological Institute space (resampled at 1x1x1mm; Mazziotta et al., 2001). Resulting flow fields from the normalization routine were applied to functional images which were then subsequently smoothed using a 4-mm Gaussian kernel FWHM. Finally, visual inspection was employed assessing EPI alignment to structural images after co-registration and accurate warping to the MNI standard space after normalization to assure quality of the preprocessing pipeline for images from all subjects and runs.

Statistical Analysis

For the functional imaging data, general linear models were defined separately for each participant and each task was analyzed separately but shared some common analytical approaches outlined here. Each task included regressors for the four conditions of interest

mentioned earlier (Label Intensity task: Abstract, Low Intensity, High Intensity, Gender; Types of Affect Labeling task: Internal Affect, External Affect, External Object, Observe; Number of Responses task: One Label, Two Labels, Free Response, Observe). Because we were primarily interested in the effects of affect labeling on negative stimuli, the positive ('happy') trials were separated out into separate nuisance regressors for each condition. Prompt cues were also separated out as an additional single nuisance regressor for each task. Blocks were modeled as box car functions spanning from onset of the first negative stimulus in the block to the offset of the last negative stimulus convolved with the canonical double-gamma hemodynamic response function (HRF). Six motion parameters were included as covariates of no interest. Additionally, regressors identifying individual volumes as representing global signal intensity change (thresholded at 2.5 standard deviations from average global signal intensity within the run) as well as regressors indicating spikes of movement (1mm in a single volume) were similarly included. High pass filters were set at SPM8 default values of 128s. Contrast images for the Label Intensity task were created at the subject-level according to the contrasts of interest for each task. For the Label Intensity task, contrasts included Abstract > Gender, Low > Gender, High > Gender, Low > High, and Abstract > Specific (Low + High). For the Types of Affect Labeling task, contrasts included Internal Affect > Observe, External Affect > Observe, External Object > Observe, External Affect > Internal Affect, External Object > Internal Object, and Affect (Internal Affect + External Affect) > Object (External Object alone). For the Number of Responses task, contrasts included One Label > Observe, Two Labels > Observe, Free Response > Observe, One Label > Two Labels, One Label > Free Response, Two Labels > Free Response, and Provided Labels (One Label + Two Labels) > Free Response.

Whole-brain Analysis

To investigate whole-brain group-level effects for each task, the resulting contrasts images from subject-level analyses described above were used in a random-effects analysis using a one sample t-test in the GLM Flex statistical software package (http://mrtools.mgh.harvard.edu, May 17 2016). Voxels with missing data from subjects were analyzed using degrees of freedom adjusted to the number of subjects contributing to that data point. Reported p-values are adjusted automatically within the GLM Flex software to reflect the equivalent p-value for the degrees of freedom dependent upon the full model. Voxels missing data from more than a quarter of the subjects in the full model were eliminated from the analysis. All visualization using surface rendering and generation of peak tables were created using the bspmview software (http://www.bobspunt.com/bspmview, May 17 2016). For the analyses of task main effects, a voxel-level threshold cutoff of $p < .005$ and cluster-level family-wise error (FWE) correction set at .05 was applied using the bspmview software. All results reported and visualized exceed these joint voxel-wise and cluster- extent thresholds.

ROI Analysis

Of particular interest for the purpose of this document are the activations across conditions in specific regions of interest including the amygdala and vlPFC which have each been implicated many times in affect labeling (e.g., Burklund et al., 2014; Lieberman et al., 2007) as well as emotion regulation more broadly (Buhle et al., 2014; Ochsner & Gross, 2007).

In neuroimaging investigations concerning the amygdala, it is typical to treat the structure as a single unit. However, known subregions of the amygdala have in recent years been accurately identified in the MR scanner using a combination of cytoarchitectonic (Amunts et al., 2005) with functional (Ball et al., 2007; Roy et al., 2009) and structural connectivity (Balderston, Schultz, Hopkins, & Helmstetter, 2014; Solano-Castiella et al., 2010, 2011). The three major

subregions of the amygdala that have been identified are the laterobasal (LB), centromedial (CM), and superficial (SF) subregions (Amunts et al., 2005). See Figure 2.5 for visualizations of these regions. While not all researchers use these definitions precisely to identify functional dissociations among subcomponents of the amygdala, there exists some amount of work finding the functional separation of these regions or at least ventral amygdala (which coincides more with LB) with dorsal amygdala (which coincides more with CM and SF). LB or ventral amygdala have been linked to the acquisition of conditioned fear (Morris, Buchel, & Dolan, 2001), visual saliency (Balderston et al., 2014), observing faces expressing negative emotions (Whalen et al., 2001), negative word stimuli (Han, Lee, Kim, & Kim, 2013), whereas more dorsal regions of the amygdala including CM and SF have been linked more specifically to aversive images (Balderston et al., 2014), a transient burst of activity upon presentation of conditioned stimulus previously paired with shock (CS+; Morris et al., 2001), and more active specifically to fearful faces than angry faces (Whalen et al., 2001). While all regions are important in processing negative content, it seems the more dorsal regions including CM and SF may be more involved specifically in threat-related processing especially when it requires translating fear into action (Fox, Oler, Tromp, Fudge, & Kalin, 2015). For this reason, we have decided to analyze not only the more typical single unit amygdala ROI taken from the WFU PickAtlas as a comparison, but also the probabilistic maps of the LB, CM, and SF amygdala subregions as defined by the SPM Anatomy Toolbox.

As we are also interested in the regulatory processes engaged within vlPFC, we have included several ROI masks containing these regions as well. Using the WFU PickAtlas, we included anatomically defined sections of the inferior frontal gyrus including pars orbitalis, pars opercularis, and pars triangularis. Although much of the pars triangularis would not be

considered ‘ventral’ prefrontal cortex, meta-analyses of emotion regulation have shown large involvement in dorsolateral prefrontal regions and so this region was included as well. Further, functional peaks were taken from unpublished neuroimaging work that collected 120 participants who engaged in two affect labeling tasks: a facial expression based task (similar to our Label Intensity task) that compared affect labeling to gender labeling (AL-GL) as well as an aversive image based task (much like our Types of Affect Labeling and Number of Responses tasks) which compared affect labeling to an observe condition (AL-OBS). Peak coordinates were taken from the vlPFC clusters in each of these SPM images on the left and right side (AL-GL: L [-51 30 0], R [51 30 -3]; AL-OBS: L [-42 45 -3], R [33 54 3]) and 6mm spherical ROI masks were created around these coordinates. See Figure 2.6 for visualizations of the anatomical and structural ROIs for the lateral prefrontal cortex used. Parameter estimates from first-level contrasts were extracted and analyzed using custom scripts in MATLAB and R Statistical software. One-sample t-tests of the contrasts created at the first-level were performed. Parameter estimates were visualized compared to baseline using the R package ggplot2, however, significance indicators on the graphs for ROI analyses indicate the results of the one-sample t-tests. ROI results that were trending towards significance were also included in the visualizations and result tables as the study is relatively underpowered for an imaging study (N = 20) and data collection is still ongoing. Tests that were non-significant or were at least not trending towards significance are not reported.

Figure 2.1 – Emotion Word Intensity

Results of the emotion word intensity pre-test used to decide which labels were appropriate for the low and high intensity label conditions as well as ensuring the abstract emotion words (angry scared, happy) were not confounded by intensity.

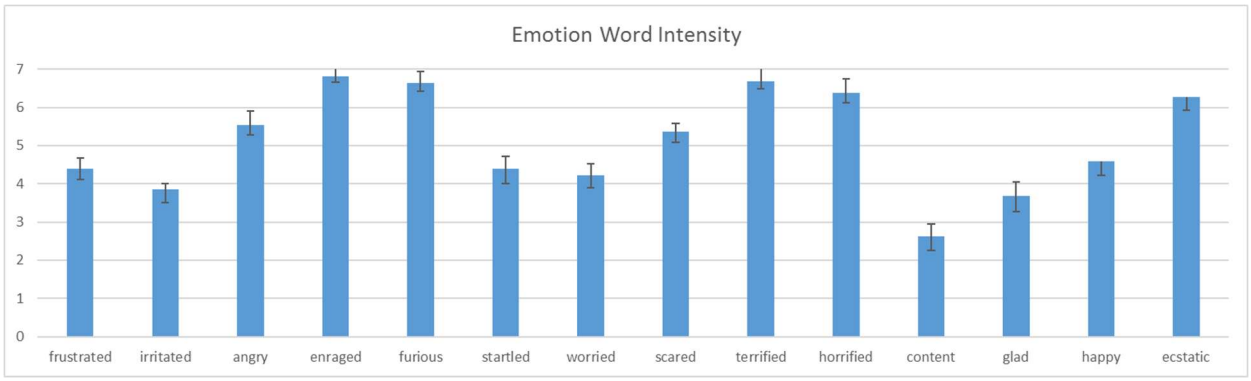


Figure 2.2 – Label Intensity Task Trial Examples

Demonstration of the four conditions within the Label Intensity task with their accompanying pre-block instruction cues: A) Abstract labels, B) Low Intensity labels, C) High Intensity labels, and D) Gender labels.

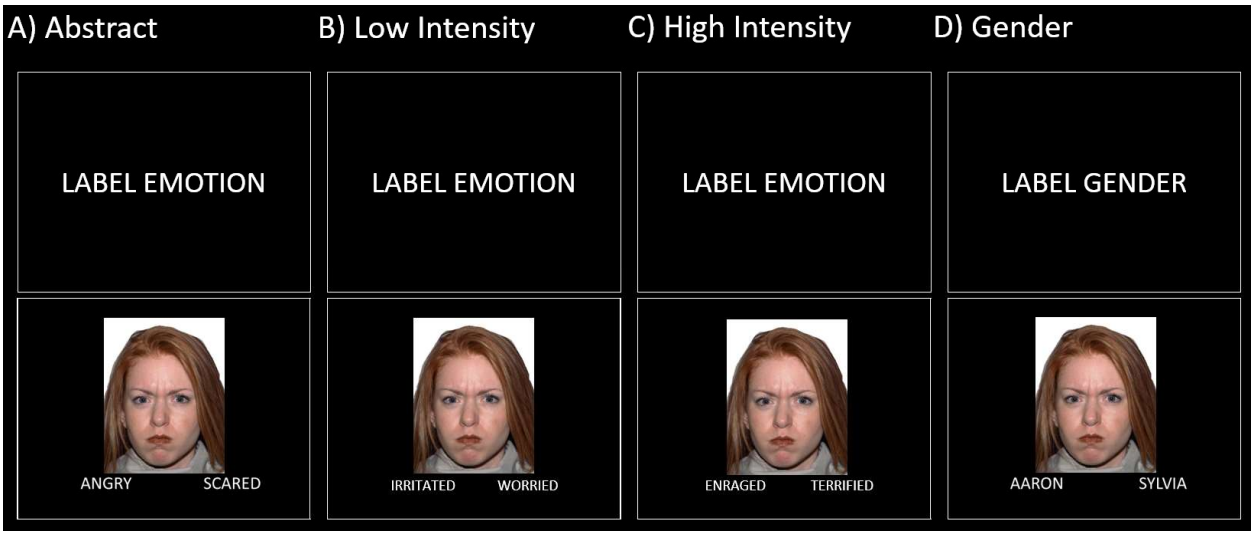


Figure 2.3 – Types of Affect Labeling Task Trial Examples

Demonstration of the four conditions within the Label Intensity task with their accompanying pre-block instruction cues: A) Internal Affect, B) External Affect, C) External Object, and D) Observe.

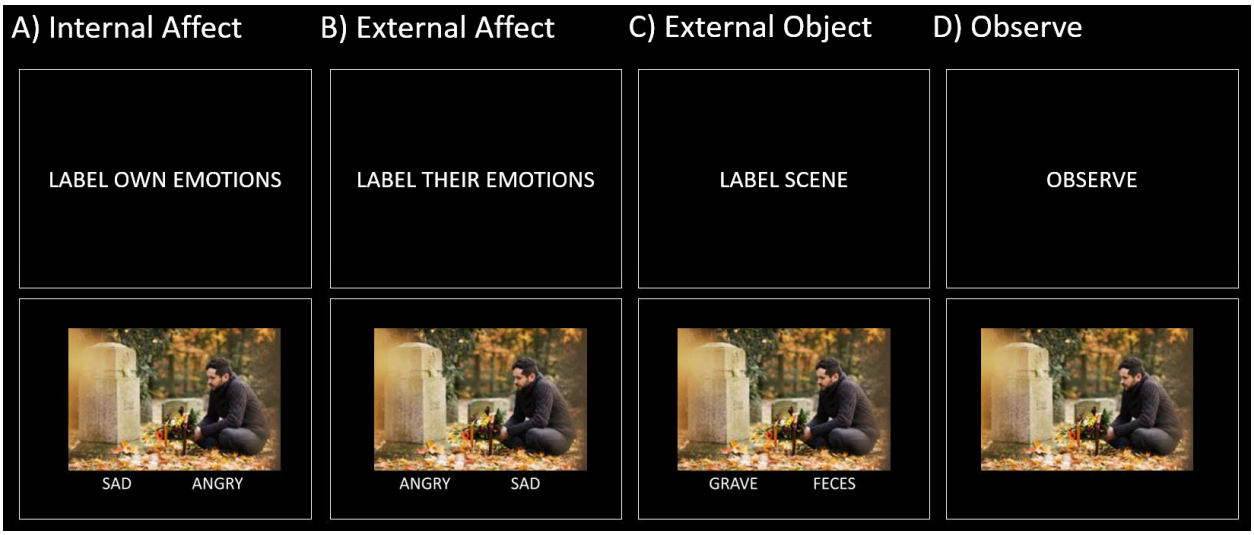


Figure 2.4 – Number of Responses Task Trial Examples

Demonstration of the four conditions within the Label Intensity task with their accompanying pre-block instruction cues: A) One Label, B) Two Labels, C) Free Response, and D) Observe.

Figure 2.5 – Amygdala ROIs

Visualization of the Amygdala ROI masks used. A) Coronal slice of WFU PickAtlas AAL Amygdala ROI at y = 0. B) Coronal slice of amygdala subregions at y = -3 (Laterobasal [LB] = blue, Centromedial [CM] = green, Superficial [SF] = magenta). C) Axial slice view of amydala subregions.

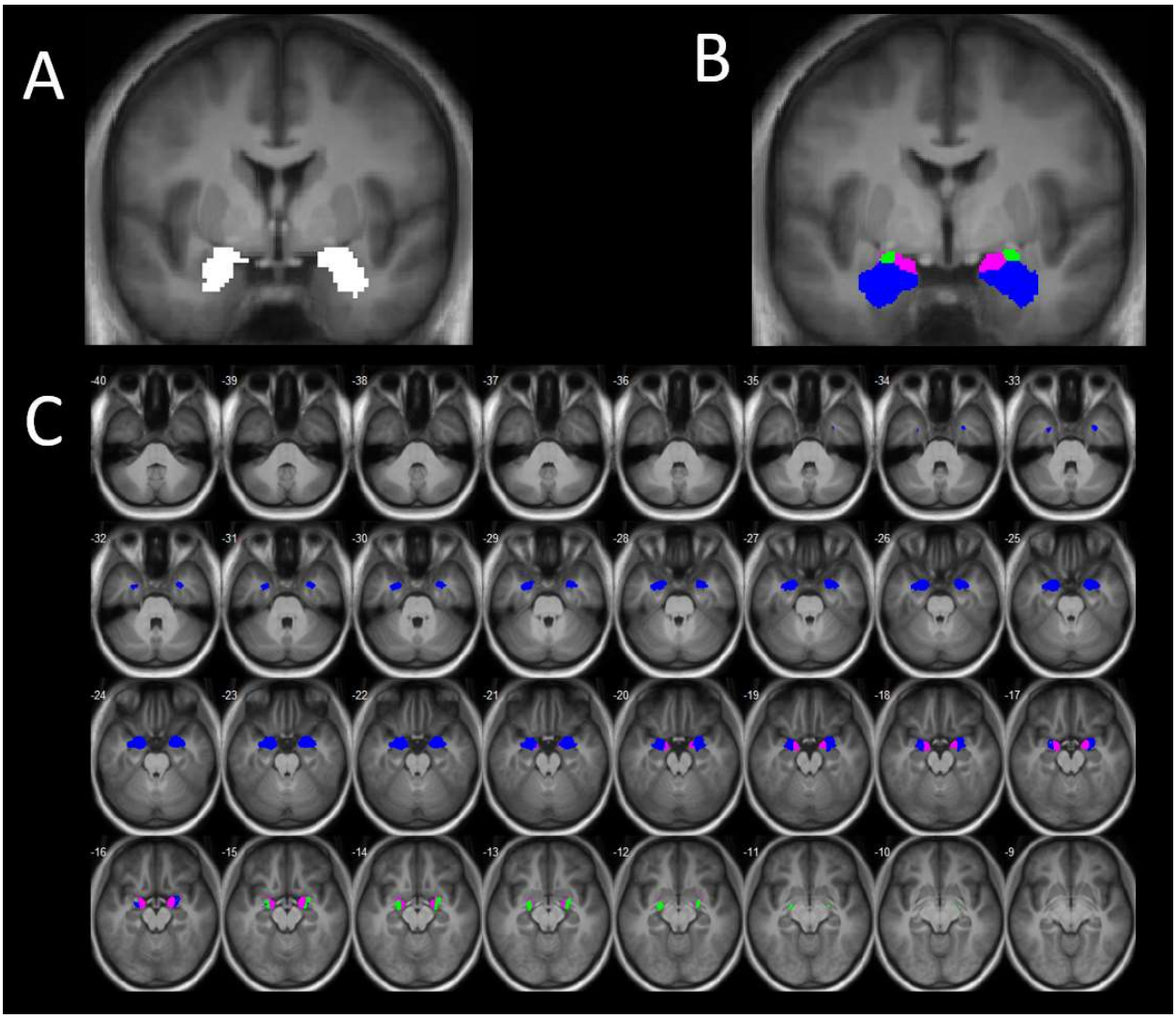
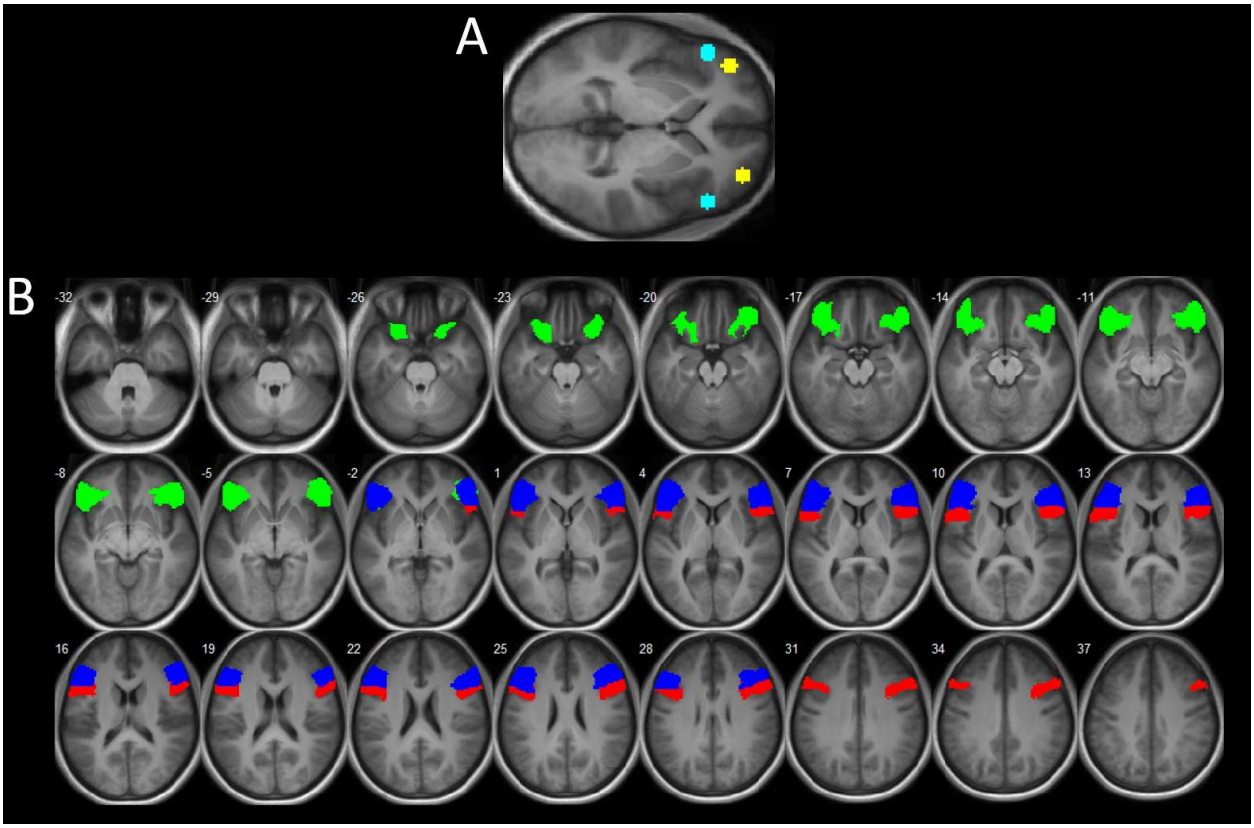


Figure 2.6 – Lateral Prefrontal Cortex ROIs

Visualization of the lateral prefrontal cortex ROI masks used. A) Axial slice of functional peaks borrowed from existing affect labeling dataset with comparing Affect Labeling > Gender Labeling (AL-GL; teal) and Affect Labeling > Observe (AL-OBS; yellow). B) Axial sliceview of WFU PickAtlas AAL inferior frontal gyrus subregions (pars orbitalis [orb] = green, pars opercularis [oper] = blue, pars triangularis [tri] = red).



Study 1 – Label Intensity Task

We first sought to answer the question about the effects of label intensity and their impact on the regulatory effectiveness of affect labeling. It has been argued that language provides a context for our emotional experiences (Lindquist, Satpute, et al., 2015). Within this framework, we might expect that more extreme or intense language used during affect labeling would induce more affect that needs to be regulated while less intense language would induce less. However, if symbolic conversion is a major driving force behind successful affect labeling, we may expect language used during affect labeling that is more abstract, regardless of its intensity, would be most effective as it forces the individual to achieve a higher level of abstractness for converting the stimulus. These two frameworks would suggest opposing results for the effect of label intensity on affect labeling success. If the labels provided during a typical affect labeling paradigm provide the context for how the stimulus is perceived, then we might expect more intense labels to induce more affect initially and thus result in higher overall affect when compared to affect labeling with less intense labels. If instead the level of abstraction of the labels used is more important, then we might expect more abstract words to reduce affect regardless of their intensity. To test this, we used a modified version of a common affect labeling paradigm and varied the labels used along two dimensions: affect intensity and level of abstraction. Using stimuli of faces expressing negative emotions (Tottenham et al., 2009), we presented the stimuli in four conditions: Abstract labels (e.g. ‘angry’ or ‘scared’), Low Intensity labels (e.g. ‘irritated’ or ‘worried’), High Intensity labels (e.g. ‘enraged’ or ‘terrified’), and Gender labels (e.g. ‘aaron’ or ‘sylvia’). We have discussed earlier the affective intensity gradient of emotion labels across the conditions and how the abstract emotion words land securely in the middle of the low intensity and high intensity words and thus do not create a

confound (see Figure 2.1 in the previous chapter). We expect that, compared to the control condition of Gender labeling, each affect labeling condition (Low, High, or Abstract) will reduce amygdala activity despite the level of intensity for the simple fact that they are all still at their core affect labeling. We further expect that only one of the following two contrasts will demonstrate reductions in amygdala: if the symbolic conversion framework is correct, then Abstract > Specific (the comparison of the abstract emotion words against the more specific emotion words in Low and High conditions) will produce reductions in amygdala activity. If instead the labels act as a context which drives the regulatory effects, then we would expect that less intense words would be more effective at reducing as should be seen in the Low > High contrast. Given the demonstrated regulatory role of the vlPFC in emotion regulation, we expect activity in vlPFC regions to show effects opposite to those of the amygdala in each contrast.

Results

Whole-brain results

Each condition of interest was first compared against the control condition of Gender labeling to assess the whole-brain effects. No amygdala results were observed at the whole-brain level for any of the contrasts analyzed so we will focus on prefrontal activations at the whole-brain instead. In the case of Abstract > Gender, deactivations were observed in the dorsomedial prefrontal cortex (dmPFC), dorsolateral prefrontal cortex (dlPFC). For Low > Gender, we observed similar decreases in the dmPFC, dlPFC as well as increased vlPFC activity bilaterally. For High > Gender, deactivations were again observed in dmPFC and left dlPFC with activations also only in the left vlPFC. Turning to the contrast of Abstract > Specific emotion words, deactivation was observed in the left vlPFC indicated higher levels of lvlPFC activity during the

Low and High intensity conditions compared to the Abstract condition. No prefrontal activations were observed in the Low > High contrast. A table with the complete listing of all activations and deactivations observed during whole-brain analysis for this task is available in Appendix A (see Table A.1).

ROI results

Activity was compared in each condition of interest against the control condition Gender within the amygdala as a single-unit ROI (AAL) and each of its subregions (CM, LB, SF) for both the left and right amygdala. Activity was also compared in lateral prefrontal areas including pars orbitalis (orb), pars opercularis (oper), and pars triangularis (tri) as well as two functional peaks taken from another affect labeling data set (AL-GL and AL-OBS). A complete table listing the results of each statistical test computed for this task is available in Appendix B (Table B1).

First, the experimental conditions were compared against the control condition Gender. The Abstract > Gender contrast revealed the left laterobasal amygdala was more active during Gender than the Abstract condition as were several regions of the vlPFC. For Low > Gender, several regions of the right amygdala (LB, CM) as well as the amygdala as a whole (AAL) were more active for Low than for Gender as were a number of vlPFC and dlPFC regions although one region of the right vlPFC was more active for Gender than for Low. For High > Gender, the left laterobasal amygdala trends toward significance yielding more activity during the High condition than Gender while the right superficial amygdala yielded more activity for Gender than for High. Regions of the lateral prefrontal cortex are likewise split with several regions of both left and right vlPFC and dlPFC more active during High than Gender and other regions in right vlPFC more active during Gender than High.

Turning to test Abstract > Specific, only right vlPFC showed trending activity for Abstract over Specific, but many regions including left laterobasal amygdala, left single-unit amygdala, and right centromedial amygdala as well as bilateral vlPFC and dlPFC regions showed more activation for Specific than for Abstract. Finally, in the Low > High contrast, right amygdala including superficial and centromedial were more active during the Low intensity condition than the High intensity condition whereas left laterobasal amygdala was trending towards significance for more activity during High than Low.

Discussion

Concerning each condition compared against the control condition Gender, we expected to see decreased amygdala activity and increased vlPFC as is typically expected for affect labeling tasks, however the results were more complicated. While we did see some evidence of amygdala deactivation in all the Abstract condition and the High condition compared to Gender, the Low condition has significant increase in amygdala activity. We did not observe increased vlPFC activity relative to Gender in the affect labeling conditions and instead saw several vlPFC ROIs. With the functional peak AL-GL taken from another dataset using facial stimuli to investigate affect labeling, both Low and High did show significant and expected increases in vlPFC compared to Gender despite several amygdala regions showing increased activity in Low rather than the expected decrease. Additionally, the Abstract condition did not reveal increases in this vlPFC region compared to Gender but it did reveal decreased amygdala activity.

When comparing the Low intensity and High intensity words directly, the Low intensity words generated more activity in the right amygdala in both superficial and centromedial subregions which suggests the account that lower intensity words should be more effective at

reducing amygdala during affect labeling was incorrect. When comparing the more abstract emotion words against the average of the more specific emotion words, if the symbolic conversion through language account was the main force behind successful affect labeling, we expected to see amygdala reductions during the Abstract condition. Compared to Abstract, the Specific emotion words generated significantly more amygdala activity in the left laterobasal amygdala, left overall amygdala, and trending significance in the right centromedial amygdala. However, as before the pattern of vlPFC activity does not mirror amygdala activity as we would expect it to given its suggested regulatory role. Instead, only one region of vlPFC, the functional peak taken from AL-OBS, shows increased activity in Abstract > Specific whereas Specific > Abstract shows a swath of increased bilateral vlPFC and dlPFC. Based on the amydala findings alone, we may be willing to conclude that the symbolic conversion framework is correct and level of abstraction is more important to successful affect labeling than the intensity of the words themselves. However, given the strange and unexpected vlPFC findings, the true story is undoubtedly more complicated.

Figure 3.1 – Label Intensity Task Whole-brain Results vs. Control

Surface rendering of whole-brain results for conditions of interest against the control condition in the Label Intensity Task. All rendering done via bspmview software using $p < .005$ voxel-wise threshold and $p < .05$ FEW cluster-level correction.

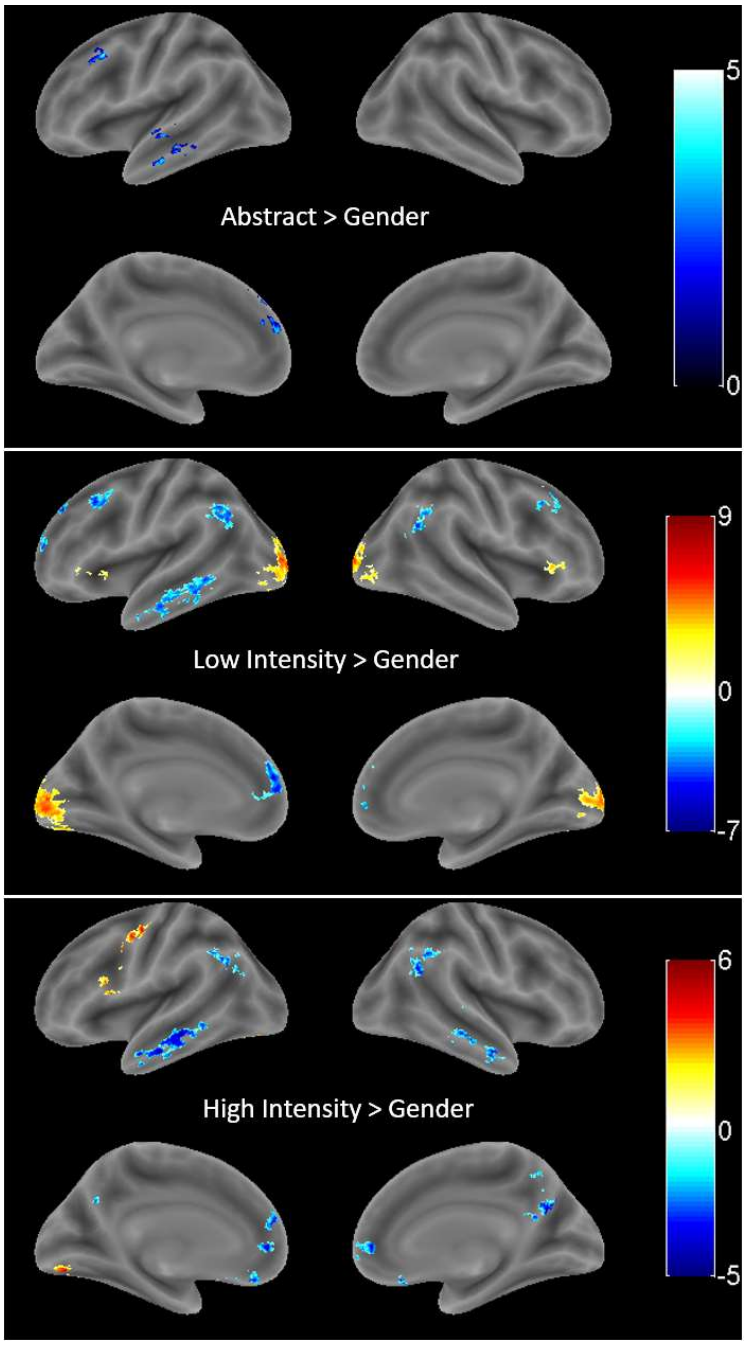


Figure 3.2 – Label Intensity Task ROI Amygdala Results

Results of the ROI analyses for amygdala. Only showing significant (* p < .05; ** p <. 01) or trending (+ p < .1) results for each contrast (Low, Abstract, High) vs. Gender. White bars represent activity in the single-unit amygdala ROI. Black bars represent activity in the constituent amygdala subregions.

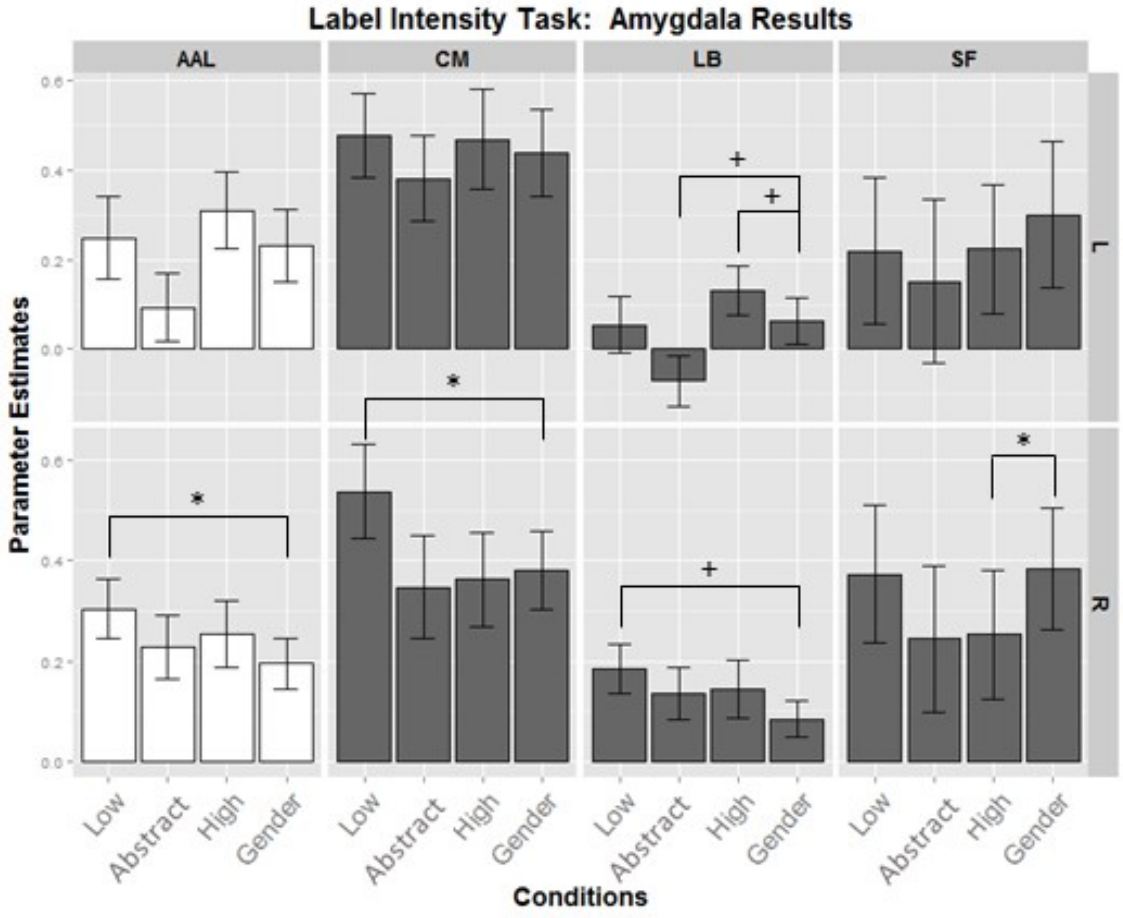


Figure 3.3 – Label Intensity Task ROI Lateral PFC Results

Results of the ROI analyses for lateral PFC. Only showing significant (* p < .05; ** p <. 01) or trending (+ p < .1) results for each contrast (Low, Abstract, High) vs. Gender. White bars represent data taken from ROIs defined by functional peaks from an existing dataset. Black bars represent data taken from anatomical ROIs.

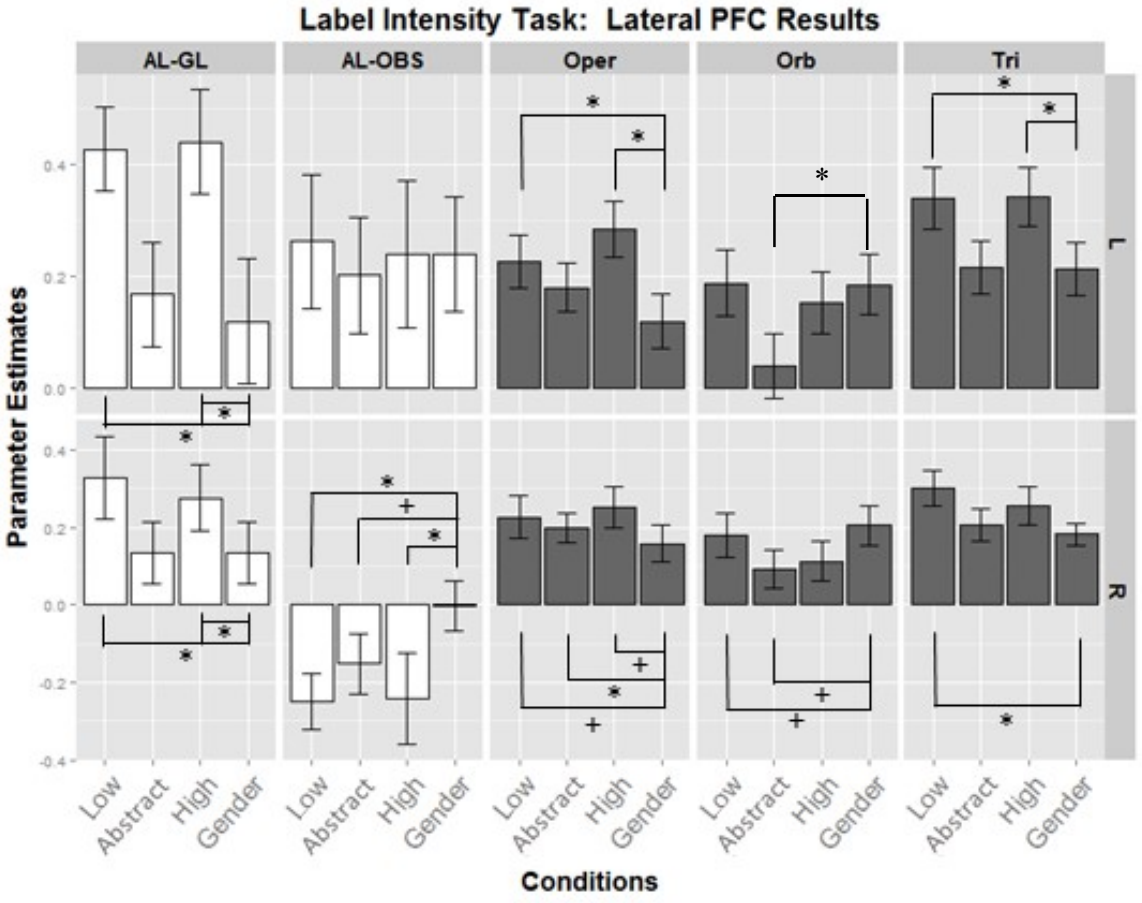


Figure 3.4 – Label Intensity Task Abstract > Specific Results

Surface rendering of whole-brain results for the specific hypothesis in the Label Intensity Task of Abstract emotion labels > Specific emotion labels (top) and results of ROI analyses for this contrast for amygdala (bottom left) and lateral PFC (bottom right). For ROI results, results are indicated as significant (* $p < .05$) or trending (+ $p < .1$).

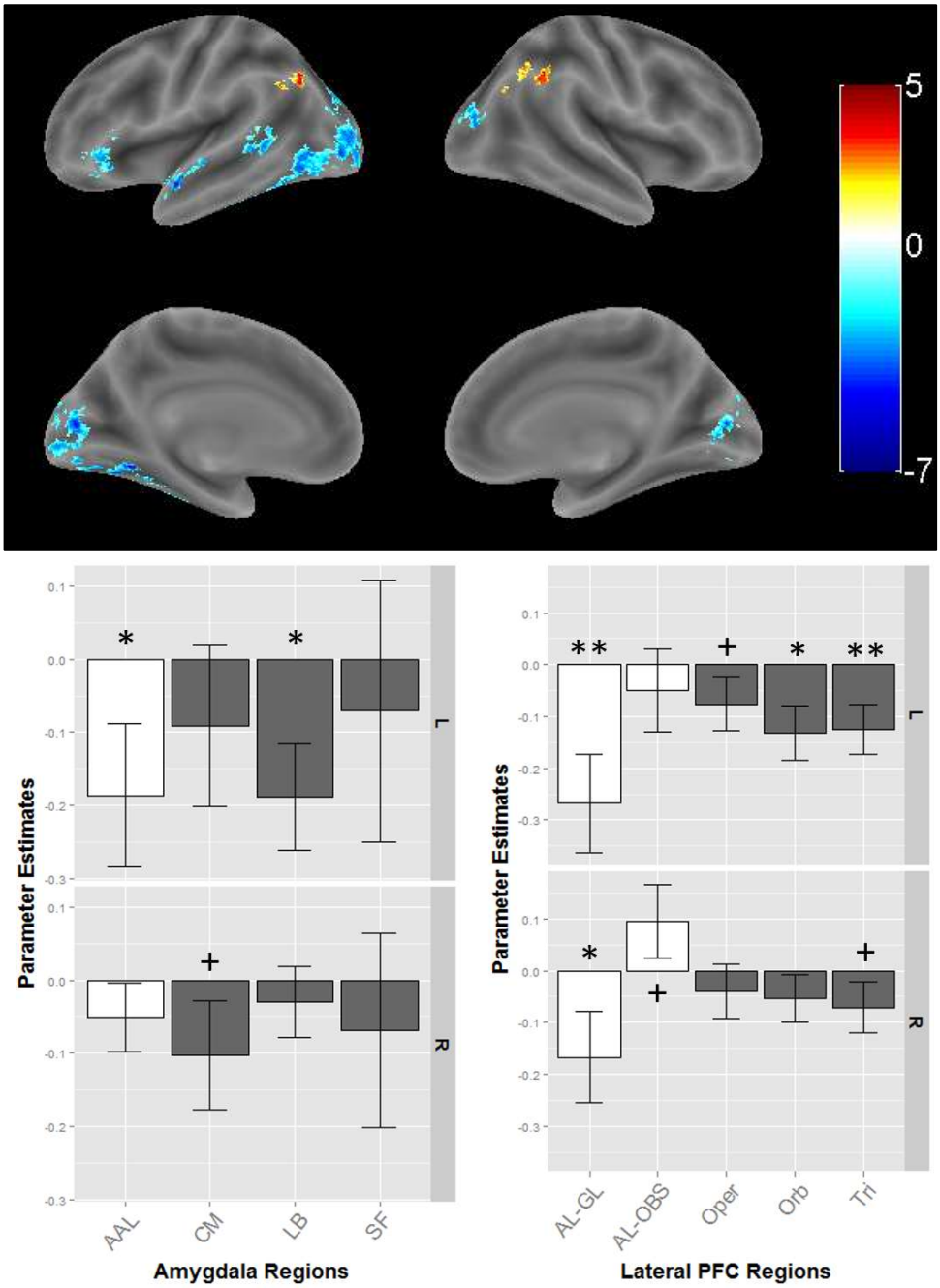
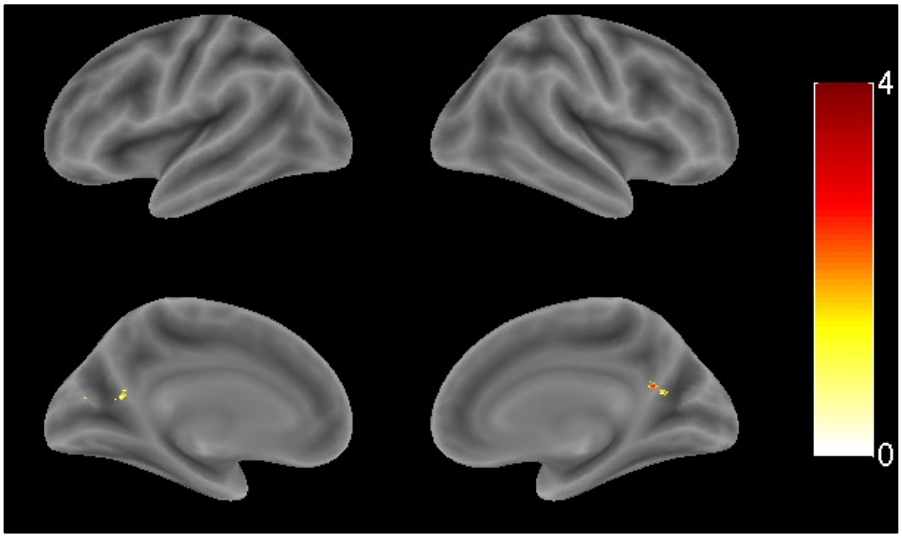
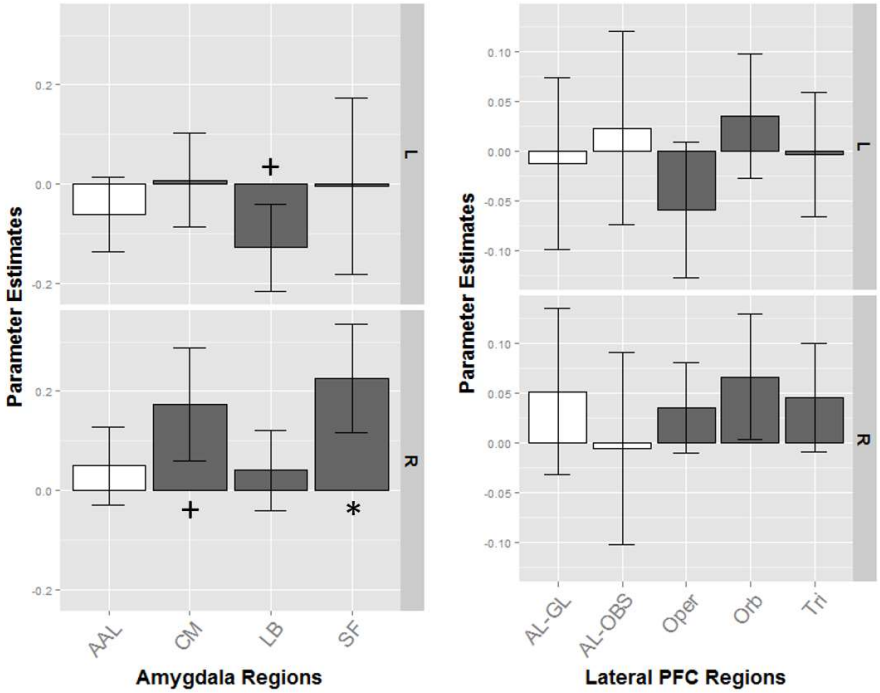


Figure 3.5 – Label Intensity Task Low Intensity > High Intensity Results

Surface rendering of whole-brain results for specific hypothesis in the Label Intensity Task of Low intensity labels > High intensity labels (top) and results of ROI analyses for this contrast for amygdala (bottom left) and lateral PFC (bottom right). For ROI results, results are indicated as significant (* $p < .05$) or trending (+ $p < .1$).





Study 2 – Types of Affect Labeling Task

Over the years of studying affect labeling in the lab or fMRI setting, the paradigms used have taken a number of forms and participants have been instructed to engage in what phenomenologically feel like very distinct tasks that are all considered affect labeling. Participants have been asked to label aversive content (e.g., Lieberman et al., 2011), label the affect they observe in others (e.g., Lieberman et al., 2007), or, in what may be the most intuitive form of affect labeling, they have been instructed to label their own emotions (e.g., Burklund et al., 2014). Yet all forms of the affect labeling paradigm have demonstrated the canonical emotion regulation effects we have come to expect with successful implementation of affect labeling. To date, there has been only one published comparison looking across these prominent and distinct affect labeling paradigms using skin conductance ratings (McRae, Taitano, et al., 2010) and none that have used neuroimaging.

In our second study, we have created a task that allows comparison across these types of affect labeling in the MR scanner and have investigated the commonalities and differences between them. Our primary goal was to identify which of the three major types of affect labeling was most successful at reducing negative affect as judged by reductions in amygdala activity across conditions. To do this we used aversive images from IAPS (Lang et al., 2008) to induce negative affect and ask participants to employ one of the three different types of affect labeling by either labeling their own feelings in response to the scene (Internal Affect), labeling the feelings of the people in the scenes (External Affect), or labeling the aversive content in the scene (External Object). Each type of affect labeling was compared against an Observe condition where participants simply observed the aversive images and allowed themselves and their emotions to respond naturally.

We expected each of the three types of affect labeling to successfully reduce negative affect when compared to Observe by way of amygdala reductions. We also anticipated increased vlPFC activity because of its suggested role in emotion regulation as outlined earlier. In previous research (McRae, Taitano, et al., 2010), it was shown that in some cases only labeling the aversive content objectively produced reduced skin conductance suggesting down-regulation of negative affect. Comparatively, labeling one's own internal feelings was not as effective at reducing skin conductance responses. Given that amygdala mediates threat elicited skin conductance response (Wood, Ver Hoef, & Knight, 2014), we might expect to see similar results with labeling internal affect being the least successful at engaging the regulatory processes involved in affect labeling and labeling the aversive content being the most successful. However, it has been argued that part of what may drive the success of affect labeling is the use of language to convert uncertain stimuli into more knowable and manageable quantities that can be dealt with (Lindquist, Satpute, et al., 2015). To the extent that using objective labels to identify aversive content in a scene reduces our uncertainty about our immediate context less than using language to sift through the whirling buzz of our own internal emotional state (or the emotional states of others), we might instead see that labeling affective states (either your own or others') may have a larger effect on reducing our uncertainty about the aversive scenes before us and thus show even greater reductions in negative affect and amygdala activity with concomitant increased activations in vlPFC.

Results

Whole-brain analysis

Each condition of interest was first compared against the control condition of Observe to assess whole-brain effects of types of affect labeling separately. No amygdala results were observed at the whole-brain level for any of the contrasts analyzed so we will focus on prefrontal activations at the whole-brain instead. In the case of Internal Affect > Observe, activations were observed in the bilateral vlPFC, bilateral dlPFC, dmPFC, and anterior cingulate cortex (ACC). For External Affect > Observe, activations were observed in the bilateral vlPFC, bilateral dlPFC, dmPFC, ACC, and deactivations were observed in the ventromedial prefrontal cortex (vmPFC). For External Object > Observe, some activation was observed in the left dlPFC and deactivations in the vmPFC. Turning to the contrast of Affect > Object, where both affect conditions (Internal Affect and External Affect) were averaged and compared against the External Object condition, increased activations were observed in bilateral vlPFC, dlPFC and in vmPFC. A table with the complete listing of all activations and deactivations observed during whole-brain analysis for this task is available in Appendix A (see Table A2). Visualization of the whole-brain results for this task is available at the end of this chapter (Figures 4.1 and the top of Figure 4.4)

ROI analysis

Each condition of interest was first compared against the control condition Observe. For Internal Affect > Observe, several regions of bilateral amygdala showed decreased activation including the superficial and centromedial amygdala bilaterally as well as the right laterobasal amygdala trending toward significance. As expected, vlPFC showed strong bilateral increased activity during this contrast along with bilateral dlPFC activity. For External Affect > Observe, a

similar pattern emerged with additional regions of the amygdala showing deactivation during the condition including bilateral laterobasal amygdala. As with the previous contrast, External Affect > Observe also demonstrated increased bilateral vlPFC and dlPFC activity. For External Object > Observe, similar amygdala deactivations were observed though they were limited to the left superficial amygdala and bilateral centromedial amygdala. Additionally, vlPFC activity during this affect labeling condition was not demonstrated and in fact one region of right vlPFC (AL-GL) showed increased activity to Observe compared to External Object. In a direct contrast comparing the average of Internal and External Affect against External Object, bilateral vlPFC and dlPFC was more active during the Affect conditions than the Object condition and regions of the left and right amygdala (LB and SF respectively) were less active during Affect than Object.

Discussion

The comparison of the experimental conditions against the control condition Observe paints a very clear picture. In each case we observed decreased amygdala activity and increased vlPFC activity compared to Observe. When comparing the Affect conditions to the Object condition, however, it was revealed that labeling affect as opposed to labeling aversive content may be more effective at engaging vlPFC bilaterally as well as reducing amygdala activity. Despite previous findings suggesting that labeling aversive content objectively may be more effective at engaging the regulatory processes involved in affect labeling, our results suggest the opposite may be true as far as amygdala activity is representative of negative affect. It may be that the gains in decreased uncertainty preventing us for dealing with our immediate context may be granted more from labeling the emotions involved rather than the aversive content we are confronted with.

Figure 4.1 – Types of Affect Labeling Task Whole-brain Results vs. Control

Surface rendering of whole-brain results for conditions of interest against the control condition in the Types of Affect Labeling Task. All rendering done via bspmview software using $p < .005$ voxel-wise threshold and $p < .05$ FEW cluster-level correction.

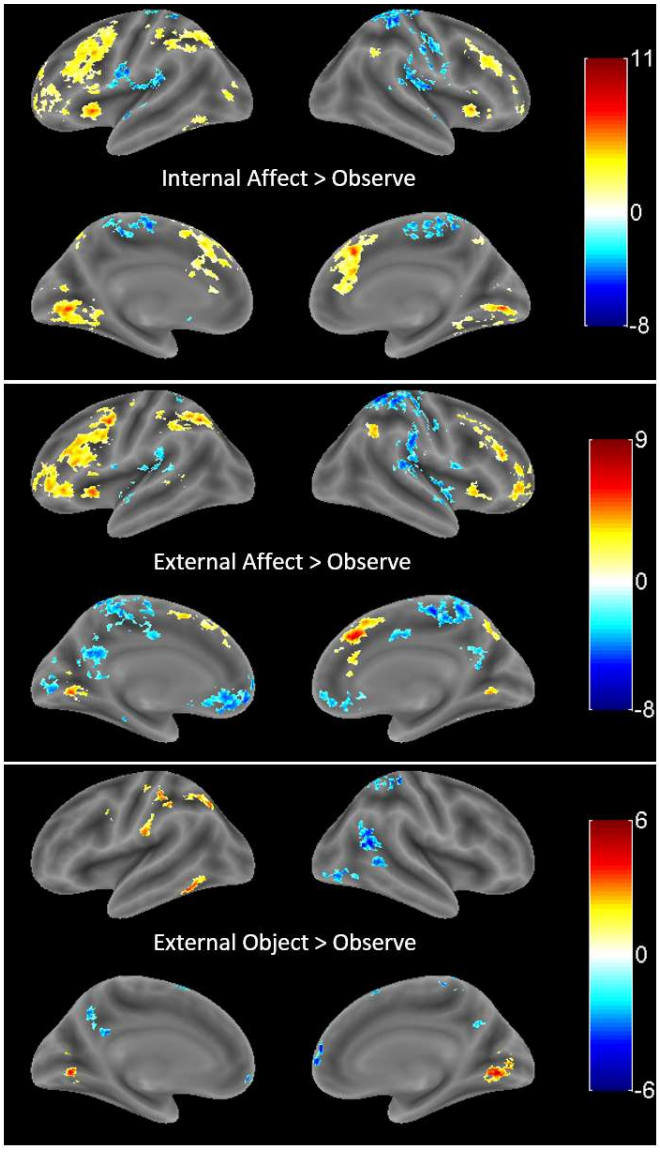


Figure 4.2 – Types of Affect Labeling Task Amygdala ROI Results

Results of the ROI analyses for amygdala. Showing significant (* p < .05; ** p <. 01) or trending (+ p < .1) results for each contrast. White bars represent activity in the single-unit amygdala ROI. Black bars represent activity in the constituent amygdala subregions.

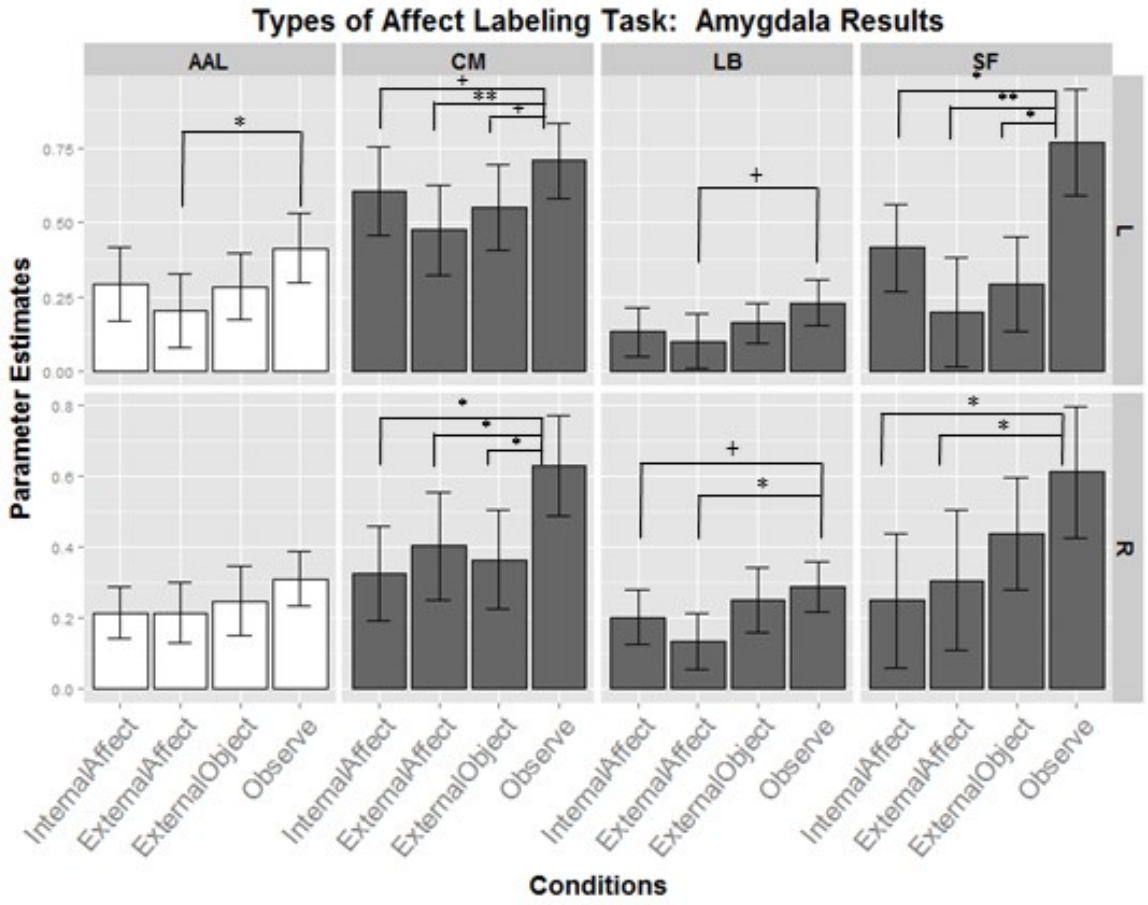


Figure 4.3 – Types of Affect Labeling Task Lateral PFC ROI Results

Results of the ROI analyses for lateral PFC. Showing significant (* p < .05; ** p <. 01) or trending (+ p < .1) results for each contrast. White bars represent data taken from ROIs defined by functional peaks from an existing dataset. Black bars represent data taken from anatomical ROIs.

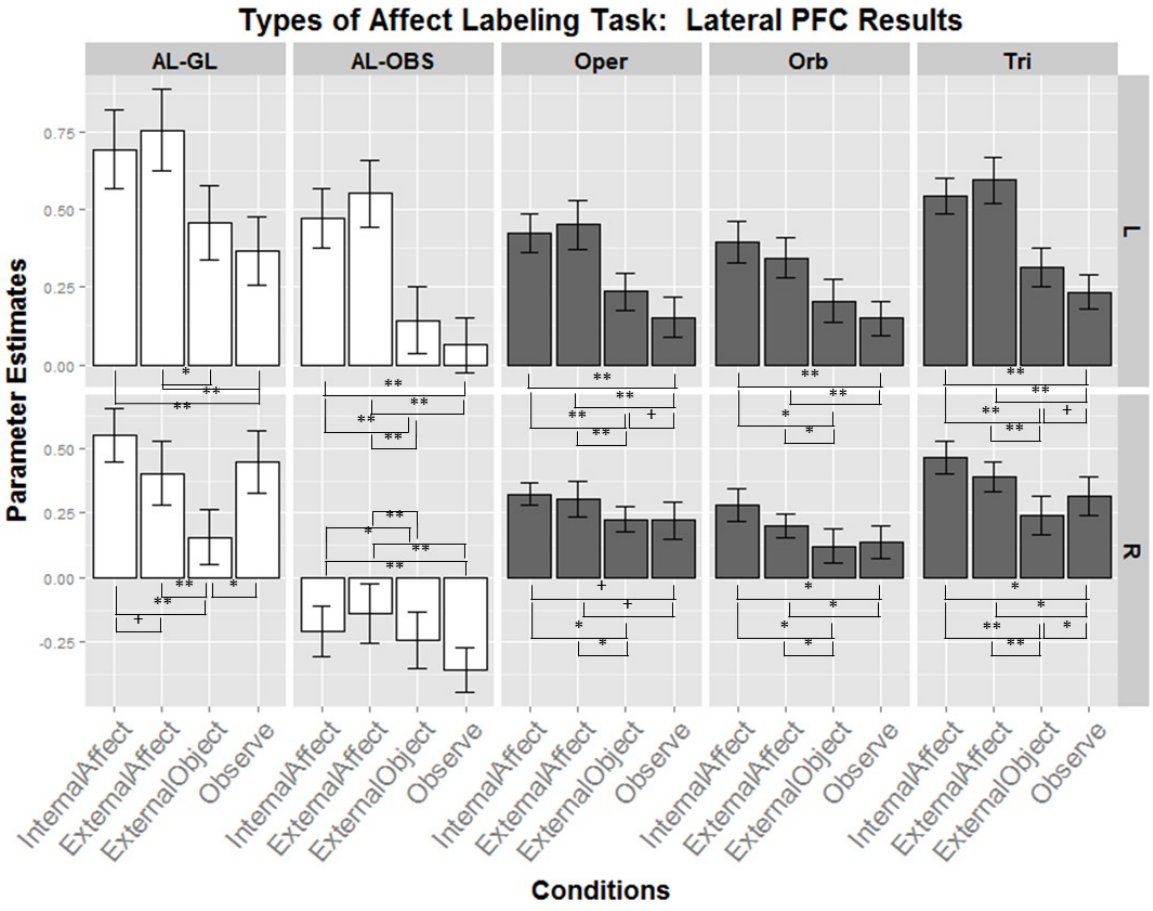
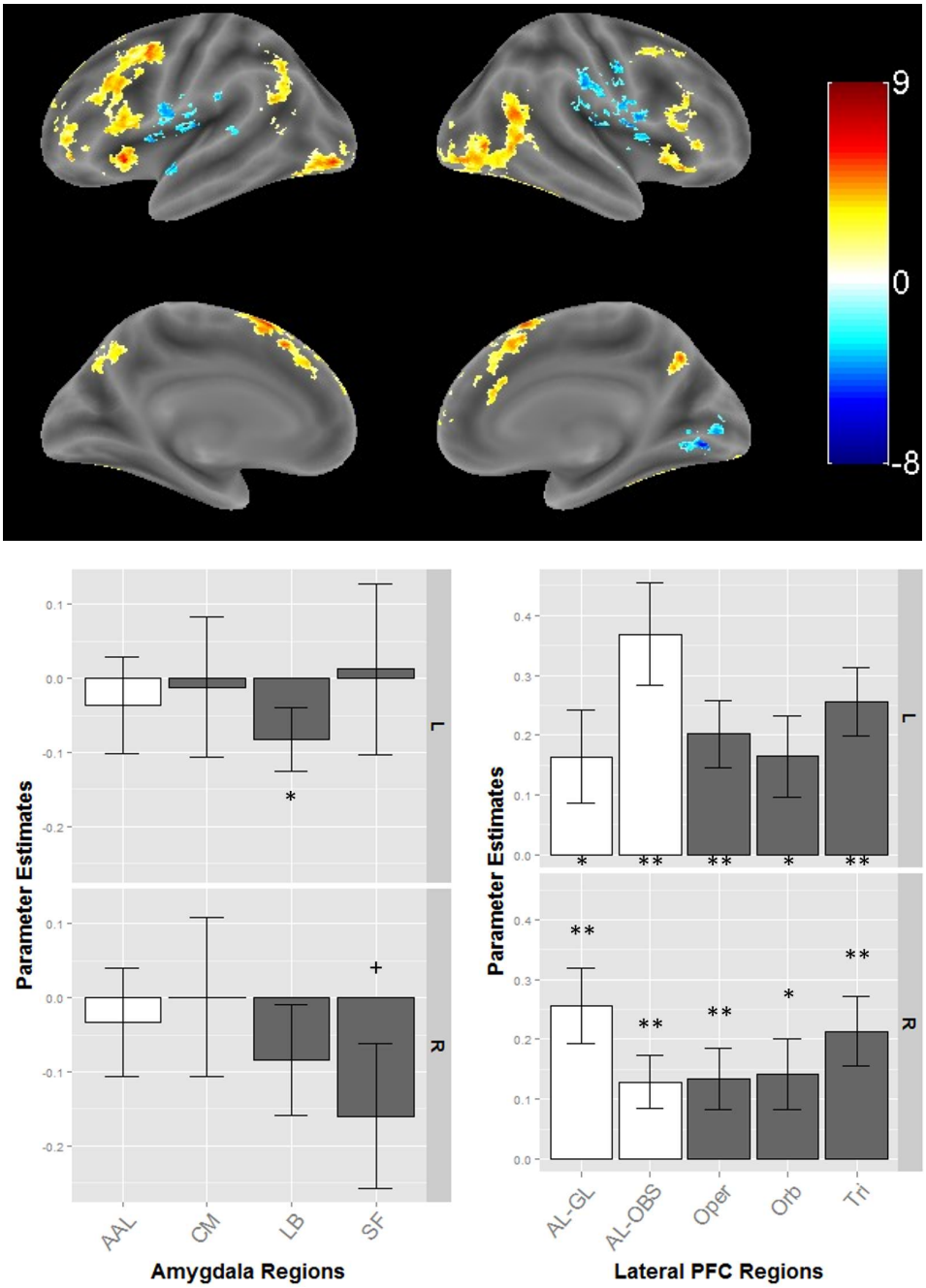


Figure 4.4 – Types of Affect Labeling Task Labeling Affect > Labeling Objects Results

Surface rendering of whole-brain results for the specific hypothesis in the Types of Affect Labeling Task of Affect labeling > Aversive content labeling (top) and results of ROI analyses for this contrast for amygdala (bottom left) and lateral PFC (bottom right). For ROI results, results are indicated as significant (* $p < .05$) or trending (+ $p < .1$).



Study 3 – Number of Responses Task

In many cases outlined earlier, affect labeling has been shown to reduce experienced negative affect in the moment. However, for a number of studies affect labeling either failed to produce immediate down-regulation of affect or in some cases produced an increase in affect. This disparity may be explained by the way affect labeling was implemented. In the former cases where affect labeling had immediate effects, participants were instructed to choose from a list of labels provided to them when engaging in affect labeling. In many of the latter cases where affect labeling failed to provide an immediate effect (but often provided a delayed and even sustained effect as in expressive writing paradigms), participants were instructed to self-generate the affective labels themselves. As discussed earlier, additional processes brought online by self-generating labels may adversely impact the success of affect labeling. To test this, we designed a study that will allow direct comparison of different modes of affect label generation in response to aversive IAPS images. We compared the traditional two label choice paradigm (Two Labels) against a free response condition (Free Response) where participants are instructed to generate the word themselves (though they were constrained to the words 'angry', 'scared', 'sad', 'disgusted', or 'happy' to avoid confounds as explained in the general methods). We also included a novel form of affect label presentation whereby only one word was presented (One Label) and participants were instructed to indicate that this word either did or did not capture a major component of their emotional response to the aversive scene. Each of these conditions that varied in the number of responses provided was compared against the Observe condition where participants were instructed to attend to the aversive scene and allow themselves and their emotions to respond naturally.

Given that all three experimental conditions of interest are forms of affect labeling, we might expect that each should, when compared to Observe, demonstrate the canonical effects of affect labeling which are decreased amygdala activity and increased vlPFC activity. However, given the number of studies which have used self-generated (or free response) affect labeling and have shown increased reported negative affect, we might also expect that the Free Response condition would not show decreased amygdala activity and may instead show increased activity. We might also expect to see increased activity in vlPFC for the Free Response condition compared to Observe despite an increase (or lack of decrease) in the amygdala as this region is brought online to regulate amygdala but is disrupted or otherwise unsuccessful.

Results

Whole-brain results

Each condition of interest was first compared against the control condition of Observe to assess whole-brain effects of the number of affect labels provided separately. No amygdala results were observed at the whole-brain level for any of the contrasts analyzed so we will focus on prefrontal activations at the whole-brain instead. In the case of One Label > Observe, activations were observed in the bilateral vlPFC, bilateral dlPFC, dmPFC, and ACC. For Two Labels > Observe, activations were observed in the bilateral vlPFC, bilateral dlPFC, dmPFC, and ACC. For Free Response > Observe, activations were also observed in the bilateral vlPFC, bilateral dlPFC, dmPFC, and ACC. Turning to the contrast of Provided Labels > Free Response, where both provided label conditions (One Label and Two Labels) were averaged and compared against the Free Response condition, no increases or decreases were observed in the prefrontal cortex at the whole-brain level. A table with the complete listing of all activations and

deactivations observed during whole-brain analysis for this task is available in Appendix A (see Table A3).

ROI results

Each condition was first compared to the Observe control condition. For One Label > Observe, significant bilateral reductions in amygdala occurred in every subregion and activity in bilateral vlPFC and dlPFC increased. For Two Label > Observe, a similar pattern emerged of bilateral decreases in the amygdala and bilateral increases in vlPFC and dlPFC. For Free Response > Observe, many regions of bilateral vlPFC and dlPFC were significant, however only the left superficial amygdala showed decreased activity. When comparing the average of the label conditions against the free response condition, we see deactivation in the right amygdala for the label conditions including laterobasal and centromedial and some increased activity in right vlPFC as well.

Discussion

Compared to the Observe control condition, each affect labeling condition in this task demonstrated a successful reduction in amygdala activity and increases in vlPFC activity. When turning to the comparison of self-generated (Free Response) versus provided labels (One Label + Two Labels), it appears that being provided affect labels may be more effective at reducing amygdala activity and increasing vlPFC activity. This result may help to explain the disparity in the literature whereby some studies which had participants self-generate affect labels reported either no decreased negative affect or instead increased negative affect. Additionally, the Free Response condition was not 'unfettered' in this paradigm as participants were instructed to choose from 'angry', 'scared', 'sad', 'disgusted', or 'happy'; it remains to be seen how

successful affect labeling would (or would not) be when participants are not constrained in such a manner.

Figure 5.1 – Number of Responses Task Whole-brain Results vs. Control

Surface rendering of whole-brain results for conditions of interest against the control condition in the Number of Responses Task. All rendering done via bspmview software using $p < .005$ voxel-wise threshold and $p < .05$ FEW cluster-level correction.

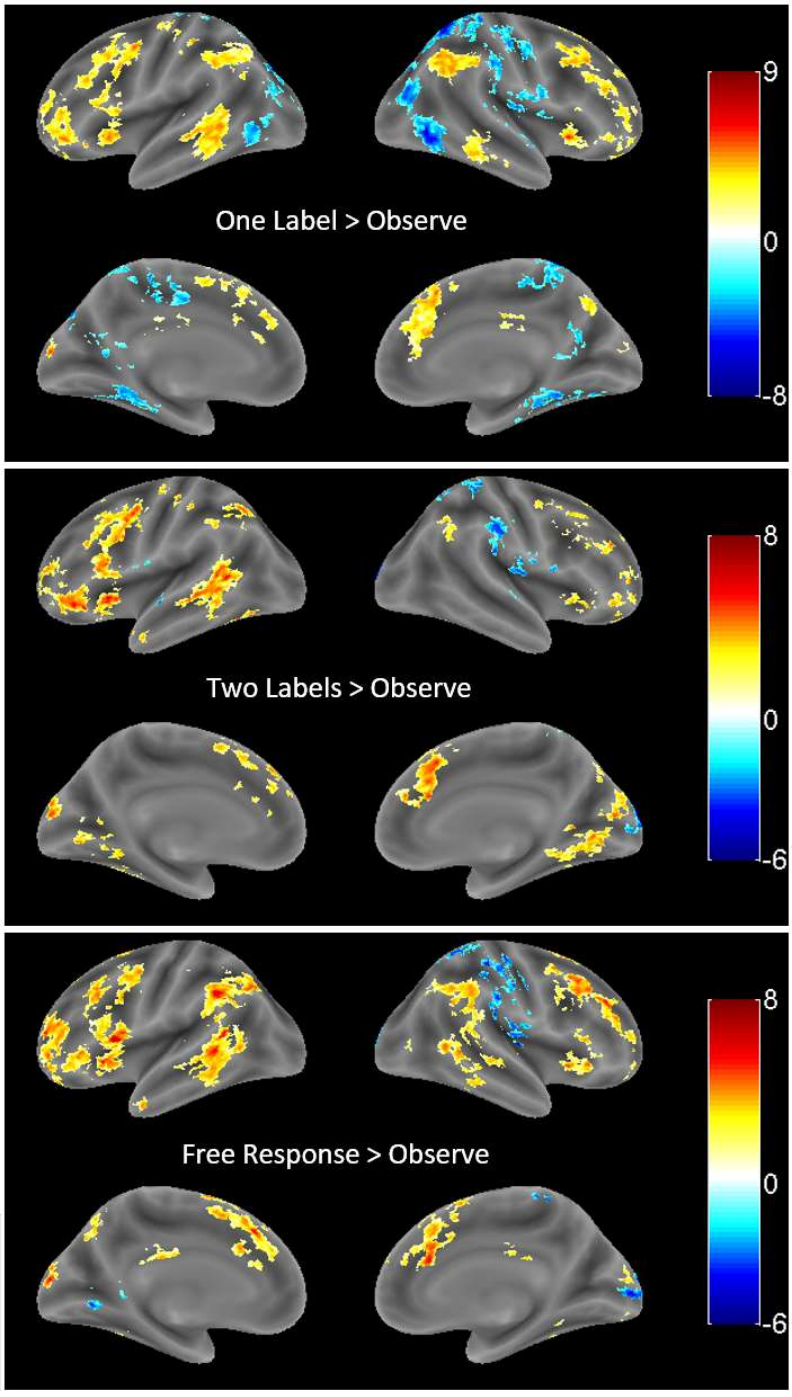


Figure 5.2 – Number of Responses Task Amygdala ROI Results

Results of the ROI analyses for amygdala. Showing significant (* p < .05; ** p <. 01) or trending (+ p < .1) results for each contrast. White bars represent activity in the single-unit amygdala ROI. Black bars represent activity in the constituent amygdala subregions.

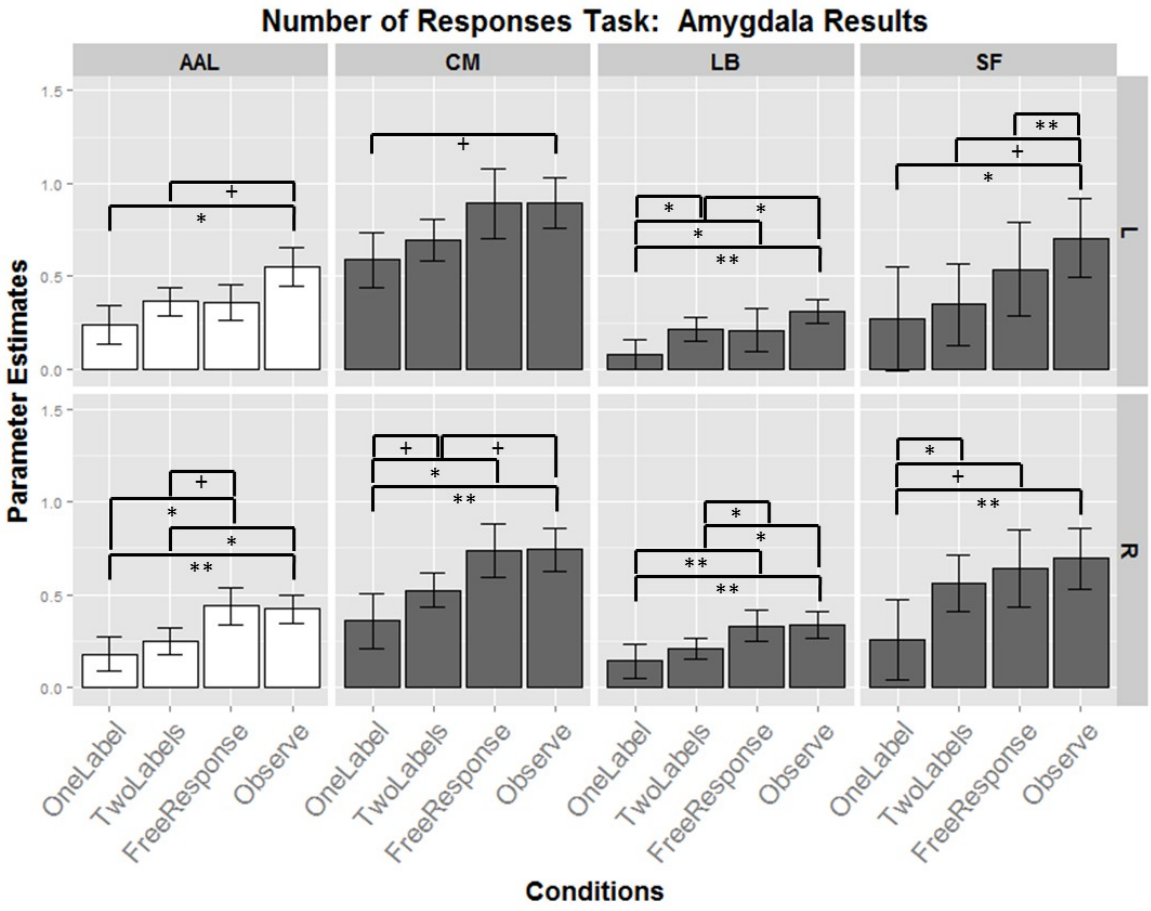


Figure 5.3 – Number of Responses Task Lateral PFC ROI Results

Results of the ROI analyses for lateral PFC. Showing significant (* p < .05; ** p <. 01) or trending (+ p < .1) results for each contrast. White bars represent data taken from ROIs defined by functional peaks from an existing dataset. Black bars represent data taken from anatomical ROIs.

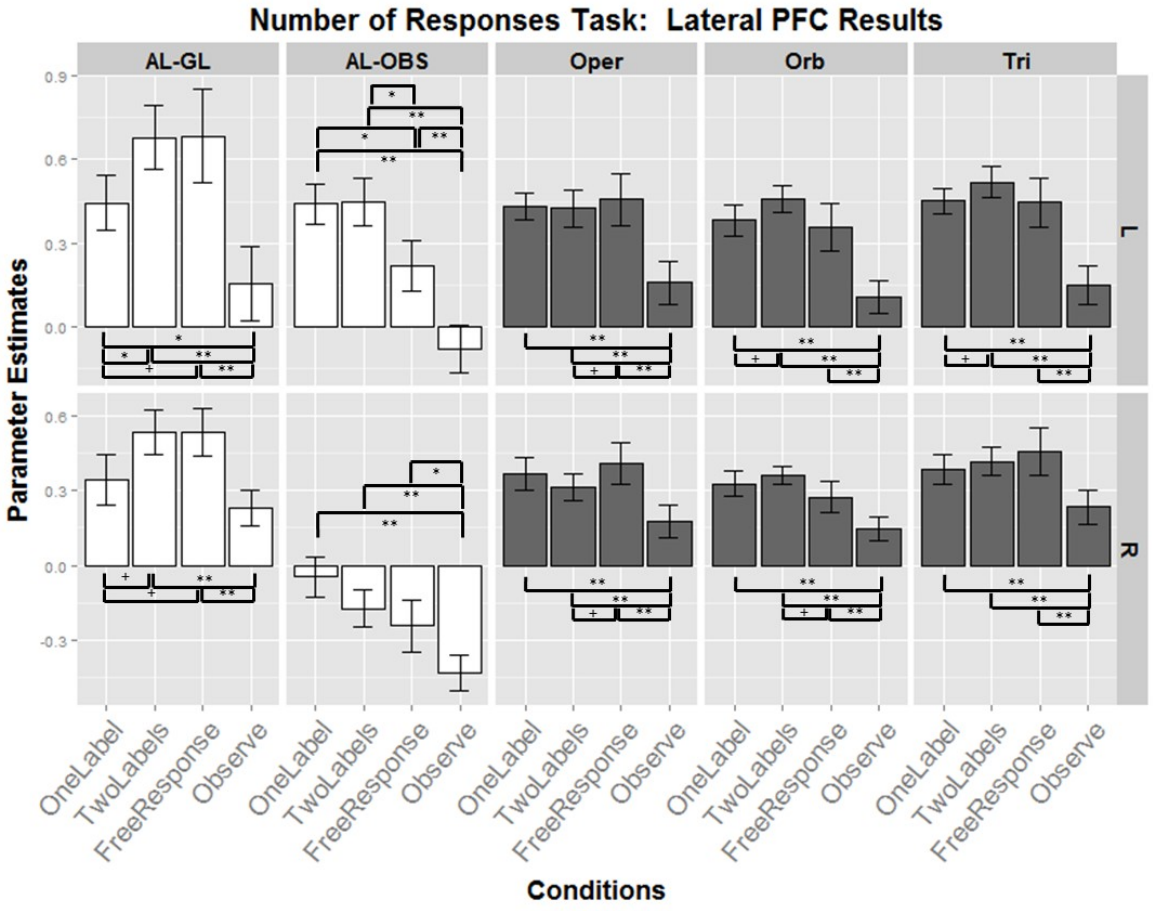
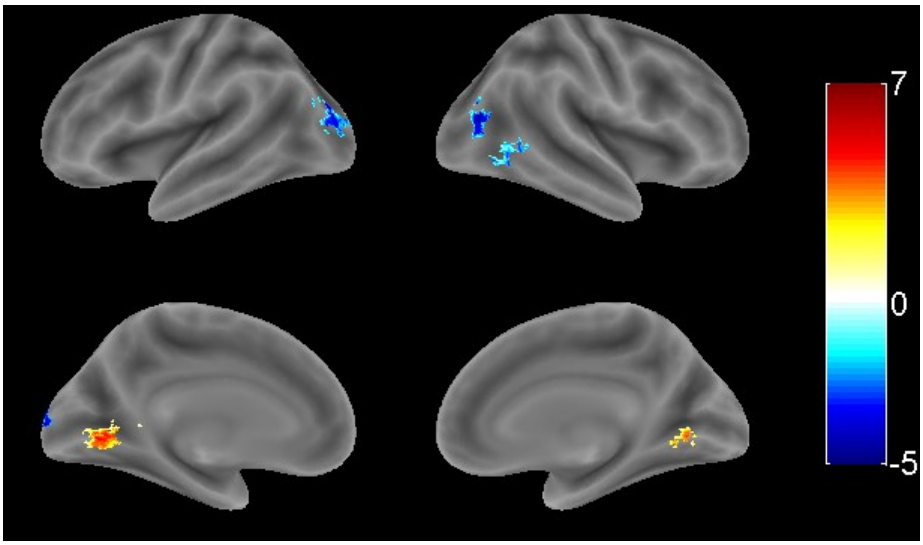
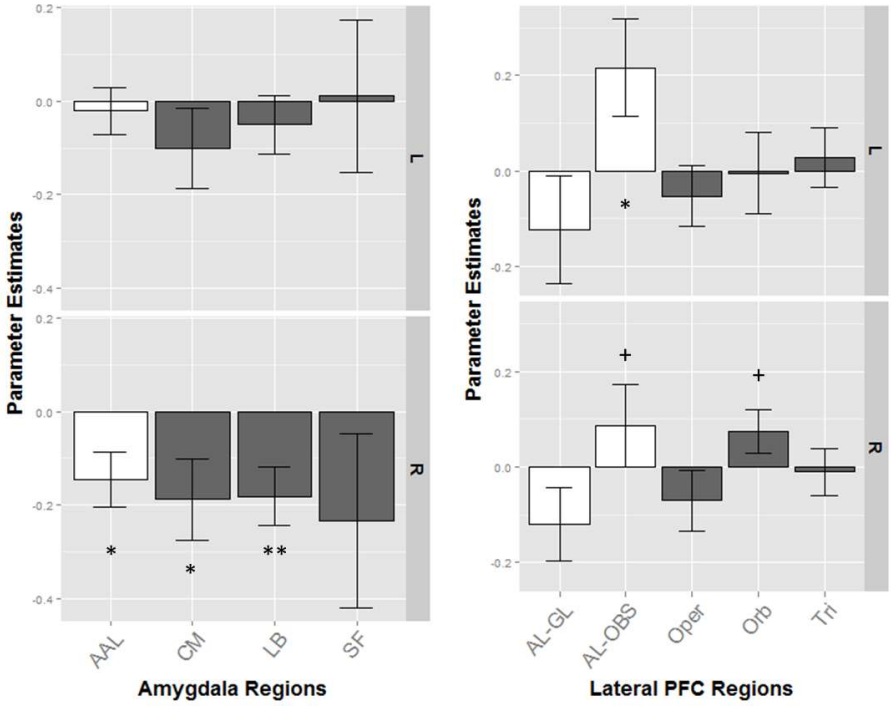


Figure 5.4 – Number of Responses Task Provided Labels > Free Response Results

Surface rendering of whole-brain results for the specific hypothesis in the Number of Responses Task of Provided Labels > Free Response (top) and results of ROI analyses for this contrast for amygdala (bottom left) and lateral PFC (bottom right). For ROI results, results are indicated as significant (* $p < .05$) or trending (+ $p < .1$).





General Discussion

The purpose of this dissertation was to further our understanding of affect labeling as a form of implicit emotion regulation by investigating three questions: 1) what are the effects of label intensity on affect labeling success, 2) of the many types of affect labeling which are more or less effective at reducing emotional experience, and 3) does self-generation of affect labels enhance or disrupt the regulatory process involved in affect labeling?

We found that it may not be label intensity that impacts the success of affect labeling, but rather the specificity of the emotion words used. Using more abstract emotion words (such as 'angry' rather than 'irritated' or 'enraged') yields larger decreases in amygdala activity when used in an affect labeling paradigm while varying the intensity of the words did not. We also found that among the many types of affect labeling including labeling your own emotions, labeling others' emotions, or labeling aversive content, paradigms that encourage the labeling of affect as opposed to aversive content may be more effective at reducing amygdala activity and thus negative affect. Finally, we found that while self-generated affect labels may still be effective at reducing negative affect, having labels provided yields a more robust decrease in amygdala activity, whether the labels are presented as a choice as per the typical affect labeling paradigm or even as a single label that is to be evaluated.

Limitations and Future Directions

Despite several promising findings, there are some limitations to the current research. Firstly, all tasks were performed by the same participants in a single session. Although tasks were counterbalanced to avoid order effects, the amygdala is known for its rapid habituation to negatively valenced or aversive stimuli (Breiter et al., 1996; Fischer et al., 2003; Plichta et al.,

2014; Wright et al., 2001). Future research with these paradigms would be best served by using different participants to investigate these effects further.

Secondly, even for a neuroimaging study our number of subjects was quite low (N = 20) leaving us underpowered to obtain reliable true effects. Recruitment and data collection are still underway so we expect this to be remedied soon.

Thirdly, as with most laboratory studies, the stimuli used were lacking in ecological validity. While many of the images depict real events, they are ultimately images of events distant from oneself. It remains to be seen how affect labeling processes are engaged or altered when confronted more directly or more personally impactful. The research on expressive writing, which at least superficially seems to share a great deal with affect labeling as they both require the act of putting feelings into words, suggests that affect labeling may still be successful in these contexts as well.

Finally, a major motivation of this work was to directly compare several different forms of affect labeling and investigate which is most effective at reducing negative affect by observing amygdala activity. However, as detailed earlier we have seen in the research on expressive writing and other research in the laboratory investigating affect labeling that oftentimes the value of an emotion regulation strategy is how it can help you maintain healthy living in the long-term. The studies conducted for the current research were focus on immediate reductions in amygdala activity, but continuing this line of research and seeing which form of affect labeling is most successful in the long-term or which is more flexible and successful against the widest variety of aversive stimuli is an important direction for this research to take.

Appendix A – Tables of Whole-brain Results

Table A1 – Whole-brain Results from Label Intensity Task

Table shows all local maxima separated by more than 20 mm. Regions were automatically labeled using the SPM Anatomy Toolbox atlas. x y and z =Montreal Neurological Institute (MNI) coordinates in the left-right anterior-posterior and inferior-superior dimensions respectively. Clusters were corrected for multiple comparisons by cluster-level FWE correction at $p < .05$.

				MNI Coordinates		
Contrast Name	Region Label	Extent	t-value	x	y	z
Low> Gender	L Calcarine Gyrus	2933	9.3411	-6	-90	2
	L Middle Occipital Gyrus	2933	6.7789	-26	-92	6
	R Calcarine Gyrus	2933	6.2842	20	-102	2
	L Middle Occipital Gyrus	2933	4.6197	-24	-94	26
	Location not in atlas	2933	4.589	-42	-84	-12
	L Cerebelum (VI)	2933	4.3572	-20	-78	-16
	L Linual Gyrus	2933	4.3244	-18	-102	-10
	R Inferior Occipital Gyrus	2933	4.2483	36	-90	-6
	Cerebellar Vermis (7)	2933	3.9748	4	-82	-24
	R Middle Occipital Gyrus	2933	3.0197	28	-90	26
	Cerebellar Vermis (6)	286	6.8743	6	-70	-18
	R Cerebelum (VI)	286	5.0887	26	-68	-18
	R Cerebelum (Crus 1)	286	4.6635	42	-62	-30
	L IFG (p. Orbitalis)	218	5.616	-32	26	2
	L Temporal Pole	218	4.3292	-48	16	-6
	R IFG (p. Orbitalis)	107	5.4314	44	26	2
Gender > Low	L Middle Temporal Gyrus	817	-2.9531	-64	-20	-12
	L Superior Medial Gyrus	766	-7.0089	-6	50	18
	L Superior Frontal Gyrus	766	-5.4247	-18	44	38
	R Mid Orbital Gyrus	766	-3.894	12	52	2
	L Middle Temporal Gyrus	817	-6.0274	-66	-36	-8
	Location not in atlas	817	-5.2508	-62	-18	-26
	Location not in atlas	817	-4.2698	-60	2	-26
	L Middle Frontal Gyrus	283	-5.5796	-44	18	44
	L Superior Frontal Gyrus	283	-3.3318	-22	32	52
	R Angular Gyrus	397	-5.2323	46	-56	28
	R Inferior Parietal Lobule	397	-4.7554	56	-56	46

	R Angular Gyrus	397	-3.4558	46	-74	38
	L Inferior Parietal Lobule	451	-4.967	-54	-46	48
	L Angular Gyrus	451	-4.8842	-48	-58	30
	L Angular Gyrus	451	-3.539	-42	-70	46
	R Middle Frontal Gyrus	220	-4.795	46	18	42
	R Middle Frontal Gyrus	220	-3.7907	28	40	36
	R Middle Frontal Gyrus	220	-3.2844	24	20	44
High > Gender	L Precentral Gyrus	159	6.2843	-40	0	40
	L Cerebelum (VI)	182	5.8459	-16	-80	-12
	L Fusiform Gyrus	182	3.4681	-34	-70	-8
	Location not in atlas	213	5.1685	22	-64	-26
	Cerebellar Vermis (6)	213	2.9228	2	-60	-16
	L Cerebelum (VI)	105	4.8701	-26	-52	-24
	L IFG (p. Opercularis)	150	4.6593	-42	14	20
Gender > High	L Inferior Temporal Gyrus	708	-5.685	-62	-16	-22
	L Middle Temporal Gyrus	708	-3.6018	-62	-48	-8
	R Calcarine Gyrus	240	-5.5886	8	-62	22
	R Rectal Gyrus	292	-5.2867	2	26	-20
	L Superior Orbital Gyrus	292	-3.8448	-10	10	-18
	L Rectal Gyrus	292	-3.2869	-4	50	-14
	R Angular Gyrus	512	-5.1702	56	-64	32
	R Middle Temporal Gyrus	512	-4.5476	60	-48	16
	R Angular Gyrus	512	-4.2958	40	-72	46
	R Inferior Parietal Lobule	512	-3.4544	54	-46	54
	R Middle Temporal Gyrus	326	-5.1175	60	-18	-12
	R Medial Temporal Pole	326	-4.3382	58	4	-26
	R Inferior Temporal Gyrus	326	-3.2455	64	-36	-20
	L Mid Orbital Gyrus	367	-5.1101	-10	48	2
	R Mid Orbital Gyrus	367	-4.8258	14	46	0
	L Superior Medial Gyrus	367	-3.792	-2	64	12
	L Angular Gyrus	334	-5.1083	-52	-70	30
	L SupraMarginal Gyrus	334	-4.0709	-60	-52	40
	L Precuneus	99	-4.5323	-4	-56	44
	L Middle Frontal Gyrus	92	-4.4537	-28	16	60
	L Middle Frontal Gyrus	92	-3.1951	-44	18	42
	L Superior Medial Gyrus	95	-4.3258	-6	54	24
Abstract > Gender						
	No results to report.					
Gender > Abstract	L Middle Frontal Gyrus	102	-5.3498	-44	20	46
	Location not in atlas	292	-5.3309	-62	-16	-26

	L Superior Temporal Gyrus	292	-3.969	-60	-10	0
	L Inferior Temporal Gyrus	292	-3.6858	-58	-34	-18
	L Superior Medial Gyrus	244	-4.5034	-6	54	24
	L Superior Medial Gyrus	244	-4.1714	-4	42	46
Abstract > Specific	L Angular Gyrus	187	5.1864	-54	-62	40
	L Angular Gyrus	187	4.5336	-40	-68	40
	L Middle Occipital Gyrus	187	3.7397	-42	-80	36
	L Middle Temporal Gyrus	187	3.1881	-58	-56	26
	R Middle Occipital Gyrus	249	4.7205	46	-70	34
	R Angular Gyrus	249	4.6217	44	-58	42
	R SupraMarginal Gyrus	249	4.2056	54	-50	42
	R Superior Temporal Gyrus	249	3.8055	62	-56	26
	R SupraMarginal Gyrus	249	3.3514	64	-48	38
Specific > Abstract	L Middle Occipital Gyrus	2324	-2.9597	-30	-90	6
	L Fusiform Gyrus	2324	-7.5069	-28	-54	-4
	R Calcarine Gyrus	2324	-5.8069	12	-76	12
	L Linual Gyrus	2324	-5.5398	-6	-84	8
	L Inferior Temporal Gyrus	2324	-5.4199	-42	-50	-12
	Location not in atlas	2324	-5.1648	-44	-32	-24
	L Fusiform Gyrus	2324	-5.0451	-30	-64	-10
	L Middle Occipital Gyrus	2324	-5.0318	-32	-92	22
	L Inferior Temporal Gyrus	2324	-5.0006	-46	-64	-4
	L Fusiform Gyrus	2324	-4.9906	-32	-46	-16
	L Cuneus	2324	-4.9457	-4	-96	22
	L Calcarine Gyrus	2324	-4.8326	-2	-94	2
	L Middle Occipital Gyrus	2324	-4.8077	-34	-86	4
	L Cerebelum (VI)	2324	-4.635	-18	-62	-12
	L Cerebelum (VI)	2324	-4.6302	-12	-82	-10
	R Cerebelum (VI)	2324	-4.4348	8	-68	-8
	L Fusiform Gyrus	2324	-4.3322	-42	-40	-14
	L Cuneus	2324	-4.3006	4	-86	26
	L Cerebelum (VI)	2324	-4.2757	-22	-74	-12
	L Linual Gyrus	2324	-4.2443	-30	-86	-8
	Location not in atlas	2324	-4.0116	-22	-84	22
	Cerebellar Vermis (4/5)	2324	-3.911	-2	-68	2
	L Linual Gyrus	2324	-3.7955	-12	-92	-2
	R Calcarine Gyrus	2324	-3.719	18	-84	10
	L Inferior Occipital Gyrus	2324	-3.6554	-42	-78	-4
	L Linual Gyrus	2324	-3.6539	-24	-94	-10
	L Middle Occipital Gyrus	2324	-3.5278	-24	-96	10
	L Calcarine Gyrus	2324	-3.3995	-10	-102	-4
	L Fusiform Gyrus	2324	-3.3805	-34	-38	-22

	R Linual Gyrus	2324	-3.3296	14	-82	-8
	Location not in atlas	2324	-3.2368	-28	-80	14
	L Inferior Temporal Gyrus	2324	-3.0637	-40	-20	-20
	L Middle Temporal Gyrus	215	-6.3522	-56	-48	6
	Location not in atlas	215	-5.3478	-42	-44	4
	L Middle Temporal Gyrus	146	-5.8528	-52	-4	-16
	L Temporal Pole	146	-4.564	-56	4	-10
	L Middle Temporal Gyrus	146	-3.8459	-64	-12	-8
	L IFG (p. Orbitalis)	258	-5.1957	-34	26	-6
	L IFG (p. Orbitalis)	258	-4.0496	-48	16	-4
	L IFG (p. Orbitalis)	258	-3.8579	-48	30	-2
	Location not in atlas	258	-3.8245	-42	30	-14
	L IFG (p. Orbitalis)	258	-3.3127	-42	38	-6
	L IFG (p. Triangularis)	258	-3.2299	-32	28	8
	L IFG (p. Triangularis)	258	-3.0628	-48	24	8
	Location not in atlas	189	-5.0121	30	-74	12
	Location not in atlas	189	-4.5162	28	-86	16
	R Middle Occipital Gyrus	189	-3.853	42	-84	14
Low > High	R Precuneus	83	4.4788	12	-54	20
	L Calcarine Gyrus	83	3.539	-8	-66	16
High > Low						
	No results to report.					

Table A2 – Whole-brain Results from Types of Affect Labeling Task

Table shows all local maxima separated by more than 20 mm. Regions were automatically labeled using the SPM Anatomy Toolbox atlas. x y and z =Montreal Neurological Institute (MNI) coordinates in the left-right anterior-posterior and inferior-superior dimensions respectively. Clusters were corrected for multiple comparisons by cluster-level FWE correction at $p < .05$.

				MNI Coordinates		
Contrast Name	Region Label	Extent	t-value	x	y	z
Internal Affect > Observe	R MCC	3128	11.871	8	26	36
	L posterior-medial frontal	3128	5.9949	-10	10	54
	R ACC	3128	5.8583	12	32	16
	L Superior Frontal Gyrus	3128	5.597	-16	56	22
	L Middle Orbital Gyrus	3128	5.3827	-26	54	-4
	L Middle Frontal Gyrus	3128	4.8988	-44	46	12
	L Superior Medial Gyrus	3128	4.5023	-6	46	42
	R Superior Frontal Gyrus	3128	4.3151	16	30	54
	Location not in atlas	3128	4.2593	-8	30	12
	Location not in atlas	3128	3.457	-10	14	28
	L Superior Medial Gyrus	3128	3.3034	-6	26	66
	R Linual Gyrus	4472	10.1894	2	-78	4
	R Cerebelum (Crus 1)	4472	7.3941	8	-80	-22
	L Cerebelum (VI)	4472	6.3565	-16	-72	-8
	R Cerebelum (VI)	4472	5.9399	28	-64	-26
	L Fusiform Gyrus	4472	5.6603	-28	-52	-8
	R Cerebelum (Crus 2)	4472	5.2825	52	-50	-34
	R Cerebelum (VIII)	4472	5.1381	40	-54	-50
	R Fusiform Gyrus	4472	5.1075	28	-42	-16
	R Cerebelum (IV-V)	4472	4.3636	8	-58	-2
	R Cerebelum (Crus 1)	4472	4.1639	28	-84	-20
	R Cerebelum (Crus 2)	4472	4.097	32	-80	-40
	L Cerebelum (Crus 1)	4472	3.933	-18	-86	-24

	L Insula Lobe	3391	8.1775	-34	18	-2
	L IFG (p. Triangularis)	3391	7.158	-38	12	36
	L Middle Frontal Gyrus	3391	5.441	-40	34	24
	L IFG (p. Orbitalis)	3391	5.2218	-48	34	-10
	L Middle Frontal Gyrus	3391	4.8709	-36	6	60
	L IFG (p. Triangularis)	3391	4.7118	-54	16	4
	Location not in atlas	3391	4.3366	-22	-6	44
	Location not in atlas	545	7.1656	30	22	2
	R IFG (p. Orbitalis)	545	5.2185	46	26	-10
	L Inferior Parietal Lobule	1804	6.9802	-48	-34	44
	L Inferior Parietal Lobule	1804	6.8635	-36	-62	48
	R Precuneus	1804	4.737	4	-66	50
	L Superior Parietal Lobule	1804	3.2863	-16	-78	52
	R Caudate Nucleus	206	6.5312	14	18	6
	Location not in atlas	206	4.1719	16	-2	18
	L Cerebelum (Crus 1)	352	6.2984	-32	-60	-30
	L Cerebelum (VII)	352	4.9259	-46	-58	-46
	L Inferior Temporal Gyrus	249	6.2584	-50	-54	-18
	L Cerebelum (VI)	249	3.9126	-36	-40	-24
	R Middle Frontal Gyrus	992	6.0801	40	30	28
	R Middle Frontal Gyrus	992	5.7574	42	20	50
	Location not in atlas	992	4.5634	36	58	20
	L Thalamus	206	5.8862	-12	-20	10
	Location not in atlas	206	4.8579	-10	-20	10
	Location not in atlas	206	3.5626	0	0	10
	Location not in atlas	91	5.5705	6	-14	-8
	L Middle Occipital Gyrus	101	4.9403	-48	-78	10
	R Inferior Parietal Lobule	181	4.8718	44	-52	54
	Location not in atlas	113	4.1862	44	54	-6
Observe > Internal Affect	Location not in atlas	2440	8.015	26	-40	50
	Location not in atlas	2440	5.8663	22	-26	66
	L posterior-medial frontal	2440	5.7917	-4	-16	56
	R Paracentral Lobule	2440	5.7061	4	-36	62
	R Precentral Gyrus	2440	5.5935	44	-14	42
	R Postcentral Gyrus	2440	5.4882	64	-6	26
	Location not in atlas	2440	4.3974	14	-14	46
	R Postcentral Gyrus	2440	3.3234	38	-42	66

	R posterior-medial frontal	2440	3.0782	8	-12	76
	Location not in atlas	363	7.1977	-22	-38	54
	Location not in atlas	391	6.6874	-4	4	-12
	Location not in atlas	391	4.6085	0	14	6
	R Amygdala	391	4.1501	20	-4	-12
	L Superior Temporal Gyrus	445	6.2344	-42	-38	18
	L Insula Lobe	445	5.5182	-40	-16	18
	L Superior Temporal Gyrus	450	5.9207	-58	0	2
	L Precentral Gyrus	450	4.8853	-56	-2	22
	R Superior Temporal Gyrus	834	5.8957	54	-6	2
	R Insula Lobe	834	5.7598	40	-14	22
	R Superior Temporal Gyrus	834	4.4379	66	-24	12
	R Superior Temporal Gyrus	834	3.7599	42	-38	22
	R Insula Lobe	834	3.7438	40	8	-8
	R Medial Temporal Pole	834	3.4501	60	6	-14
	R Postcentral Gyrus	834	3.1275	64	-18	34
	L IFG (p. Triangularis)	3391	2.8892	-44	28	26
	L IFG (p. Orbitalis)	3391	3.8237	-36	20	-6
	L Inferior Parietal Lobule	1804	2.9352	-32	-54	44
	L Superior Medial Gyrus	3128	3.4896	-2	24	44
External Affect > Observe	R MCC	1287	9.9679	8	28	36
	L posterior-medial frontal	1287	8.0743	-4	18	50
	R ACC	1287	4.7416	6	36	12
	L Superior Medial Gyrus	1287	4.6664	-8	42	42
	L Inferior Parietal Lobule	1200	9.6833	-30	-56	48
	L Inferior Parietal Lobule	1200	6.4323	-50	-34	48
	Cerebellar Vermis (4/5)	1339	8.0147	-2	-66	-2
	R Cerebelum (Crus 2)	1339	7.8949	8	-78	-24
	L Cerebelum (Crus 1)	1339	6.0486	-14	-84	-22
	R Cerebelum (IX)	1339	5.6333	8	-62	-38
	R Cerebelum (VIII)	1339	4.0829	24	-54	-48
	L Linual Gyrus	1339	3.0039	-20	-52	-2
	L Precentral Gyrus	3544	7.5859	-42	4	48
	L IFG (p. Triangularis)	3544	6.9972	-52	28	22
	L Insula Lobe	3544	6.8278	-30	22	0
	L IFG (p. Orbitalis)	3544	6.7038	-50	24	-6
	L Middle Orbital Gyrus	3544	5.7953	-36	46	-2

	L Middle Frontal Gyrus	3544	4.4316	-30	6	64
	L IFG (p. Triangularis)	3544	4.0515	-34	20	34
	L IFG (p. Opercularis)	3544	4.0461	-54	8	12
	L Precentral Gyrus	3544	3.252	-60	4	34
	R Middle Frontal Gyrus	739	6.9609	42	36	24
	R Middle Frontal Gyrus	739	6.1368	46	20	44
	R Middle Frontal Gyrus	739	4.407	38	8	58
	R Middle Orbital Gyrus	697	6.146	40	46	-6
	R Superior Orbital Gyrus	697	5.2205	18	54	-8
	Location not in atlas	697	5.1725	36	58	20
	R Inferior Parietal Lobule	311	5.86	40	-54	52
	R Angular Gyrus	311	4.7997	60	-56	42
	R IFG (p. Orbitalis)	325	5.6683	34	22	-8
	R IFG (p. Orbitalis)	325	3.1885	52	18	2
	R Precuneus	99	5.2129	8	-68	44
	R Cerebelum (VI)	284	5.202	24	-58	-18
	Location not in atlas	284	4.6433	-2	-50	-20
	R Cerebelum (IV-V)	284	3.9084	24	-36	-24
	R Caudate Nucleus	100	4.2215	10	8	4
	L Middle Temporal Gyrus	102	4.147	-62	-38	-2
	R Cerebelum (Crus 1)	116	3.8855	30	-64	-28
Observe > External Affect	L Mid Orbital Gyrus	1405	8.29	0	52	-2
	Location not in atlas	1405	7.0915	0	4	-8
	L Rectal Gyrus	1405	6.0643	-8	36	-14
	L Superior Medial Gyrus	1405	4.5052	-10	66	-10
	R Rectal Gyrus	1405	3.4458	10	24	-16
	R Postcentral Gyrus	4275	7.9023	28	-34	58
	L Paracentral Lobule	4275	6.2731	0	-42	60
	Location not in atlas	4275	6.1784	-28	-70	16
	L Precuneus	4275	6.1258	-6	-58	22
	R Superior Parietal Lobule	4275	5.9695	18	-54	60
	R posterior-medial frontal	4275	5.5977	2	-18	62
	L Postcentral Gyrus	4275	5.3175	-28	-44	56
	L MCC	4275	4.9883	-2	-10	42
	Location not in atlas	4275	4.9062	44	-12	30
	Location not in atlas	4275	4.491	-14	-28	44
	Location not in atlas	4275	4.1311	20	-44	38
	R Precentral Gyrus	4275	4.0465	44	-18	58

	R Superior Frontal Gyrus	4275	3.8499	18	-12	74
	R Insula Lobe	729	7.4363	42	-8	-4
	R Temporal Pole	729	5.3251	62	8	0
	R Superior Temporal Gyrus	729	3.3817	66	-10	12
	L Superior Temporal Gyrus	104	6.6932	-40	-8	-8
	R SupraMarginal Gyrus	1076	6.668	64	-24	40
	R Superior Temporal Gyrus	1076	6.0979	52	-30	14
	Location not in atlas	1076	3.4331	32	-28	14
	L Superior Temporal Gyrus	222	5.6491	-52	-2	-6
	Location not in atlas	222	4.6529	-64	0	-22
	L Middle Temporal Gyrus	222	4.323	-68	-18	-8
	L Superior Temporal Gyrus	376	5.2867	-56	-32	14
	L Insula Lobe	376	4.3199	-34	-26	18
	L ParaHippocampal Gyrus	110	5.2188	-30	-30	-16
	L ParaHippocampal Gyrus	110	4.476	-18	-8	-20
	L Linual Gyrus	147	4.8366	-8	-88	4
	L Cuneus	147	3.8256	-4	-78	26
	L Middle Orbital Gyrus	3544	2.9135	-42	44	4
	L IFG (p. Triangularis)	3544	3.2672	-50	26	30
	L posterior-medial frontal	1287	3.0329	0	14	52
	L Inferior Parietal Lobule	1200	3.9564	-34	-56	46
External Object > Observe	L Inferior Parietal Lobule	424	6.9593	-46	-34	44
	R Linual Gyrus	902	6.2747	6	-68	4
	L Linual Gyrus	902	4.7994	-14	-66	4
	Location not in atlas	902	4.7233	20	-48	-24
	L Inferior Temporal Gyrus	141	5.4478	-50	-54	-18
	L Inferior Parietal Lobule	252	4.94	-30	-58	48
	L Postcentral Gyrus	95	4.6128	-52	-24	28
	L IFG (p. Opercularis)	98	4.3955	-50	4	34
Observe > External Objet	R Superior Medial Gyrus	211	6.492	8	56	18
	R Paracentral Lobule	228	5.414	6	-36	62
	R Inferior Parietal Lobule	228	5.2074	30	-48	56
	R Middle Temporal Gyrus	251	5.3229	48	-56	16
	Location not in atlas	122	5.1047	0	0	-14
	R Postcentral Gyrus	116	5.067	28	-34	58
	R Precentral Gyrus	116	4.3949	16	-36	76

	L Middle Temporal Gyrus	97	5.0639	-66	-14	-10
	L Mid Orbital Gyrus	125	4.7788	-6	54	-8
	R Inferior Temporal Gyrus	273	4.7448	54	-72	-4
	R Cerebelum (Crus 1)	273	4.0513	36	-82	-16
	R PCC	284	4.657	2	-52	32
	R Middle Temporal Gyrus	107	4.4651	54	-48	2
	L posterior-medial frontal	103	4.4042	-6	6	68
	R posterior-medial frontal	103	3.2744	6	20	58
Internal Affect > External Affect						
	No results to report.					
External Affect > Internal Affect	L Linual Gyrus	1251	6.6074	-8	-76	-4
	R Fusiform Gyrus	1251	5.6853	24	-76	-6
	L Calcarine Gyrus	1251	3.928	-4	-94	6
	L Mid Orbital Gyrus	438	6.482	-4	36	-8
	L Mid Orbital Gyrus	438	5.6712	-2	58	4
	L Fusiform Gyrus	333	6.0982	-28	-48	-6
	L Fusiform Gyrus	333	5.3088	-36	-34	-18
	L Middle Occipital Gyrus	173	5.8461	-32	-86	30
	L Calcarine Gyrus	543	5.8131	-16	-58	10
	Cerebellar Vermis (4/5)	543	5.2796	6	-52	8
	L PCC	543	5.2273	-8	-52	28
	R Fusiform Gyrus	143	5.5377	34	-44	-10
	R ACC	134	5.1145	8	34	14
	R Superior Medial Gyrus	134	3.4279	2	54	16
	L Precuneus	286	5.0018	-6	-54	56
	L Precuneus	286	3.7465	-8	-62	32
	L SupraMarginal Gyrus	95	4.535	-66	-36	28
	L Inferior Parietal Lobule	95	2.9698	-54	-36	50
	L Superior Frontal Gyrus	133	4.5332	-20	50	22
	L Superior Frontal Gyrus	133	4.1295	-20	40	46
Internal Affect > External Object	L IFG (p. Orbitalis)	5971	2.9826	-34	20	-10
	L Superior Medial Gyrus	5971	3.1132	-8	24	62
	L MCC	5971	9.1043	-2	24	40
	L IFG (p. Orbitalis)	5971	8.5328	-40	20	-2
	L posterior-medial frontal	5971	8.1981	-10	12	66

L IFG (p. Triangularis)	5971	6.942	-58	18	26
L Superior Frontal Gyrus	5971	6.1367	-14	58	28
R posterior-medial frontal	5971	6.0594	12	18	60
L IFG (p. Orbitalis)	5971	6.0551	-42	46	-8
L Middle Frontal Gyrus	5971	5.6819	-40	24	36
L Middle Frontal Gyrus	5971	5.6697	-40	12	56
R ACC	5971	5.2852	10	36	26
L Middle Frontal Gyrus	5971	5.2837	-22	26	58
L Middle Frontal Gyrus	5971	5.2691	-34	54	22
L Superior Frontal Gyrus	5971	4.4955	-16	44	44
L IFG (p. Orbitalis)	5971	4.3047	-28	10	-16
R Superior Frontal Gyrus	5971	4.2423	16	40	52
R Superior Medial Gyrus	5971	4.1554	6	54	34
R Middle Frontal Gyrus	5971	3.7477	30	24	48
R Middle Temporal Gyrus	6784	8.4923	48	-58	14
R Cerebelum (Crus 1)	6784	6.9745	38	-58	-26
L Cerebelum (Crus 2)	6784	6.9319	-18	-86	-30
L Middle Occipital Gyrus	6784	6.6984	-10	-100	6
R Inferior Occipital Gyrus	6784	6.6794	50	-78	-6
L Linual Gyrus	6784	6.1828	-8	-74	-2
R Fusiform Gyrus	6784	6.1411	46	-36	-20
Cerebellar Vermis (7)	6784	6.0035	2	-76	-20
R Cerebelum (VIII)	6784	5.9608	42	-52	-46
R Cerebelum (Crus 1)	6784	5.7013	24	-74	-32
L Cerebelum (VII)	6784	5.6124	-42	-58	-42
R Fusiform Gyrus	6784	5.4622	26	-74	-4
L Angular Gyrus	6784	5.3569	-40	-60	48
Location not in atlas	6784	5.0451	-36	-56	-18
L Cerebelum (VI)	6784	4.746	-36	-68	-14
L Middle Occipital Gyrus	6784	4.407	-42	-82	10
L Middle Temporal Gyrus	6784	4.3787	-54	-60	26
R Linual Gyrus	6784	4.1906	24	-94	-6
L Middle Occipital Gyrus	6784	4.1224	-26	-94	-22
L Linual Gyrus	6784	4.0247	-34	-92	-10
Location not in atlas	6784	3.9159	-30	-88	-

						46
	Location not in atlas	6784	3.5809	6	-84	-40
	Location not in atlas	6784	3.326	-14	-64	-30
	L Cuneus	6784	3.0966	-2	-96	26
	L Fusiform Gyrus	211	7.0228	-26	-48	-8
	R Precuneus	995	6.7823	4	-62	40
	Location not in atlas	995	4.4674	0	-50	20
	L Cuneus	995	3.605	-16	-72	30
	L Precuneus	995	3.5523	-6	-66	60
	R Fusiform Gyrus	139	6.1808	30	-44	-6
	R Insula Lobe	697	5.5892	34	16	-4
	R IFG (p. Triangularis)	697	5.141	56	20	8
	R IFG (p. Triangularis)	697	3.6812	58	22	28
	R Caudate Nucleus	139	5.3302	14	6	14
	Location not in atlas	171	5.1815	-4	-30	-18
	Location not in atlas	171	4.582	14	-20	-10
	R Cuneus	191	4.9032	14	-100	12
	R Middle Occipital Gyrus	191	3.8433	30	-92	26
	R Middle Frontal Gyrus	113	4.5546	24	44	32
	R Superior Medial Gyrus	122	4.3673	6	50	10
External Object > Internal Affect	L Superior Temporal Gyrus	231	5.528	-56	-2	-4
	L IFG (p. Opercularis)	231	4.239	-64	0	20
	Location not in atlas	593	5.2339	62	8	6
	R Insula Lobe	593	4.7681	42	-20	16
	R SupraMarginal Gyrus	593	4.4307	60	-22	30
	R Insula Lobe	593	3.9988	38	6	18
	R Precentral Gyrus	593	3.7964	50	-8	42
	R IFG (p. Opercularis)	593	3.2671	58	4	28
	L Heschls Gyrus	130	4.8895	-48	-16	16
External Affect > External Object	L Superior Medial Gyrus	1453	8.6134	-4	22	46
	R posterior-medial frontal	1453	4.4572	6	20	64
	L posterior-medial frontal	1453	4.4388	-14	12	70
	R ACC	1453	4.3434	10	36	24
	L Superior Medial Gyrus	1453	3.7339	-8	42	46
	R Superior Medial Gyrus	1453	3.6478	4	54	34
	L Insula Lobe	1235	7.7261	-28	22	0

	L IFG (p. Triangularis)	1235	5.3838	-54	24	28
	Location not in atlas	1235	5.1914	-58	20	4
	Location not in atlas	1235	4.7757	-30	10	30
	R Cerebelum (VI)	1784	6.9639	30	-68	-24
	R Cerebelum (VIII)	1784	6.122	40	-64	-44
	R Inferior Occipital Gyrus	1784	5.7743	34	-96	0
	R Cerebelum (Crus 2)	1784	5.1979	20	-84	-34
	R Inferior Temporal Gyrus	1784	4.3158	50	-76	-4
	R Cerebelum (Crus 1)	1784	3.4221	48	-58	-22
	L Cerebelum (VIII)	1574	6.7241	-32	-72	-48
	Location not in atlas	1574	6.0992	-42	-86	-10
	L Cerebelum (Crus 2)	1574	6.0007	-18	-78	-30
	L Cerebelum (Crus 1)	1574	5.9674	-38	-64	-24
	R IFG (p. Orbitalis)	168	6.0242	32	22	-6
	L Middle Frontal Gyrus	512	5.9628	-38	12	46
	L Middle Frontal Gyrus	512	5.0854	-32	4	64
	L Inferior Parietal Lobule	272	5.3024	-38	-58	44
	L Middle Temporal Gyrus	272	3.8414	-42	-58	24
	L Middle Orbital Gyrus	517	5.2289	-42	46	2
	L Middle Frontal Gyrus	517	5.0538	-34	58	16
	Location not in atlas	425	4.7903	30	18	8
	R Precentral Gyrus	425	4.7191	48	6	38
	R IFG (p. Triangularis)	425	4.5303	54	22	12
	L Precuneus	118	4.5497	-2	-58	40
	R Middle Frontal Gyrus	100	4.3787	40	6	58
	R Superior Frontal Gyrus	140	4.3478	36	52	16
External Object > External Affect	L Insula Lobe	302	6.8104	-34	2	-12
	Location not in atlas	302	5.4305	-42	-6	-12
	R Superior Temporal Gyrus	263	6.6399	52	-34	20
	R Postcentral Gyrus	263	4.4568	64	-16	22
	R Linual Gyrus	280	6.2568	12	-76	0
	R Insula Lobe	112	5.5328	44	2	-2
	L Linual Gyrus	119	5.4634	-8	-80	6
	L Superior Temporal Gyrus	110	5.1762	-62	-34	20

	L Precuneus	95	5.035	-4	-58	22
	L Mid Orbital Gyrus	222	4.7111	-4	26	-6
	R Mid Orbital Gyrus	222	3.8974	10	42	-6
	L Precuneus	226	4.6758	-4	-52	54
	L MCC	226	4.0985	-2	-26	48
	R MCC	226	3.5464	12	-44	44
	R Postcentral Gyrus	95	4.5654	50	-28	48
	L IFG (p. Triangularis)	1235	2.9897	-50	18	24
	L posterior-medial frontal	1453	3.0274	-4	20	62
Affect > Object	L MCC	6380	9.2603	-2	22	42
	L IFG (p. Orbitalis)	6380	7.8441	-40	20	-2
	L posterior-medial frontal	6380	7.6439	-8	16	64
	R ACC	6380	6.9894	8	32	26
	L Superior Frontal Gyrus	6380	6.7524	-14	58	28
	L Precentral Gyrus	6380	6.4639	-34	2	52
	L Middle Frontal Gyrus	6380	6.1611	-38	52	18
	L Middle Frontal Gyrus	6380	5.9813	-40	24	36
	L IFG (p. Triangularis)	6380	5.8397	-60	16	22
	L IFG (p. Orbitalis)	6380	5.7954	-42	46	-8
	L Superior Medial Gyrus	6380	4.9861	-2	42	44
	R Superior Medial Gyrus	6380	4.5148	6	52	10
	R Superior Medial Gyrus	6380	4.4889	12	30	62
	L Middle Frontal Gyrus	6380	4.2259	-28	22	56
	L IFG (p. Orbitalis)	6380	3.9341	-28	12	-18
	R Superior Frontal Gyrus	6380	3.9265	14	10	56
	R Superior Medial Gyrus	6380	3.5986	12	62	28
	L Cerebelum (Crus 2)	6652	8.1264	-16	-86	-28
	R Cerebelum (VIII)	6652	6.9961	40	-66	-44
	L Cerebelum (Crus 2)	6652	6.702	-44	-70	-34
	R Cerebelum (Crus 1)	6652	6.6496	26	-74	-30
	R Inferior Temporal Gyrus	6652	6.4628	44	-72	2
	L Fusiform Gyrus	6652	6.1333	-38	-88	-8
	R Middle Temporal Gyrus	6652	6.0015	50	-56	18
	L Cerebelum (VIII)	6652	5.8343	-42	-48	-44
	R Fusiform Gyrus	6652	5.4349	46	-36	-20
	R Cerebelum (Crus 2)	6652	5.1768	8	-84	-30

	R Fusiform Gyrus	6652	5.1089	42	-56	-18
	L Fusiform Gyrus	6652	4.6755	-42	-68	-14
	Location not in atlas	6652	4.6334	-22	-62	-26
	R Cerebelum (VII)	6652	4.5472	22	-82	-48
	R Linual Gyrus	6652	4.4194	24	-92	-6
	R Middle Temporal Gyrus	6652	3.7464	62	-46	2
	R Precuneus	655	6.1733	4	-62	40
	R Precuneus	655	5.5593	2	-62	60
	L Superior Occipital Gyrus	655	3.2862	-12	-76	44
	Location not in atlas	191	6.0389	6	-24	-20
	R Caudate Nucleus	248	6.0272	10	8	8
	R Thalamus	248	3.537	8	-16	12
	R Cerebelum (IX)	113	5.9013	6	-58	-38
	R IFG (p. Orbitalis)	959	5.556	32	22	-4
	R IFG (p. Triangularis)	959	5.3283	60	26	10
	R IFG (p. Triangularis)	959	4.5211	40	26	14
	R IFG (p. Triangularis)	959	4.4464	54	18	28
	R IFG (p. Orbitalis)	959	3.0543	52	32	-12
	L Caudate Nucleus	103	5.453	-10	8	4
	L Angular Gyrus	654	5.4072	-40	-60	46
	L Middle Temporal Gyrus	654	4.9193	-48	-56	24
	Cerebellar Vermis (1/2)	98	5.1682	-2	-50	-18
	L Thalamus	94	5.103	-10	-14	18
	R Middle Frontal Gyrus	239	4.9696	38	6	60
	R Middle Frontal Gyrus	239	4.0079	28	20	42
Object > Affect	R Postcentral Gyrus	465	8.0241	48	-10	38
	R Insula Lobe	465	5.2855	34	-24	20
	R Superior Temporal Gyrus	465	5.2686	52	-34	20
	R Postcentral Gyrus	465	3.8889	64	-16	24
	R Linual Gyrus	226	6.1435	10	-74	0
	R Temporal Pole	431	5.8369	62	6	4
	R Heschls Gyrus	431	5.2638	54	-12	10
	R Insula Lobe	431	4.6741	36	6	12
	L Insula Lobe	381	5.0067	-34	4	12
	L Insula Lobe	381	4.9554	-34	-18	20
	L Superior Temporal Gyrus	381	4.4756	-48	-10	6

L IFG (p. Opercularis)	381	3.3706	-64	0	20
L Superior Temporal Gyrus	88	4.672	-54	-2	-4
L Superior Temporal Gyrus	100	4.3808	-62	-32	20

Table A3 – Whole-brain Results from Number of Responses Task

This table shows all local maxima separated by more than 20 mm. Regions were labeled using the SPM Anatomy Toolbox atlas. x y and z =Montreal Neurological Institute (MNI) coordinates in the left-right anterior-posterior and inferior-superior dimensions respectively. Clusters were corrected for multiple comparisons by cluster-level FWE correction at $p < .05$.

				MNI Coordinates		
Contrast Name	Region Label	Extent	t-value	x	y	z
One Label > Observe	R ACC	5213	9.5263	8	32	26
	Location not in atlas	5213	8.2036	48	50	-6
	R MCC	5213	7.3145	4	22	44
	Location not in atlas	5213	6.8664	20	44	-18
	R Middle Frontal Gyrus	5213	5.8045	38	28	44
	L posterior-medial frontal	5213	5.7519	-2	6	56
	R Superior Frontal Gyrus	5213	5.5032	24	60	26
	R Middle Frontal Gyrus	5213	5.2053	42	38	24
	R Superior Frontal Gyrus	5213	5.0656	24	46	42
	R Superior Medial Gyrus	5213	4.2672	10	38	56
	Location not in atlas	5213	4.2195	28	62	-8
	R IFG (p. Triangularis)	5213	4.169	56	20	34
	R Middle Frontal Gyrus	5213	3.6453	26	10	62
	Location not in atlas	5213	3.5088	-10	36	18
	R Middle Frontal Gyrus	5213	3.4013	26	48	8
	L Superior Frontal Gyrus	5213	3.0098	-12	28	56
	L Cerebelum (VII)	998	7.272	-40	-58	-40
	L Cerebelum (Crus 1)	998	5.805	-46	-74	-28
	L Cerebelum (VII)	998	4.8701	-30	-80	-48
	L Calcarine Gyrus	195	7.1952	-2	-92	14
	L Precentral Gyrus	2743	7.1571	-44	4	52
	L IFG (p. Orbitalis)	2743	6.5649	-38	18	-8
	L IFG (p. Triangularis)	2743	5.5854	-50	22	26
	L Precentral Gyrus	2743	5.358	-34	-12	64
	L IFG (p. Triangularis)	2743	4.9301	-32	28	14
	L Middle Frontal Gyrus	2743	4.3042	-26	16	58
	L IFG (p. Triangularis)	2743	4.2689	-34	8	30
	L Middle Frontal Gyrus	2743	3.6469	-34	26	42
	Location not in atlas	2743	3.0958	-58	26	2
	L IFG (p. Opercularis)	2743	3.0434	-50	8	8
	Location not in atlas	1120	6.9934	-44	46	-10
	Location not in atlas	1120	5.352	-26	40	-18

	L Superior Frontal Gyrus	1120	5.0783	-24	52	10
	L Middle Temporal Gyrus	1036	6.7765	-54	-48	6
	Location not in atlas	1036	5.7643	-48	-28	-2
	L Inferior Temporal Gyrus	1036	5.2082	-60	-42	-16
	R IFG (p. Orbitalis)	486	6.7679	34	18	-4
	R IFG (p. Triangularis)	486	3.668	58	22	4
	R Cerebelum (Crus 1)	1784	6.6124	34	-58	-32
	R Cerebelum (VII)	1784	5.3756	32	-78	-42
	Cerebellar Vermis (7)	1784	5.1627	6	-80	-20
	L Cerebelum (Crus 1)	1784	4.5335	-16	-80	-24
	R Cerebelum (VI)	1784	3.9756	20	-58	-16
	R Cerebelum (Crus 1)	1784	3.3568	28	-84	-22
	L Inferior Parietal Lobule	1497	6.2948	-44	-32	50
	L Inferior Parietal Lobule	1497	5.7857	-38	-54	46
	L Inferior Parietal Lobule	1497	4.4577	-60	-48	46
	L Inferior Parietal Lobule	1497	3.289	-28	-74	52
	R Angular Gyrus	1208	6.0905	50	-62	46
	R SupraMarginal Gyrus	1208	5.3994	52	-36	46
	Location not in atlas	1208	3.4033	46	-44	26
	R Inferior Temporal Gyrus	538	5.8015	66	-24	-20
	R Inferior Temporal Gyrus	538	5.0611	56	-42	-18
	R Middle Temporal Gyrus	538	3.5164	70	-46	-2
	Location not in atlas	119	5.3972	4	-16	32
	Location not in atlas	119	4.6736	-2	-36	22
	R Precuneus	121	4.8778	4	-72	42
Observe > One Label	R Insula Lobe	223	8.8004	38	-18	20
	R Rolandic Operculum	223	3.9271	46	2	14
	Location not in atlas	223	3.8432	24	-16	4
	R Superior Parietal Lobule	1610	8.7849	24	-58	62
	R Superior Occipital Gyrus	1610	5.9919	28	-72	32
	R Precentral Gyrus	1610	5.9067	22	-36	68
	R Precuneus	1610	4.9695	8	-46	52
	Location not in atlas	1610	4.0257	30	-38	42
	R Superior Occipital Gyrus	1610	3.5182	26	-76	52
	R Inferior Temporal Gyrus	1005	8.3946	44	-64	-2
	R Fusiform Gyrus	1005	6.412	32	-34	-18
	R Fusiform Gyrus	1005	5.2595	26	-60	-10
	Location not in atlas	124	7.0389	-12	-42	-48
	L Cerebelum (VIII)	124	4.0183	-16	-62	-46
	L Superior Occipital Gyrus	783	7.0143	-24	-74	30
	L Superior Occipital Gyrus	783	5.5795	-18	-82	48
	L Middle Occipital Gyrus	783	4.6767	-44	-78	30
	L Middle Occipital Gyrus	783	3.5165	-44	-86	10

	L Middle Occipital Gyrus	783	3.5149	-24	-96	20
	L Precuneus	100	6.9934	-14	-50	10
	Location not in atlas	300	6.971	48	-24	30
	R Postcentral Gyrus	300	3.5897	56	-28	56
	L Middle Temporal Gyrus	695	6.2491	-44	-56	2
	L Fusiform Gyrus	695	6.1544	-24	-46	-8
	L Fusiform Gyrus	695	4.2923	-28	-30	-20
	L Middle Occipital Gyrus	695	4.2067	-50	-78	8
	Location not in atlas	642	6.0433	34	-20	56
	R Precentral Gyrus	642	5.6001	50	-10	44
	L MCC	212	5.9756	-12	-36	50
	L MCC	212	5.6185	-10	-12	42
	R Middle Occipital Gyrus	513	5.8822	36	-88	20
	Location not in atlas	513	3.127	40	-68	26
	R Superior Temporal Gyrus	119	5.3203	54	-30	14
	R Superior Temporal Gyrus	119	3.1364	70	-20	4
	Location not in atlas	110	5.074	-20	-52	44
	L MCC	110	4.3687	-4	-40	58
	R Temporal Pole	302	5.05	56	2	-8
	R Rolandic Operculum	302	4.7286	56	-12	18
	L Paracentral Lobule	271	4.6401	-14	-28	70
	L Superior Parietal Lobule	271	4.1931	-16	-60	64
	L Postcentral Gyrus	271	4.0364	-26	-42	62
	R Precuneus	185	4.3843	8	-58	24
	R Linual Gyrus	185	3.3475	8	-46	6
	L Middle Orbital Gyrus	1120	2.8892	-42	46	-4
	R Angular Gyrus	1208	2.991	44	-64	46
	L Inferior Parietal Lobule	1497	2.9926	-48	-38	42
	L Middle Temporal Gyrus	1036	3.0531	-52	-38	0
	L Precentral Gyrus	2743	3.2363	-40	4	44
Two Labels > Observe	L Middle Temporal Gyrus	1008	8.7256	-50	-54	10
	L Middle Temporal Gyrus	1008	5.9073	-48	-28	-4
	L Inferior Temporal Gyrus	1008	3.6045	-58	-42	-18
	L IFG (p. Triangularis)	4367	8.5535	-54	16	22
	L IFG (p. Orbitalis)	4367	7.925	-28	24	-6
	L Precentral Gyrus	4367	7.812	-44	6	50
	Location not in atlas	4367	7.7479	-48	40	-18
	L Inferior Parietal Lobule	4367	5.6504	-48	-34	52
	L IFG (p. Opercularis)	4367	5.486	-38	2	30
	L Middle Frontal Gyrus	4367	5.362	-26	48	12
	L IFG (p. Orbitalis)	4367	5.0679	-50	22	-8
	L Precentral Gyrus	4367	5.0198	-34	-12	66
	Location not in atlas	4367	4.7074	-28	28	24

L Middle Orbital Gyrus	4367	4.403	-50	44	4
L Middle Frontal Gyrus	4367	3.9135	-26	20	56
L Insula Lobe	4367	3.5606	-36	14	10
L Middle Orbital Gyrus	4367	3.1797	-36	54	-8
L Inferior Parietal Lobule	504	7.6579	-28	-58	46
L Inferior Parietal Lobule	504	3.7769	-48	-52	48
Location not in atlas	3510	7.509	20	-54	-24
R Cerebelum (Crus 2)	3510	7.336	28	-84	-38
L Calcarine Gyrus	3510	6.7229	-2	-88	14
Location not in atlas	3510	6.3541	36	-58	-36
Cerebellar Vermis (7)	3510	6.2153	6	-78	-18
Cerebellar Vermis (4/5)	3510	5.6846	6	-62	4
L Precuneus	3510	5.2942	-20	-58	8
R Cuneus	3510	4.6898	12	-78	38
L Cerebelum (Crus 1)	3510	4.502	-20	-80	-24
R ParaHippocampal Gyrus	3510	4.2774	34	-42	-6
R Linual Gyrus	3510	3.9921	26	-64	-2
R Cerebelum (Crus 1)	3510	3.6668	54	-54	-28
L Cerebelum (VI)	3510	3.6627	-20	-62	-14
L Cerebelum (VII)	128	7.1678	-40	-58	-40
L posterior-medial frontal	2030	6.7975	-2	6	54
R ACC	2030	6.6653	12	28	30
L Superior Medial Gyrus	2030	5.4558	-4	42	44
L Superior Medial Gyrus	2030	4.3527	-4	28	60
L ACC	2030	3.9881	-10	40	12
R Superior Frontal Gyrus	2030	3.7085	16	30	54
L Fusiform Gyrus	267	6.7428	-36	-46	-16
L Fusiform Gyrus	267	2.9947	-38	-72	-10
R Middle Frontal Gyrus	377	6.2739	34	50	6
Location not in atlas	377	5.5591	48	46	-16
Location not in atlas	759	6.0659	32	34	24
R IFG (p. Triangularis)	759	5.5196	48	18	34
R Middle Frontal Gyrus	759	4.003	30	26	44
R IFG (p. Triangularis)	759	3.7353	58	34	12
R IFG (p. Orbitalis)	369	5.6922	32	26	0
R IFG (p. Orbitalis)	369	3.6205	52	28	-6
R Temporal Pole	369	3.2074	40	16	-20
R Cerebelum (IX)	102	5.3928	14	-60	-40
L Medial Temporal Pole	107	5.2264	-50	10	-24
R Precentral Gyrus	124	4.9593	38	-4	46
R Middle Frontal Gyrus	124	3.2689	36	14	56
R Caudate Nucleus	148	4.9534	8	4	2
R Middle Orbital Gyrus	111	4.787	20	44	-16
R Superior Frontal Gyrus	129	4.6538	20	60	26

	Location not in atlas	160	4.4612	-34	-58	-32
	L Cerebelum (Crus 2)	160	4.4471	-32	-78	-38
	R Inferior Parietal Lobule	235	4.3947	42	-56	48
	Location not in atlas	235	4.1859	42	-46	28
Observe > Two Labels	R Calcarine Gyrus	281	6.9136	14	-96	12
	Location not in atlas	557	6.5069	44	-26	30
	R Postcentral Gyrus	557	6.1237	62	-22	38
	L Superior Temporal Gyrus	117	6.1997	-56	-8	6
	R Superior Temporal Gyrus	141	5.4395	62	-8	2
	R Insula Lobe	136	5.2291	42	-14	18
	Location not in atlas	331	4.5675	28	-36	56
	R Superior Parietal Lobule	331	4.3928	22	-62	60
	R Precentral Gyrus	331	3.5891	22	-36	76
	R Postcentral Gyrus	331	3.3533	48	-36	62
	L Middle Temporal Gyrus	1008	3.1571	-62	-54	8
	L IFG (p. Triangularis)	4367	3.271	-48	24	34
	L IFG (p. Orbitalis)	4367	3.4852	-40	18	-6
Free Response > Observe	L Middle Frontal Gyrus	10420	8.4534	-32	30	46
	L MCC	10420	8.1857	-4	26	40
	L IFG (p. Orbitalis)	10420	8.1212	-44	14	4
	R Middle Frontal Gyrus	10420	7.6874	36	22	46
	L IFG (p. Orbitalis)	10420	7.5147	-28	28	0
	L posterior-medial frontal	10420	7.4904	0	6	54
	R Middle Frontal Gyrus	10420	6.2971	32	2	56
	L Middle Frontal Gyrus	10420	6.2378	-30	58	22
	L Superior Orbital Gyrus	10420	6.2043	-28	54	2
	R Middle Frontal Gyrus	10420	6.0282	30	36	28
	L IFG (p. Triangularis)	10420	5.9789	-42	10	36
	L posterior-medial frontal	10420	5.4968	-10	4	72
	L IFG (p. Orbitalis)	10420	5.4884	-52	34	-4
	L Middle Frontal Gyrus	10420	5.2045	-32	2	64
	L IFG (p. Triangularis)	10420	5.2036	-54	24	28
	R ACC	10420	4.8792	4	42	20
	R Middle Frontal Gyrus	10420	4.8277	26	58	26
	Location not in atlas	10420	4.796	-26	48	-18
	R IFG (p. Opercularis)	10420	4.6191	38	4	30
	L Temporal Pole	10420	4.5462	-36	16	-16
	R Superior Frontal Gyrus	10420	4.1255	20	20	64
	R MCC	10420	3.9619	12	12	36
	L Superior Frontal Gyrus	10420	3.824	-18	46	42
	R Middle Frontal Gyrus	10420	3.7654	30	52	6

	R Superior Frontal Gyrus	10420	3.4486	22	38	48
	R Cerebelum (Crus 1)	2514	7.2049	46	-58	-26
	R Cerebelum (Crus 1)	2514	6.6527	32	-76	-30
	R Cerebelum (VIII)	2514	5.7882	36	-48	-42
	R ParaHippocampal Gyrus	2514	5.4399	30	-42	-6
	R Cerebelum (VI)	2514	5.3347	26	-56	-20
	R Cerebelum (Crus 2)	2514	5.2635	10	-82	-26
	R Cerebelum (IV-V)	2514	3.0557	30	-32	-24
	L Inferior Parietal Lobule	2975	7.1735	-56	-48	42
	L Middle Temporal Gyrus	2975	7.0285	-50	-48	12
	L Inferior Parietal Lobule	2975	6.2833	-36	-58	46
	L Middle Temporal Gyrus	2975	6.0332	-60	-40	-4
	L Superior Parietal Lobule	2975	4.7339	-22	-76	52
	L Middle Temporal Gyrus	2975	4.1926	-50	-22	-6
	L Inferior Parietal Lobule	2975	3.2082	-48	-30	48
	L Calcarine Gyrus	374	7.171	0	-92	16
	R IFG (p. Orbitalis)	836	7.1072	40	22	-10
	R IFG (p. Triangularis)	836	6.6474	58	30	10
	R IFG (p. Triangularis)	836	4.294	34	22	16
	R Inferior Temporal Gyrus	1807	6.2308	64	-36	-20
	R Middle Temporal Gyrus	1807	6.0348	58	-48	4
	R SupraMarginal Gyrus	1807	5.6425	56	-44	44
	R Middle Temporal Gyrus	1807	5.2513	52	-68	10
	R Superior Temporal Gyrus	1807	4.2809	62	-40	24
	R Inferior Parietal Lobule	1807	4.0347	48	-62	50
	L Inferior Temporal Gyrus	94	6.0916	-52	0	-32
	L Cerebelum (VII)	776	6.0641	-46	-58	-44
	L Cerebelum (VI)	776	5.6196	-32	-54	-30
	L Cerebelum (VIII)	776	5.0707	-30	-72	-50
	L Cerebelum (Crus 1)	776	4.0967	-32	-76	-26
	L Cerebelum (Crus 1)	183	5.9185	-10	-84	-22
	Location not in atlas	169	5.0708	4	-14	-2
	Location not in atlas	169	4.9013	4	8	8
	R Middle Orbital Gyrus	124	5.0639	30	58	-6
	L Thalamus	232	4.9572	-8	-14	10
	L Putamen	232	3.7678	-16	8	2
	Location not in atlas	170	4.6664	-6	-26	28
	R Middle Temporal Gyrus	126	4.6383	52	-32	-6
	L Precuneus	222	4.4882	-6	-68	38
	L Precuneus	222	3.4137	-6	-64	60
Observe > Free Response	Location not in atlas	278	6.4314	36	-18	8
	R Rolandic Operculum	278	3.7056	54	-12	16

	Location not in atlas	119	6.2873	-24	-54	16
	Location not in atlas	389	6.0463	24	-40	60
	R Superior Parietal Lobule	389	3.9775	22	-62	56
	R Precentral Gyrus	557	5.4567	52	-14	52
	Location not in atlas	557	5.2231	34	-26	46
	R Postcentral Gyrus	557	3.8286	46	-34	60
	L Cerebelum (IV-V)	103	5.2731	-8	-62	-2
	R Superior Occipital Gyrus	274	5.0536	22	-102	12
	R SupraMarginal Gyrus	216	5.0004	64	-24	38
	R Rolandic Operculum	216	4.9807	44	-30	28
	R Middle Frontal Gyrus	10420	2.914	36	24	42
	L Middle Frontal Gyrus	10420	2.9872	-30	48	20
	L IFG (p. Triangularis)	10420	3.0434	-46	20	24
	R IFG (p. Orbitalis)	836	3.1013	38	24	-2
	R Cerebelum (Crus 1)	2514	3.1067	18	-76	-26
One Label > Two Labels	L Cerebelum (Crus 2)	160	6.3151	-38	-70	-42
	R Angular Gyrus	617	5.6372	46	-60	36
	R Inferior Parietal Lobule	617	4.2945	48	-56	56
	R Inferior Temporal Gyrus	181	5.3221	66	-30	-18
	L Inferior Parietal Lobule	152	4.3824	-54	-54	50
Two Labels > One Label	R Middle Temporal Gyrus	4046	3.1823	42	-72	14
	R Fusiform Gyrus	4046	8.1886	26	-42	-8
	R Middle Occipital Gyrus	4046	8.0052	36	-84	18
	R Linual Gyrus	4046	7.2631	14	-68	0
	R Middle Temporal Gyrus	4046	6.3233	52	-72	6
	R Linual Gyrus	4046	5.9302	14	-50	10
	R Superior Occipital Gyrus	4046	5.8684	26	-76	36
	R Middle Temporal Gyrus	4046	5.1648	40	-64	22
	R ParaHippocampal Gyrus	4046	4.5786	34	-26	-18
	R Fusiform Gyrus	4046	4.0286	46	-62	-12
	R Fusiform Gyrus	4046	3.9921	46	-42	-22
	L Fusiform Gyrus	3790	8.044	-20	-46	-10
	L Middle Occipital Gyrus	3790	7.904	-36	-84	24
	L Inferior Occipital Gyrus	3790	6.7642	-44	-72	-6
	L Superior Occipital Gyrus	3790	6.5554	-22	-76	42
	L Calcarine Gyrus	3790	5.5939	-14	-58	12
	L Fusiform Gyrus	3790	5.5852	-42	-52	-14
	Location not in atlas	3790	4.7374	-38	-60	10
	L Inferior Parietal Lobule	3790	4.4354	-28	-58	54
	L Fusiform Gyrus	3790	3.8522	-32	-26	-26
	L Linual Gyrus	3790	3.813	-18	-68	-6

	Location not in atlas	322	5.4296	24	-54	58
	R Precentral Gyrus	172	5.0799	34	-10	54
	L MCC	140	4.3435	0	-48	54
	R Angular Gyrus	617	2.9197	44	-60	46
One Label > Free Response	Location not in atlas	151	6.9202	-6	-64	4
	L Cerebelum (VIII)	125	4.5544	-30	-66	-38
Free Response > One Label	R Fusiform Gyrus	405	2.9147	20	-42	-12
	L Fusiform Gyrus	383	3.0706	-28	-50	-10
	L Middle Occipital Gyrus	1318	3.1814	-40	-82	18
	R Inferior Temporal Gyrus	1677	3.4169	54	-66	0
	R Fusiform Gyrus	405	9.3076	30	-42	-8
	R Cerebelum (III)	405	3.1871	22	-28	-20
	L Superior Occipital Gyrus	1318	7.8737	-22	-76	42
	L Middle Occipital Gyrus	1318	7.0381	-36	-82	14
	L Middle Temporal Gyrus	1318	4.2618	-50	-66	2
	L Calcarine Gyrus	1318	3.9387	-24	-66	18
	L Superior Occipital Gyrus	1318	3.1143	-22	-92	28
	R Middle Temporal Gyrus	1677	7.8195	44	-74	20
	R Middle Occipital Gyrus	1677	6.41	28	-74	36
	R Inferior Temporal Gyrus	1677	5.1174	52	-70	-4
	R Fusiform Gyrus	1677	4.3657	32	-70	-6
	L Fusiform Gyrus	383	7.0643	-28	-46	-8
	R Superior Parietal Lobule	194	5.9286	20	-58	66
	L MCC	230	5.3364	-2	-50	56
	Location not in atlas	230	3.5085	16	-40	42
Two Labels > Free Response	L Linual Gyrus	1413	6.5562	-18	-58	0
	R Fusiform Gyrus	1413	5.825	22	-60	-8
	Cerebellar Vermis (4/5)	1413	4.778	2	-64	6
	R Linual Gyrus	1413	4.1775	12	-82	4
Free Response > Two Labels	L Superior Occipital Gyrus	207	2.9042	-8	-100	12
	L Posterior-medial Frontal Gyurs	112	5.8137	-12	6	68
	L Calcarine Gyrus	207	5.4516	-6	-100	2
	L Middle Occipital Gyrus	207	3.1893	-18	-98	22
	Location not in atlas	232	5.3964	-60	8	4
	L Rolandic Operculum	232	4.0152	-44	0	18

	L Insula Lobe	232	3.7202	-40	4	-4
	R MCC	261	5.2505	4	-24	42
	R MCC	261	5.0574	10	-48	38
	R Superior Temporal Gyrus	522	5.2425	56	-40	28
	R SupraMarginal Gyrus	522	4.4804	54	-40	48
	R Postcentral Gyrus	522	3.6146	62	-22	38
	L Inferior Parietal Lobule	253	4.8495	-56	-40	48
	L SupraMarginal Gyrus	253	3.5382	-60	-56	30
	L Middle Frontal Gyrus	112	4.7284	-28	44	26
	Location not in atlas	100	4.6359	62	10	6
Labels > Free Response	Location not in atlas	511	7.3815	-6	-64	4
	R Linual Gyrus	511	4.3747	14	-64	6
Free Response > Labels	L Middle Occipital Gyrus	377	5.356	-30	-92	20
	L Middle Occipital Gyrus	377	2.9512	-38	-78	8
	R Middle Temporal Gyrus	406	5.2544	44	-76	16
	R Inferior Temporal Gyrus	406	4.0442	54	-64	-6
	L Calcarine Gyrus	112	4.7548	-6	-102	4
	L Middle Occipital Gyrus	112	3.1116	-18	-100	20

Appendix B – Tables of ROI Results

Appendix B1 – ROI Results from Label Intensity Task

Contrast	Region	L/R	Mean	SD	CI (upper)	CI (lower)	t	p
Abstract > Gender	LPFC (pars opercularis)	R	0.092	0.204	0.186	-0.002	1.910	0.037
Gender > Abstract	LPFC (pars orbitalis)	L	0.147	0.351	0.304	-0.011	1.822	0.043
	Amygdala (laterobasal)	L	0.081	0.204	0.175	-0.013	1.687	0.055
	LPFC (pars orbitalis)	R	0.112	0.299	0.246	-0.022	1.633	0.060
	VLPFC (AL-OBS peak)	R	0.148	0.414	0.334	-0.039	1.555	0.069
Low > Gender	LPFC (pars triangularis)	L	0.127	0.222	0.227	0.028	2.506	0.011
	LPFC (pars opercularis)	L	0.108	0.198	0.196	0.019	2.376	0.014
	VLPFC (AL-GL peak)	L	0.310	0.581	0.571	0.049	2.324	0.016
	VLPFC (AL-GL peak)	R	0.193	0.383	0.365	0.021	2.200	0.021
	Amygdala AAL	R	0.109	0.222	0.209	0.009	2.145	0.023
	LPFC (pars triangularis)	R	0.082	0.184	0.165	-0.001	1.940	0.034
	Amygdala (centromedial)	R	0.156	0.356	0.316	-0.004	1.916	0.036
	Amygdala (laterobasal)	R	0.070	0.193	0.156	-0.017	1.578	0.066
	LPFC (pars opercularis)	R	0.069	0.193	0.155	-0.018	1.551	0.069
Gender > Low	VLPFC (AL-OBS peak)	R	0.245	0.346	0.401	0.090	3.093	0.003
High > Gender	LPFC (pars opercularis)	L	0.167	0.260	0.284	0.050	2.799	0.006
	VLPFC (AL-GL peak)	L	0.322	0.624	0.603	0.042	2.252	0.019
	LPFC (pars triangularis)	L	0.131	0.267	0.251	0.011	2.142	0.023
	VLPFC (AL-GL peak)	R	0.141	0.302	0.277	0.006	2.043	0.028
	LPFC (pars opercularis)	R	0.094	0.264	0.213	-0.025	1.554	0.069
	Amygdala (laterobasal)	L	0.118	0.338	0.270	-0.034	1.527	0.072
Gender > High	Amygdala (superficial)	R	0.229	0.422	0.424	0.034	2.303	0.017
	VLPFC (AL-OBS peak)	R	0.240	0.515	0.471	0.009	2.032	0.029
	LPFC (pars orbitalis)	R	0.092	0.299	0.226	-0.043	1.337	0.099
Abstract > Specific	VLPFC (AL-OBS peak)	R	0.095	0.305	0.232	-0.042	1.358	0.096

Specific > Abstract	VLPFC (AL-GL peak)	L	0.267	0.414	0.453	0.081	2.818	0.006
	Amygdala (laterobasal)	L	0.189	0.318	0.332	0.046	2.590	0.009
	LPFC (pars triangularis)	L	0.125	0.213	0.221	0.029	2.559	0.010
	LPFC (pars orbitalis)	L	0.132	0.232	0.236	0.027	2.475	0.012
	VLPFC (AL-GL peak)	R	0.167	0.381	0.339	-0.004	1.913	0.036
	Amygdala AAL	L	0.186	0.425	0.377	-0.005	1.910	0.036
	LPFC (pars opercularis)	L	0.076	0.226	0.178	-0.025	1.469	0.080
	LPFC (pars triangularis)	R	0.072	0.214	0.168	-0.024	1.464	0.080
	Amygdala (centromedial)	R	0.103	0.323	0.248	-0.043	1.386	0.091
Low > High	Amygdala (superficial)	R	0.226	0.467	0.442	0.011	2.058	0.028
	Amygdala (centromedial)	R	0.174	0.500	0.399	-0.051	1.517	0.073
High > Low	Amygdala (laterobasal)	L	0.128	0.384	0.301	-0.045	1.452	0.082

Appendix B2 – ROI Results from Types of Affect Labeling Task

Contrast	Region	L/R	Mean	SD	CI (upper)	CI (lower)	t	p
Internal Affect > Observe	LPFC (pars triangularis)	L	0.312	0.249	0.422	0.203	5.598	0.000
	LPFC (pars opercularis)	L	0.272	0.273	0.391	0.152	4.458	0.000
	LPFC (pars orbitalis)	L	0.245	0.304	0.378	0.112	3.610	0.001
	VLPFC (AL-GL peak)	L	0.327	0.410	0.507	0.148	3.575	0.001
	VLPFC (AL-OBS peak)	L	0.408	0.531	0.641	0.175	3.438	0.001
	VLPFC (AL-OBS peak)	R	0.150	0.248	0.258	0.041	2.696	0.007
	LPFC (pars orbitalis)	R	0.144	0.323	0.285	0.002	1.991	0.031
	LPFC (pars triangularis)	R	0.101	0.234	0.206	-0.004	1.882	0.038
	LPFC (pars opercularis)	R	0.101	0.304	0.235	-0.032	1.492	0.076
Observe > Internal Affect	Amygdala (centromedial)	R	0.305	0.574	0.556	0.053	2.373	0.014
	Amygdala (superficial)	R	0.362	0.742	0.687	0.037	2.182	0.021
	Amygdala (superficial)	L	0.351	0.752	0.681	0.022	2.090	0.025
	Amygdala (centromedial)	L	0.202	0.588	0.460	-0.055	1.538	0.070
	Amygdala (laterobasal)	R	0.085	0.287	0.211	-0.040	1.330	0.100
External Affect > Observe	LPFC (pars triangularis)	L	0.362	0.280	0.485	0.240	5.785	0.000
	VLPFC (AL-OBS peak)	L	0.490	0.415	0.672	0.309	5.288	0.000
	LPFC (pars opercularis)	L	0.299	0.312	0.436	0.163	4.293	0.000
	VLPFC (AL-OBS peak)	R	0.218	0.250	0.327	0.108	3.890	0.000
	VLPFC (AL-GL peak)	L	0.390	0.476	0.598	0.181	3.666	0.001
	LPFC (pars orbitalis)	L	0.195	0.260	0.309	0.081	3.361	0.002
	LPFC (pars orbitalis)	R	0.111	0.257	0.223	-0.002	1.923	0.035
	LPFC (pars triangularis)	R	0.127	0.297	0.258	-0.003	1.913	0.035
	LPFC (pars opercularis)	R	0.085	0.260	0.199	-0.029	1.466	0.079
Observe > External Affect	Amygdala (superficial)	L	0.569	0.773	0.908	0.230	3.289	0.002
	Amygdala (centromedial)	L	0.332	0.553	0.574	0.089	2.683	0.007
	Amygdala (laterobasal)	R	0.154	0.286	0.279	0.028	2.406	0.013
	Amygdala AAL	L	0.207	0.473	0.414	-0.001	1.953	0.033
	Amygdala (superficial)	R	0.305	0.721	0.621	-0.011	1.889	0.037
	Amygdala (centromedial)	R	0.226	0.541	0.463	-0.011	1.866	0.039
	Amygdala (laterobasal)	L	0.127	0.367	0.288	-0.033	1.552	0.069
External Object > Observe	LPFC (pars opercularis)	L	0.083	0.244	0.190	-0.024	1.529	0.071
	LPFC (pars triangularis)	L	0.081	0.261	0.195	-0.033	1.390	0.090

Observe > External Object	Amygdala (superficial)	L	0.473	0.904	0.869	0.076	2.338	0.015
	VLPFC (AL-GL peak)	R	0.292	0.644	0.574	0.009	2.026	0.029
	LPFC (pars triangularis)	R	0.130	0.281	0.257	0.004	2.023	0.029
	Amygdala (centromedial)	R	0.266	0.618	0.537	-0.005	1.921	0.035
	Amygdala (centromedial)	L	0.255	0.675	0.551	-0.041	1.690	0.054
Internal Affect > External Affect	VLPFC (AL-GL peak)	R	0.146	0.469	0.352	-0.059	1.396	0.089
Internal Affect > External Object	VLPFC (AL-GL peak)	R	0.327	0.388	0.501	0.153	3.679	0.001
	LPFC (pars triangularis)	L	0.231	0.282	0.355	0.108	3.667	0.001
	LPFC (pars opercularis)	L	0.149	0.182	0.231	0.067	3.573	0.001
	VLPFC (AL-OBS peak)	L	0.328	0.417	0.511	0.145	3.514	0.001
	LPFC (pars triangularis)	R	0.224	0.297	0.355	0.094	3.382	0.002
	LPFC (pars opercularis)	R	0.103	0.186	0.186	0.019	2.408	0.013
	LPFC (pars orbitalis)	L	0.189	0.367	0.350	0.029	2.310	0.016
	LPFC (pars orbitalis)	R	0.158	0.347	0.311	0.006	2.042	0.028
	VLPFC (AL-OBS peak)	R	0.097	0.240	0.205	-0.010	1.769	0.047
External Affect > External Object	LPFC (pars triangularis)	L	0.281	0.289	0.408	0.155	4.348	0.000
	VLPFC (AL-OBS peak)	L	0.410	0.481	0.621	0.199	3.809	0.001
	LPFC (pars triangularis)	R	0.203	0.271	0.321	0.084	3.346	0.002
	LPFC (pars opercularis)	L	0.216	0.308	0.351	0.080	3.126	0.003
	VLPFC (AL-OBS peak)	R	0.159	0.242	0.268	0.050	2.867	0.005
	VLPFC (AL-GL peak)	R	0.247	0.430	0.435	0.059	2.571	0.009
	VLPFC (AL-GL peak)	L	0.206	0.387	0.380	0.032	2.321	0.016
	LPFC (pars orbitalis)	R	0.125	0.246	0.233	0.018	2.279	0.017
	LPFC (pars opercularis)	R	0.125	0.264	0.241	0.010	2.123	0.024
	LPFC (pars orbitalis)	L	0.139	0.299	0.270	0.008	2.086	0.025
Affect > Object	LPFC (pars triangularis)	L	0.256	0.254	0.368	0.145	4.507	0.000
	VLPFC (AL-OBS peak)	L	0.369	0.382	0.536	0.202	4.323	0.000
	VLPFC (AL-GL peak)	R	0.257	0.275	0.380	0.133	4.065	0.000
	LPFC (pars triangularis)	R	0.214	0.258	0.327	0.101	3.706	0.001
	LPFC (pars opercularis)	L	0.202	0.252	0.313	0.091	3.580	0.001
	VLPFC (AL-OBS peak)	R	0.128	0.193	0.215	0.042	2.906	0.005
	LPFC (pars opercularis)	R	0.133	0.229	0.234	0.033	2.597	0.009
	LPFC (pars orbitalis)	L	0.164	0.302	0.297	0.032	2.431	0.013
	LPFC (pars orbitalis)	R	0.142	0.267	0.259	0.025	2.378	0.014
	VLPFC (AL-GL peak)	L	0.164	0.339	0.316	0.011	2.107	0.025

Object > Affect	Amygdala (laterobasal)	L	0.083	0.187	0.167	-0.002	1.923	0.035
	Amygdala (superficial)	R	0.160	0.435	0.351	-0.031	1.644	0.058

Appendix B3 – ROI Results from Number of Responses Task

Contrast	Region	L/R	Mean	SD	CI (upper)	CI (lower)	t	p
One Label > Observe	LPFC (pars opercularis)	L	0.276	0.247	0.385	0.168	5.005	0.000
	LPFC (pars triangularis)	L	0.304	0.276	0.425	0.184	4.938	0.000
	VLPFC (AL-OBS peak)	L	0.524	0.510	0.747	0.300	4.588	0.000
	LPFC (pars orbitalis)	L	0.219	0.240	0.326	0.111	3.979	0.000
	VLPFC (AL-OBS peak)	R	0.286	0.422	0.471	0.101	3.030	0.003
	LPFC (pars orbitalis)	R	0.137	0.207	0.230	0.044	2.885	0.005
	LPFC (pars opercularis)	R	0.143	0.236	0.249	0.037	2.646	0.008
	LPFC (pars triangularis)	R	0.112	0.193	0.199	0.025	2.531	0.010
	VLPFC (AL-GL peak)	L	0.291	0.571	0.541	0.041	2.278	0.017
Observe > One Label	Amygdala (laterobasal)	L	0.243	0.347	0.399	0.088	3.062	0.003
	Amygdala AAL	R	0.242	0.393	0.415	0.070	2.760	0.006
	Amygdala (laterobasal)	R	0.145	0.230	0.248	0.042	2.749	0.007
	Amygdala (superficial)	R	0.435	0.722	0.751	0.118	2.694	0.007
	Amygdala (centromedial)	R	0.386	0.651	0.672	0.101	2.653	0.008
	Amygdala AAL	L	0.216	0.503	0.442	-0.010	1.871	0.039
	Amygdala (superficial)	L	0.431	1.071	0.901	-0.038	1.802	0.044
	Amygdala (centromedial)	L	0.306	0.842	0.675	-0.063	1.624	0.060
Two Labels > Observe	LPFC (pars triangularis)	L	0.371	0.267	0.488	0.254	6.226	0.000
	LPFC (pars orbitalis)	L	0.353	0.281	0.476	0.230	5.615	0.000
	LPFC (pars opercularis)	L	0.266	0.225	0.364	0.167	5.295	0.000
	VLPFC (AL-OBS peak)	L	0.529	0.518	0.756	0.303	4.575	0.000
	VLPFC (AL-GL peak)	L	0.527	0.536	0.761	0.292	4.394	0.000
	LPFC (pars orbitalis)	R	0.214	0.234	0.316	0.111	4.084	0.000
	VLPFC (AL-OBS peak)	R	0.233	0.286	0.358	0.108	3.647	0.001
	LPFC (pars triangularis)	R	0.182	0.262	0.297	0.068	3.114	0.003
	VLPFC (AL-GL peak)	R	0.233	0.370	0.395	0.071	2.815	0.006
	LPFC (pars opercularis)	R	0.137	0.301	0.268	0.005	2.034	0.028
Observe > Two Labels	Amygdala (laterobasal)	R	0.179	0.367	0.340	0.018	2.180	0.021
	Amygdala AAL	R	0.172	0.427	0.359	-0.015	1.798	0.044
	Amygdala (laterobasal)	L	0.151	0.385	0.319	-0.018	1.753	0.048
	Amygdala (superficial)	L	0.359	0.974	0.785	-0.068	1.647	0.058
	Amygdala (centromedial)	R	0.220	0.619	0.491	-0.052	1.587	0.064
	Amygdala AAL	L	0.186	0.583	0.442	-0.069	1.430	0.085

Free Response > Observe	LPFC (pars triangularis)	L	0.370	0.339	0.518	0.221	4.877	0.000
	VLPFC (AL-GL peak)	L	0.531	0.488	0.745	0.318	4.874	0.000
	LPFC (pars opercularis)	L	0.385	0.354	0.540	0.230	4.868	0.000
	VLPFC (AL-GL peak)	R	0.366	0.435	0.557	0.176	3.769	0.001
	LPFC (pars orbitalis)	L	0.319	0.386	0.488	0.150	3.694	0.001
	LPFC (pars opercularis)	R	0.234	0.286	0.360	0.109	3.659	0.001
	LPFC (pars triangularis)	R	0.223	0.293	0.352	0.095	3.409	0.001
	VLPFC (AL-OBS peak)	L	0.343	0.465	0.552	0.134	3.212	0.002
	LPFC (pars orbitalis)	R	0.180	0.304	0.313	0.046	2.640	0.008
	VLPFC (AL-OBS peak)	R	0.173	0.316	0.312	0.035	2.448	0.012
Observe > Free Response	Amygdala (superficial)	L	0.279	0.459	0.485	0.072	2.643	0.008
One Label > Free Response	VLPFC (AL-OBS peak)	L	0.241	0.471	0.453	0.029	2.232	0.019
Free Response > One Label	Amygdala (laterobasal)	R	0.189	0.303	0.322	0.057	2.795	0.006
	Amygdala AAL	R	0.179	0.309	0.318	0.040	2.529	0.010
	Amygdala (centromedial)	R	0.263	0.457	0.469	0.058	2.513	0.011
	Amygdala (laterobasal)	L	0.123	0.289	0.253	-0.007	1.856	0.040
	VLPFC (AL-GL peak)	R	0.198	0.500	0.423	-0.027	1.729	0.050
	VLPFC (AL-GL peak)	L	0.241	0.644	0.523	-0.042	1.670	0.056
	Amygdala (superficial)	R	0.385	1.042	0.841	-0.072	1.652	0.058
Two Labels > Free Response	VLPFC (AL-OBS peak)	L	0.190	0.478	0.405	-0.025	1.734	0.050
	LPFC (pars orbitalis)	R	0.087	0.230	0.190	-0.016	1.651	0.058
Free Response > Two Labels	Amygdala (laterobasal)	R	0.174	0.319	0.313	0.034	2.434	0.012
	LPFC (pars opercularis)	L	0.119	0.340	0.268	-0.030	1.563	0.067
	Amygdala AAL	R	0.114	0.319	0.257	-0.030	1.550	0.069
	LPFC (pars opercularis)	R	0.098	0.286	0.223	-0.028	1.524	0.072
Two Labels > One Label	VLPFC (AL-GL peak)	L	0.236	0.436	0.427	0.044	2.415	0.013
	Amygdala (superficial)	R	0.304	0.730	0.624	-0.017	1.859	0.039
	Amygdala (laterobasal)	L	0.158	0.398	0.332	-0.017	1.773	0.046
	Amygdala (centromedial)	R	0.166	0.451	0.364	-0.031	1.651	0.058
	VLPFC (AL-GL peak)	R	0.191	0.535	0.426	-0.043	1.601	0.063
	LPFC (pars orbitalis)	L	0.076	0.255	0.188	-0.035	1.340	0.098
	LPFC (pars triangularis)	L	0.067	0.224	0.165	-0.031	1.335	0.099

Provided Labels > Free Response	VLPFC (AL-OBS peak)	L	0.216	0.445	0.416	0.016	2.112	0.024
	LPFC (pars orbitalis)	R	0.075	0.202	0.166	-0.016	1.614	0.062
Free Response > Provided Labels	Amygdala (laterobasal)	R	0.182	0.277	0.303	0.060	2.930	0.004
	Amygdala AAL	R	0.146	0.256	0.261	0.032	2.497	0.011
	Amygdala (centromedial)	R	0.187	0.380	0.358	0.017	2.150	0.023
	VLPFC (AL-GL peak)	R	0.120	0.338	0.272	-0.032	1.550	0.069

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