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The neuroscience of emotion regulation: Basic mechanisms and their role in
development, aging and psychopathology

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I can't change the direction of the wind, but I can adjust my sails.

- James Dean

When life's gentle breezes turn into threatening gales, we humans have a remarkable ability to adapt accordingly. This adaptability grants us a degree of control not just over our circumstances, but also our emotional responses to them. We can keep our cool under stress, resist harmful temptations, and emerge resilient from all manner of trials and tribulations. We do so using a diversity of emotion regulation strategies (Gross & Thompson, 2007) that allow us to alter the nature, magnitude, and duration of our emotional responses in a variety of circumstances.

The ability to regulate one's emotions is one of the keys to leading a healthy and productive life. Indeed, emotion regulation failures are a hallmark of many types of psychopathology, as well as a normal part of development for children and adolescents. A major motivator of the emerging science of emotion regulation is the need to better understand why and how these failures occur, and to lay the foundation for efforts to improve emotion regulation skills.

With this in mind, the goals of this chapter are two-fold. In the first section, we outline a model of the cognitive control of emotion in healthy adults, updating a core model first proposed in 2002 (Ochsner, Bunge, Gross, & Gabrieli, 2002; Ochsner, Silvers, & Buhle, Submitted) and modified several times since (Ochsner & Gross, 2005). In the second section, we use this model as a vantage point from which to survey recent efforts to examine emotion regulation in the contexts of development, aging, and psychopathology.

Cognitive Control of Emotion

In this initial section of the chapter we outline a model that broadly describes the psychological and neural processes by which cognitive strategies can be used to control our emotions (Figure 1). First, we will outline a psychological process model of emotion generation and regulation. Next, we will consider the neural sources of regulation—the regions that generate and implement the regulatory processes that comprise a given strategy. Finally, we will consider the neural targets of regulation—i.e., the regions that are impacted by regulatory processes.

Process Model of Emotion Generation and Regulation

Emotion generation The time line at the bottom of Figure 1A depicts four basic steps involved in the generation of an emotional response (Barrett, Mesquita, Ochsner, & Gross, 2007). In the first step, a stimulus is perceived in its current situational context. The stimulus could be internal in origin, such as a thought, feeling, or sensation, or external, such as a facial expression, gesture, action or event. At the second stage, one attends to some of these stimuli or their attributes. The focus of attention

determines what information feeds forward to subsequent emotion generation stages. Ignored or unattended stimuli may be either excluded from these subsequent stages or receive reduced processing. The third stage involves appraising the significance of stimuli in terms of their relevance to one's current goals, wants or needs. According to appraisal theories of emotion, it is here where an emotion takes on both its valence (whether it is positive or negative) and its more specific characterization (anger, fear, sadness, etc.) (Scherer, Schorr, & Johnstone, 2001). Finally, in the fourth stage these appraisals coalesce into an emotional response, consisting of some combination of emotional experience, emotion-expressive behavior, and autonomic activity. Although these three indicators of emotional response do not always correlate with one another for reasons that are not perfectly understood (Mauss, Levenson, McCarter, Wilhelm, & Gross, 2005), as noted below, emotion regulation strategies can effect changes in some or all of them, depending on the strategy.

Emotion regulation Emotion regulation involves the modification of emotional responses through the engagement of top-down control processes. Building on previous work, our model of emotion regulation distinguishes among five classes of strategies whose effects on emotion can be understood in terms of the stage of the emotion generation sequence that they impact (Gross, 1998b). In the present chapter, we focus on conscious, goal-driven regulatory strategies rather than implicit or non-conscious ones because they have been studied far more frequently.

As illustrated by the top portion of Figure 1A, the first two strategies involve changing the nature of the stimulus inputs to the emotion generation cycle. In *situation selection*, you keep yourself away from stimuli that elicit unwanted emotions and put yourself in the presence of stimuli that elicit desired emotions. For example, a recovering alcoholic may choose to skip a happy hour with coworkers or a dieter may stay away from the sections of the grocery store that offer desserts. *Situation modification* is when you find yourself in the presence of a stimulus that elicits an unwanted emotion and change something about the situation to alter its impact on you (J. I. Davis, Gross, & Ochsner, 2009). For the recovering alcoholic, this may mean leaving a party when one's friends begin to become intoxicated.

Given that we cannot always avoid or alter our external circumstances, at times we must regulate our emotions by changing our internal responses. A third strategy, *Attentional deployment*, describes the allocation of attention among stimuli or components of a particular stimulus. This strategy can be further broken down into two subtypes (Ochsner & Gross, 2005). *Selective attention* involves moving the focus of attention towards or away from stimuli or their attributes (e.g., (Anderson, Christoff, Panitz, De Rosa, & Gabrieli, 2003 ; Etkin, Egner, Peraza, Kandel, & Hirsch, 2006)). For example, the recovering alcoholic may attempt to avert his gaze from an ad on the subway promoting the latest flavored vodka. *Distraction* involves limiting attentional resources by introducing a competing working memory demand (e.g., (Buhle, Stevens, Friedman, & Wager, 2012; Buhle & Wager, 2009; Kanske, Heissler, Schonfelder, Bongers, & Wessa, 2011; McRae et al., 2010)). For example, the alcoholic may throw himself into a difficult problem at work when the urge to drink arises.

Cognitive change involves altering the way one thinks about an emotional stimulus so as to alter one's emotional response to it. This change may occur when generating an initial emotional response, as when expectations, beliefs or mindsets influence one's appraisal of a stimulus, or subsequently, as when one changes the way one thinks about the stimulus (Atlas & Wager, 2012; Buhle & Wager, 2010; Meissner et al., 2011). The most commonly studied exemplar of cognitive change is *reappraisal*, which involves reinterpreting the meaning of a stimulus, or mentally modifying the emotional relevance of it, in order to change subsequent emotional responses. Reappraisal is one of the most cognitively complex strategies, drawing on language and memory to reference semantic knowledge about the stimulus, working memory to maintain and manipulate appraisals, response selection to pick goal-congruent appraisals, and self-monitoring to ensure that one is reappraising successfully (Ochsner et al., 2002; Ochsner & Gross, 2005).

Reappraisals themselves can be accomplished in a number of ways. Two of the most common subtypes are *reinterpretation* and *distancing*. Reinterpretation involves changing how one understands or 'interprets' the emotion-eliciting situation or stimulus (e.g., (Ochsner et al., 2002)). In the case of the alcoholic, he might think about how a beer contains many calories, and that turning down a drink will help him achieve his weight loss goals. *Distancing* involves changing one's personal connection to, or psychological distance from, the emotion-eliciting stimulus (e.g., (Ochsner et al., 2004)). For example, the alcoholic facing a surging desire to drink may reduce the intensity of his emotional response by imagining himself as an objective friend viewing the situation from the outside.

Finally, *response modulation* strategies target the systems for emotion-expressive behavior. The most commonly studied exemplar is *expressive suppression* (e.g., (Goldin, McRae, Ramel, & Gross, 2008; Gross, 1998a; Hayes et al., 2010)), which entails keeping the face still so that an observer could not detect that one is experiencing an emotion. Because expressive suppression only targets the final stage of the emotion generation process, it influences emotional experience subtly, if at all (J. I. Davis, Senghas, & Ochsner, 2009; Goldin et al., 2008). However, the effort expended during expressive suppression may increase physiological arousal (Gross, 1998a). Other forms of response modulation, such as expressive enhancement (Bonanno, Papa, Lalande, Westphal, & Coifman, 2004; Jackson, Malmstadt, Larson, & Davidson, 2000) and expressive change, also exist, but they have received little attention in the neuroimaging literature thus far.

The differences among these strategies may lead to differences in long-term efficacy and real-world use. For example, reappraisal, but not distraction, has been shown to have long-lasting effects on one's tendency to have an emotional response to a stimulus (Kross & Ayduk, 2008), presumably because only reappraisal involves an active change in how one represents the affective meaning of that stimulus. However, the active change induced by reappraisal requires engagement with the emotional content, which may be difficult or unappealing. One recent study showed that participants, when

allowed to choose which emotion regulation strategy to implement, typically used reappraisal in low-intensity negative situations, but preferred the disengagement-based strategy of distraction in high-intensity negative situations (Sheppes, Scheibe, Suri, & Gross, 2011).

Neural Bases of Emotion Regulation

Over the last decade, functional imaging research in healthy human adults has provided tremendous insight into the nature of the source regions that implement regulatory strategies as well as the target systems that are acted upon during reappraisal. This section discusses core conclusions that can be drawn from reappraisal research and a general model of emotion regulation that can be derived from it.

Reappraisal as a paradigm case Reappraisal is an appropriate starting point for developing a model of the cognitive control of emotion for five reasons. First, because reappraisal is among the most cognitively complex strategies, a model of emotion regulation derived from reappraisal work may be generally applicable to relatively simpler strategies and phenomena. Second, the majority of neuroimaging studies to date have focused on reappraisal. Third, reappraisal is deeply engrained in our culture, as evidenced by countless well-known aphorisms that remind us that, "Life is what you make of it", "April showers bring forth may flowers" and "It's all grist to the mill". Fourth, in contrast to other areas of emotion regulation research (see section *Extending the Model to Other Forms of Emotion Regulation*, below) reappraisal studies tend to be more methodologically and conceptually similar to one another and therefore provide a stronger base for mechanistic inferences. Finally, reappraisal is an important component of many therapeutic techniques, including cognitive behavioral therapy (Beck, 2005) and dialectical behavioral therapy (Lynch, Trost, Salsman, & Linehan, 2007). With these considerations in mind, we now review the neural systems that have been most consistently observed in studies of reappraisal (Ochsner & Gross, 2008).

Implementation of reappraisal Figure 1B schematically illustrates the brain systems shown by current research to be involved in the cognitive control of emotion via reappraisal, while Figure 2 plots peak activation foci for 43 studies (see Table 1) of reappraisal in healthy individuals. As these plots demonstrate, a great deal of evidence now supports the hypothesis that the cognitive regulation of emotion is implemented largely by the same frontoparietal control regions that regulate memory, attention, and other thought processes (Ochsner et al., 2002). In this section we consider the possible roles in reappraisal of some of the most commonly observed regions, including dorsolateral prefrontal cortex (dlPFC), inferior parietal cortex (iPC), dorsal anterior cingulate (dACC) and adjacent posterior medial prefrontal cortex (dmPFC), ventrolateral prefrontal cortex (VLPFC), and anterior dmPFC.

It is important to note that neuroimaging studies of reappraisal have varied along a number of experimentally significant dimensions, including tactics (reinterpretation or distancing), valence of stimuli/emotions elicited (positive or negative), modulatory direction (decrease or increase), and a number of studies have even directly examined

how altering these dimensions impacts neural responses during reappraisal. In keeping with the scope of this chapter, the present discussion will largely consider reappraisal as a whole, looking at general patterns that are consistent across these variations. We encourage the interested reader to see another recent review for a more thorough assessment of how paradigm differences meaningfully influence patterns of neural recruitment (Ochsner et al., Submitted).

Dorsolateral prefrontal cortex and inferior parietal cortex Together, dlPFC and iPFC are believed to constitute a dorsal frontoparietal network for the endogenous control of attention and working memory (Corbetta & Shulman, 2002). While attention and working memory are supported by complex interactions between dlPFC and iPC, the precise roles they play in these processes are somewhat distinct. On the one hand, dlPFC appears to play more of an executive role in working memory and attentional processes by maintaining goals (Ptak, 2011) and monitoring the contents of working memory (Chamod & Petrides, 2010). On the other hand, iPC appears to initiate shifts in attention to goal-relevant stimuli (Ptak, 2011) and to manipulate components in working memory in accordance with goal states represented in dlPFC (Chamod & Petrides, 2010). In the context of reappraisal, this network is may be used to direct attention to reappraisal-relevant stimulus features, to hold in mind reappraisal goals, and to manipulate information during the construction of new appraisals.

Dorsal anterior cingulate and posterior dorsomedial prefrontal cortex Neuroimaging and lesion research has broadly associated the dACC and posterior dmPFC with the initiation and maintenance of controlled processing (Bush, Luu, & Posner, 2000; Ochsner et al., 2001; Paus, 2001). In the most well-known models, dACC and posterior dmPFC detect conflict, errors, or other performance signals, and may then call upon dlPFC to implement needed control (Botvinick, Braver, Barch, Carter, & Cohen, 2001; Botvinick, Cohen, & Carter, 2004; Gehring & Knight, 2000; MacDonald, Cohen, Stenger, & Carter, 2000; Miller, 2000; Miller & Cohen, 2001). In other models, dACC and posterior dmPFC do not play a role in monitoring, but are directly responsible for activating and sustaining task representations and goals (Stuss & Alexander, 2007). In the context of reappraisal, these regions might track the extent to which one's current reappraisals are changing emotional responses in the intended way and recruit dlPFC to improve or modify reappraisal processes as necessary (Denny, Silvers, & Ochsner, 2009; Ochsner & Barrett, 2001; Ochsner & Gross, 2004), or dACC and posterior dmPFC may serve to directly activate and sustain the task representations and goals needed to implement reappraisals.

An alternative view of the dACC and posterior dmPFC is that these regions help coordinate appropriate autonomic responses during emotion generation and regulation. When stimulated, the dACC is one of the few cortical areas that can cause changes in heart rate, respiration and gastric motility (Burns & Wyss, 1985; Hurley-Gius & Neafsey, 1986; Kaada, Pribram, & Epstein, 1949; Pool & Ransohoff, 1949). In both animals and humans, dACC and posterior dmPFC lesions have been shown to alter cardiac and skin conductance responses to stimuli with learned affective/physiological significance as well as to increasingly demanding cognitive tasks (Buchanan & Powell, 1982a, 1982b;

Naccache et al., 2005; Neafsey, 1990; Zahn, Grafman, & Tranel, 1999). Neuroimaging studies also suggest that dACC and posterior dmPFC mediates skin conductance responses to conditioned stimuli and is specifically involved in the acquisition of conditioned aversive responses (Etkin, Egner, & Kalisch, 2011; Schiller & Delgado, 2010). Taken together, this suggests that the dACC is important for controlling learned physiological responses and may therefore also be critical for modifying physiological responses in the context of reappraisal (e.g., reducing skin conductance responses to an aversive stimulus that is being reappraised).

Ventrolateral prefrontal cortex Left vIPFC has been implicated in selecting goal-appropriate responses and information from semantic memory (Badre & Wagner, 2007; Thompson-Schill, Bedny, & Goldberg, 2005) and in the production of speech (Bookheimer, 2002; Huang, Carr, & Cao, 2002), including internal speech (Hinke et al., 1993; Huang et al., 2002). In the context of reappraisal, left vIPFC may be used to deliberately select semantic elements needed to construct a new stimulus-appropriate, verbally-mediated reappraisal. We would expect this type of semantic selection to be especially important during *reinterpretation*, a reappraisal tactic that involves changing one's interpretation of the elements of the situation or stimulus that elicits emotion. Conversely, we would expect less of a need for semantic selection during *distancing*, a reappraisal tactic that involves changing one's personal connection to, or psychological distance from, the stimulus that elicits emotion. In line with this psychological prediction, reinterpretation more frequently evokes activity in left vIPFC than does distancing (Ochsner et al., Submitted).

Right vIPFC has been implicated in the inhibition of prepotent, goal-inappropriate responses (Aron, Robbins, & Poldrack, 2004; Konishi et al., 1999; Lieberman et al., 2007; Ochsner, 2005). In the context of reappraisal, right vIPFC may serve to inhibit one's initial appraisal in favor of a goal-congruent reappraisal. Consideration of the type of reappraisal implemented can provide a useful test of this hypothesized function. While decreasing an emotional response would typically require that the inhibition of an initial appraisal and its replacement with a less negative alternative, increasing a response would typically only involve amplifying one's initial response, and so inhibitory demands should be lower. In keeping with this psychological interpretation, several studies that compared decreasing and increasing found greater right vIPFC activation for the decreasing condition (Kim & Hamann, 2007; Ochsner et al., 2004; Urry, 2009), and this pattern seems to hold across the larger set of studies that have examined decreasing or increasing conditions separately (Ochsner et al., Submitted).

Anterior dorsomedial prefrontal cortex Anterior dmPFC has been implicated in reflection on and making judgments about the mental states of oneself and others (Amodio & Frith, 2006; Denny, Kober, Wager, & Ochsner, in press; Mitchell, 2009; Olsson & Ochsner, 2008) (see Chapter on Mental State Attribution). In the context of reappraisal, one possibility is that anterior dmPFC might be important both for assessing the effect of one's initial appraisal on one's mental state, and also for assessing one's new mental state following reappraisal. Alternatively, or perhaps additionally, anterior dmPFC might support attention to and elaboration of emotional

states, intentions, and outcomes of the individuals depicted in the photographic stimuli typically used in these studies. This second possibility is consistent with the predominance of anterior dmPFC in reappraisal tasks in which the goal is to increase emotion (Ochsner et al., Submitted). Of the 12 studies that directly compared increasing emotion to a control condition where participants responded naturally, six showed increases in anterior dmPFC (Domes et al., 2010; Ichikawa et al., 2011; S. Lang et al., 2011; Ochsner et al., 2004; Ochsner et al., 2009). Of the six that did not, most showed activation in neighboring areas (such as anterior cingulate and paracingulate cortex) (Eippert et al., 2007; New, Fan, et al., 2009; Pitskel, Bolling, Kaiser, Crowley, & Pelfrey, 2011; Schulze et al., 2010; Urry, 2009; van Reekum et al., 2007).

Targets of reappraisal In the previous section, we reviewed the regions believed to be responsible for the implementation of reappraisal. These regions can be thought of as the sources of emotional control. But what are the targets of this control? Given that reappraisal effectively modulates self-reported emotional experience, as well as other behavioral and physiological correlates of emotion, we would also expect to see modulation in regions involved in generating emotions. In this section, we will review the putative target regions that have garnered the most attention in the reappraisal literature thus far, including the amygdala, ventral striatum, and insula.

As we described earlier, neuroimaging studies of reappraisal have varied along a number of dimensions, including tactics, stimulus/emotional valence, and regulatory goals. Again, we will largely consider reappraisal as a whole, looking at patterns that cut across together these different task dimensions. However, we will pay special attention to task differences that occur as a function of valence, as we would expect differently valenced emotions to involve considerably different appraisal systems. For a more detailed discussion of the variability seen in different types of reappraisal tasks, we again direct the interested reader to a recent review (Ochsner et al., Submitted).

Amygdala. The amygdala is believed to support the detection and appraisal of stimuli relevant to one's current or chronic affective goals (Cunningham, Arbuckle, Jahn, Mowrer, & Abduljalil, 2011; Cunningham, Van Bavel, & Johnsen, 2008). While the amygdala most consistently has been shown to respond to aversive stimuli such as punishments, fearful facial expressions, and negatively valenced films or images (Neta & Whalen, 2011; Vuilleumier & Pourtois, 2007; Whalen et al., 2004), it has also been shown to respond to positive stimuli, such as rewards, as well as other forms of salient, non-affective stimuli (M. Davis & Whalen, 2001; Hariri & Whalen; Phelps, 2006) (Anderson, Christoff, Stappen, et al., 2003).

As can be seen in Figure 2B, many studies have reported changes in the amygdala as a consequence of reappraisal. These studies have most typically involved the reduction of negative affect, with only a few studies examining, and showing, increases in amygdala when the goal was to increase an emotion, or the valence was positive (see Table 1). Notably, the few cases in which the modulation of positively valenced emotions resulted in amygdala modulation were left-lateralized (Herwig et al., 2007;

Ohira et al., 2006; Vrticka, Sander, & Vuilleumier, 2011; Winecoff, Labar, Madden, Cabeza, & Huettel, 2010). While this is consistent with some older hypotheses about valence lateralization (Davidson & Sutton, 1995), the imaging literature in general hasn't borne this out (Sergerie, Chochol, & Armony, 2008; Wager, Phan, Liberzon, & Taylor, 2003) and the reliability and reasons for lateralization effects in reappraisal await further work.

Ventral Striatum The ventral striatum is believed to be involved in learning the relationships between cues (ranging from social signals, like smiling faces, to actions to abstract objects) and rewarding or reinforcing outcomes (Knutson & Cooper, 2005; O'Doherty, 2004; Schultz, 2007).

As can be seen in Figure 2B, a number of studies have reported changes in the ventral striatum as a consequence of reappraisal. As with the amygdala, both reappraisal of positive and of negative emotions have been shown to modulate the striatum. However, in contrast to the amygdala, changes in ventral striatum have been seen less frequently in the case of negative emotions, and more frequently in the case of positive emotions (Ochsner et al., Submitted). That said, because only a relatively small number of studies have examined positively-valenced emotions, it is difficult to draw firm conclusions. As noted above, an important future direction for reappraisal studies is to more thoroughly examine how valence influences regulatory targets.

Insula The insular cortex has been theorized to represent a viscerotopic map of ascending inputs from the body (Mufson & Mesulam, 1982) that some believe is essential to negative affective experience in general (Craig, 2009; Wager & Feldman Barrett, 2004). Within the insula, more posterior regions are associated with the representation of sensations from the body while more anterior regions have been linked with motivational and affective states, like disgust, that have a strong visceral component (Augustine, 1996; Calder, Lawrence, & Young, 2001; Craig, 2009; Critchley, Wiens, Rotshtein, Ohman, & Dolan, 2004; Nitschke, Sarinopoulos, Mackiewicz, Schaefer, & Davidson, 2006; Wager & Barrett, 2004).

While several studies have reported target-related activity in the posterior insula, only a few studies have a few have reported modulation in anterior insula. One possible explanation for this surprising dearth of findings in the anterior insula may stem from the proximity of this region to vIPFC regions involved in the implementation of reappraisal. Target regions are typically detected using the reverse contrast used to detect source regions. For example, a contrast between reappraisal and a passive viewing, naturalistic response condition may be used to detect source regions that support the implementation of reappraisal, while the reverse contrast may be used to detect target regions modulated by reappraisal. As described above and depicted in Figure 2A, vIPFC regions are often activated in the implementation of reappraisal. This increase in activity may overwhelm any decrease that would otherwise be observed in adjacent portions of anterior insula.

Other areas involved in reappraisal Several other areas that appear to be involved in reappraisal do not fit neatly into the source-target dichotomy. One such area is ventromedial prefrontal cortex (VMPFC), which has been proposed as both a source and target of reappraisal. We will also review evidence in support of the hypothesis that modulation of these emotion processing regions results from earlier modulation of semantic and perceptual representations in temporal and occipital regions known to support perceptual and semantic representations (see Figure 1B).

Ventromedial prefrontal cortex The ventromedial prefrontal cortex (vmPFC) is hypothesized to integrate memorial and semantic information stored in the medial temporal lobes, affective appraisals of specific stimuli formed by subcortical structures such as the amygdala and ventral striatum, and inputs from other regions that provide information about current behavioral and motivational goals such as the brainstem and prefrontal cortex (Cunningham, Johnsen, & Waggoner, 2011; Davachi, 2006; Fellows, 2011; Murray, O'Doherty, & Schoenbaum, 2007; Ochsner et al., 2002; Ongur, Ferry, & Price, 2003; Price, 1999; Rudebeck & Murray, 2011; Schoenbaum, Takahashi, Liu, & McDannald, 2011). As such, vmPFC activity may scale with the affective value one attributes to a stimulus in a situational and goal-dependent manner (Oya et al., 2005; Roy, Shohamy, & Wager, 2012; Schoenbaum, Saddoris, & Stalnaker, 2007; Schoenbaum et al., 2011). Examples of this include affective learning, including fear extinction and reversal learning, the finding that vmPFC responses to an image of a healthy food are modulated by whether one has the goal to eat healthily (Hare, Camerer, & Rangel, 2009), and the finding that vmPFC lesions lead to context-inappropriate affective responses in both humans and animals (Beer, Heerey, Keltner, Scabini, & Knight, 2003; Damasio, 1994; Murray et al., 2007).

At present, vmPFC's role in reappraisal remains somewhat ambiguous. While some have suggested that it may be used to implement reappraisal (Diekhof, Geier, Falkai, & Gruber, 2011; Schiller & Delgado, 2010), in simple main effect contrast analyses no studies have found vmPFC to be more strongly recruiting during reappraisal in comparison to responding naturally. However, in such contrasts a handful of studies have found vmPFC activity to be *diminished* when downregulating emotion in comparison to when responding naturally, or when upregulating emotion, particularly for positive stimuli (Kanske et al., 2011; Kim & Hamann, 2007; Kober et al., 2010; Schardt et al., 2010; Winecoff et al., 2010). This suggests that vmPFC may actually be a target of reappraisal.

Several studies have also found that individuals showing more robust modulation of the amygdala or insula during reappraisal tend to show stronger inverse connectivity between vmPFC and such appraisal structures (Pitskel et al., 2011; Urry et al., 2006). Additionally, it appears as though such connectivity differs between individuals according to psychiatric status (Erk, Mikschl, et al., 2010; Johnstone, van Reekum, Urry, Kalin, & Davidson, 2007), genotyping (Schardt et al., 2010) or reappraisal success (Wager, Davidson, Hughes, Lindquist, & Ochsner, 2008). Taken together, this suggests

that vmPFC may relate to between-individual differences in regulation, although more work is needed to further clarify why this may be the case.

Perceptual and semantic representations Related to how we conceptualize the role of the VMPFC is the question of how reappraisal may not only modulate affective appraisal systems but systems that process and represent perceptual and semantic properties of affective stimuli as well. As is shown in Figure 2, reappraisal often involves activation of middle and superior temporal cortex, regions known to represent high-level visual stimuli including facial features and biological motion (Allison, Puce, & McCarthy, 2000; Brefczynski-Lewis, Berrebi, McNeely, Probst, & Puce, 2011; Wheaton, Thompson, Syngienotis, Abbott, & Puce, 2004), temporal polar regions thought to be involved semantic knowledge about emotion and the binding of emotional and perceptual representations (Olson, Plotzker, & Ezzyat, 2007), and the temporal-parietal junction, a region involved in perspective taking that may be critical when one generates and compares alternative appraisals of a stimulus (Saxe, 2006; Young, Camprodon, Hauser, Pascual-Leone, & Saxe, 2010).

In order to understand the functional role that these temporal regions play in reappraisal, at least three issues that need to be addressed. First, there is the issue of how consistently these regions are recruited across different types of emotion regulation. It makes theoretical sense that such regions would be important for reappraisal given that reappraisal involves changing the meaning of a stimulus. However, it is less clear whether other regulatory strategies that do not involve changing the meaning of a stimulus might rely on such areas. The fact that direct comparisons between reappraisal and distraction have found that the two strategies differentially recruit posterior temporal cortex (reappraisal relies on these regions more) supports the notion that activity in this region may differ across regulatory strategies (Kanske et al., 2011; McRae et al., 2010).

Second, there is the question as to what cognitive process or processes are driving activation of these temporal regions. Greater activity might reflect increased attention to perceptual and semantic aspects of stimuli. Alternatively, greater activity may reflect increased retrieval of alternative interpretations of reappraised stimuli, or the process of actively restructuring one's mental image of the stimulus. Future work should attempt to distinguish among these possibilities.

Third, there is the question of how these temporal regions fit into the neural model of emotion regulation described earlier and depicted in Figure 1B. One possibility is that they play an intermediary role between prefrontal control systems and affective appraisal systems (Ochsner et al., 2002; Ochsner et al., Submitted). According to this view, PFC and parietal regions could change one's mental representation of a stimulus's meaning from the top down, directly altering perceptual and semantic processing in these temporal regions. This reappraised representation in turn feeds forward to the amygdala and other structures that trigger affective responses. Because the amygdala now, 'sees,' the reappraised stimulus, its response changes. While consistent with the existing data, these hypotheses have yet to be directly tested.

Extending the Model to Other Forms of Emotion Regulation

The majority of functional imaging studies of emotion regulation have focused on reappraisal. That said, the other four main classes of emotion regulation strategies diagrammed in Figure 1A have been targeted by imaging studies to varying degrees. Given the robustness of the neurocognitive model depicted in Figure 1B in accounting for reappraisal, the question naturally arises as to whether this model can be generalized to account for other types of emotion regulation strategies. Here, we briefly discuss each of the four other classes of regulation, using the model derived from reappraisal as a starting point for analysis.

Situation selection and modification While situation-focused strategies are effective in certain situations (J. I. Davis, Gross, et al., 2009), they are difficult to study in a neuroimaging context and so little is known about the neural processes involved in humans. In the rodent literature, a typical avoidance conditioning paradigm consists of a rat learning to perform an action that allows it to avoid or remove the presentation of an aversive stimulus (Everitt et al., 1999; LeDoux & Gorman, 2001). In a handful of studies examining avoidance conditioning in humans, it has been shown that avoidance conditioning relies upon vIPFC and dlPFC control systems and modulates the amygdala (Delgado, Jou, Ledoux, & Phelps, 2009; Prevost, McCabe, Jessup, Bossaerts, & O'Doherty, 2011; Schlund & Cataldo, 2010; Schlund et al., 2010). These findings provide preliminary evidence for the notion that situation selection may depend upon systems relevant for maintaining regulatory goals and selecting context-appropriate avoidance responses.

Attentional Deployment In comparison to situation selection and modification, there have been numerous human neuroimaging studies of attentional deployment, second in number only to studies of reappraisal. As described above, such research can be broken down into two types of studies. The first of these types involves the use of selective attention to shift visual spatial attention away from an affectively valenced stimulus or stimulus attribute and towards a neutral one. The second type focuses on the use of distraction to shift the focus of attention away from an affective stimulus and onto some internally maintained mental representation (e.g. a relevant working memory load, self generated stimulus-irrelevant thoughts, a pleasant mental image, and so on). As has been suggested elsewhere (Buhle, Wager, & Smith, 2010; Ochsner & Gross, 2005), interpretation of results found in both types of studies is challenged by three issues. First, the vast majority of selective attention studies, and many studies of distraction, use stimuli that do not elicit strong emotional responses, such as facial expressions of emotion. As such, these studies are concerned with the regulation of evaluative judgment or perception rather than affective responding, per se. Second, among the studies that have used highly arousing and affect-inducing stimuli, the stimulus of choice has almost always been physical pain. While the experience of pain has a strong negatively-valenced affective component, this component may itself have a distinct neural signature that depends in part upon dedicated pain-specific neural pathways (Apkarian, Bushnell, Treede, & Zubieta, 2005; Tracey & Mantyh, 2007). Whether or not regulation of pain is similar to or different from the regulation of negative

affective responses more generally remains an empirical question in need of testing. Third, attentional deployment studies tend to be highly heterogeneous, often employing very different methods of controlling the focus and level of attention, often without a clear or consistently defined dependent variable for how well attention was controlled. Despite these limitations, it is worth noting that the results of attentional deployment studies are generally consistent with the model depicted in Figure 1B in so far as activation of prefrontal systems and modulation of affect systems (like the amygdala) are often (but not always) reported.

Response Modulation Only two imaging studies have examined response modulation (Goldin et al., 2008; Hayes et al., 2010). Both focused on expressive suppression, the ability to hide behavioral manifestations of emotion (Gross, 1998a), asking participants to suppress facial expressions of disgust elicited by a film clip (Goldin et al., 2008; Hayes et al., 2010). Both studies found that expressive suppression activated dorsolateral and ventrolateral PFC regions associated with maintaining goals, response selection and inhibition (Aron et al., 2004; Badre & Wagner, 2007; Thompson-Schill et al., 2005), as well as the insula, which is involved in the formation of affective responses. Amygdala findings were more mixed, however, with one study reporting increases (Goldin et al., 2008) and one decreases (Hayes et al., 2010) in activity during suppression. Increases in insula and amygdala fit with psychophysiological studies demonstrating that expressive suppression enhances autonomic measures of emotional responding (Gross, 1998a).

In total, the available literature on emotion regulation strategies other than reappraisal is in some cases limited and in other cases somewhat confusing, but in general supports the idea that all emotion regulation strategies depend on interactions between cognitive control and affect generative regions.

Emotion Regulation in Development, Aging and Psychopathology

The first goal of this paper was to review and synthesize current functional imaging research on emotion regulation in healthy adults. However, another important direction for emotion regulation research is the translation of basic findings to special populations. Two domains in which this will prove particularly significant is understanding 1) how our emotional lives evolve as we grow from childhood through adolescence into adulthood and old age, and 2) how emotional reactivity and regulation are impacted in psychopathology.

Development of emotion regulation

There is growing evidence that childhood and adolescence are critical times for development of the emotion regulatory abilities needed to adaptively balance affective impulses and the deleterious health behaviors they can promote. Demands for self-regulation are high in adolescence in particular as individuals experience increased independence, hormonal changes and a changing social environment (Blakemore, 2008; Casey, Getz, & Galvan, 2008; Somerville, Jones, & Casey, 2010). Most

individuals successfully navigate the challenges of adolescence by developing regulatory skills that will help them to cope with stressors for the rest of their lives. However, for some individuals adolescence marks the beginning of a lifelong struggle with emotion regulation and mental and physical health. Not only does the peak age of onset for mental illness occur during adolescence (Kessler et al., 2005), lifelong problems with alcohol and substance abuse, obesity and eating disorders (Aldao, Nolen-Hoeksema, & Schweizer, 2010; Baumeister & Heatherton, 1996; Chandler, Fletcher, & Volkow, 2009; Gupta, Zachary Rosenthal, Mancini, Cheavens, & Lynch, 2008; Herman, Polivy, Lank, & Heatherton, 1987; Houben & Wiers, 2009; Volkow et al., 2010) often have their origins in adolescence. Given this, a critical question is how emotion regulatory mechanisms develop during adolescence when individuals are at greatest risk for developing maladaptive patterns of emotional regulation and unhealthy behaviors.

While a considerable number of behavioral studies have suggested that on average adolescents experience more extreme affect (both positive and negative) and more variable mood states in their everyday lives than do adults (Larson, Csikszentmihalyi, & Graef, 1980; Larson, Moneta, Richards, & Wilson, 2002; Larson & Richards, 1994), this work has been somewhat contradictory with regards to the reasons these data are observed. For example, this work doesn't make clear whether emotional responsivity decreases linearly from childhood to adolescence to adulthood (Carthy, Horesh, Apter, Edge, & Gross, 2010; Murphy, Eisenberg, Fabes, Shepard, & Guthrie, 1999), whether it changes in a quadratic fashion, with emotionality being highest in adolescents (Casey et al., 2008; Casey et al., 2010), or whether it is both linear and quadratic in nature (R. W. Larson, Moneta, Richards, & Wilson, 2002; L. A. Thomas, De Bellis, Graham, & LaBar, 2007) (see Figure 3).

To address this issue, studies would need to disentangle the developmental trajectories of emotional reactivity and regulation. To date, however, few studies have done so (for notable exceptions, see Murphy, Eisenberg, Fabes, Shepard, & Guthrie, 1999; Silk, Steinberg, & Morris, 2003), which makes it difficult to determine whether age-related differences in emotional responsivity are due to differences in bottom-up emotional reactivity or top-down emotion regulation. That said, reappraisal has been examined in limited age groups (Carthy et al., 2010; Levesque et al., 2004; Moore, Mischel, & Zeiss, 1976; Pitskel et al., 2011), with two studies comparing emotional reactivity (baseline responsiveness to affective stimuli) and regulation success (the ability to use regulatory strategies to modulate emotional responses) in individuals at the beginning, middle and end of adolescence (McRae, Gross, et al., 2012; Silvers et al., in press). Both of these studies found that emotional reactivity remains relatively constant across adolescence while regulation success improves.

Imaging studies have just begun to address the reactivity vs. regulation issue using a combination of structural and functional methods. In general, this work has been motivated by two kinds of findings, the first from studies of emotional reactivity and the second from studies of the development of control systems.

First, volumetric MRI studies indicate that the amygdala increases in size during puberty, with some studies concluding that these changes occur rapidly at the beginning of adolescence before tapering off (Ostby et al., 2009) and others reporting steady linear increases over the course of adolescence (Giedd et al., 1996; Schumann et al., 2004). These structural findings may help to explain why in functional imaging studies adolescents are particularly sensitive to the motivational properties of affective stimuli (Casey et al., 2010), showing enhanced striatal and amygdala responsivity to the receipt of rewards (Ernst et al., 2005; Galvan et al., 2006; Geier, Terwilliger, Teslovich, Velanova, & Luna, 2009; van Leijenhorst, Crone, & Bunge, 2006; Van Leijenhorst et al., 2010) and perception of fear faces (Hare et al., 2008; Killgore & Yurgelun-Todd, 2001, 2007; Monk et al., 2003; K. M. Thomas et al., 2001), respectively. Second, there is evidence of structure-function-behavior relationships in “cold” forms of cognitive control (like working memory and response inhibition) during adolescent development. Here, studies have shown that prefrontal control systems mature later than subcortical systems, with PFC white matter increasing linearly throughout adolescence (Barnea-Goraly et al., 2005; Giedd et al., 1999; Pfefferbaum et al., 1994) and pruning of PFC gray matter starting around puberty and continuing into one’s twenties (Gogtay et al., 2004). Strikingly, these structural changes are paralleled by improved performance on control tasks, with performance improvements correlating with decreased activation of task-irrelevant regions and increasingly focal activations in task-relevant regions (Casey, Tottenham, Liston, & Durston, 2005; Durston & Casey, 2006; Durston et al., 2006; Luna, Padmanabhan, & O’Hearn, 2010).

Building on these findings, current studies have aimed to examine emotion regulatory processes in adolescence in two ways. First, studies of selective attention have shown that children and adolescents are more susceptible to the influence of affective stimuli than adults, showing age-related inverted-U response patterns in the striatum and amygdala to happy and fear faces, respectively, that peak during adolescence (Hare et al., 2008; Somerville, Hare, & Casey, 2011), coupled with age-related declines in vIPFC activation (Somerville et al., 2011). While face stimuli do not generally elicit strong affective responses, these data are generally consistent with adolescent hyperresponsivity to affective stimuli coupled with immature control circuitry (Somerville et al., 2010). Second, a handful of imaging studies have begun to assess reappraisal in developmental samples (Levesque et al., 2004; McRae, Gross, et al., 2012; Perlman et al., 2012; Pitskel et al., 2011). While limited age ranges and small sample sizes have constrained the interpretability of some studies, there is evidence to suggest that age is associated with both linear and quadratic (inverted U-shaped) increases in activity in dorsal and lateral prefrontal regions known to support reappraisal in adults (McRae, Gross, et al., 2012). What’s more, a recent study comparing adolescents with major depressive disorder (MDD) to healthy controls (HCs) found that HCs recruited vIPFC to a greater extent than did adolescents with MDD (Perlman et al., 2012).

While developmental cognitive neuroscience research on emotion regulation remains a relatively “young” area, two conclusions can be gleaned from extant data. The first is that the ability to control one’s emotions appears to follow a developmental trajectory similar to what has been observed in studies of “cold” cognitive tasks, although perhaps

more protracted. The second is that this shift towards greater emotional control is supported by structural and functional maturation in prefrontal control regions known to support reappraisal in healthy adults.

Emotion regulation in aging

Over the next 20 years, the age distribution in the United States will shift such that older adults (65 years of age or older) will comprise ~20% of the population (Vincent & Velkoff, 2010). As such, understanding the psychological and neural mechanisms supporting emotional health in older adults is of importance at both the individual and societal levels. When considering emotion regulation in aging, we are faced with something of a paradox (Mather, In press). On the one hand, much behavioral research has suggested that with old age comes a more positive outlook, or “rosy glow”, that brings with it more stable and satisfying emotional wellbeing (Carstensen & Mikels, 2005; Carstensen et al., 2011; Scheibe & Carstensen, 2010). On the other hand, age-related cortical thinning is seen in lateral and dorsal PFC regions known to support reappraisal (Fjell et al., 2009) along with declines in performance on “cold” cognitive control tasks (e.g. response inhibition) that depend on these regions (Park & Reuter-Lorenz, 2009). How might we reconcile these two observations?

In comparison to younger adults, older adults report less negative affect and show reduced autonomic and amygdala responses to aversive stimuli (Levenson, Carstensen, & Gottman, 1994; St. Jacques, Bessette-Symons, & Cabeza, 2009; Tsai, Levenson, & Carstensen, 2000). Given that the amygdala remains structurally intact during aging and that age-related decreases in amygdala activity are often accompanied by enhanced prefrontal activity (Nashiro, Sakaki, & Mather, 2011), some have suggested that age-related decreases in negative affect may be driven by the use of top-down regulation strategies. Supporting this notion is the fact that age-related biases to report more positive and less negative emotion in response to stimuli are strongest for low arousal stimuli for which top-down appraisal processes may play a greater role in generating and regulating emotions (Streubel & Kunzmann, 2011). While this evidence is suggestive, only two studies have directly examined whether there are age-related changes in reappraisal-related modulation of the amygdala. One found that older adults could successfully decrease amygdala responses to both positive and negative photos (Winecoff et al., 2010). The other reported no baseline amygdala responses in old or young adults (Opitz, Rauch, Terry, & Urry, in press), so regulatory effects could not be examined.

While older adults report that they regularly use reappraisal (Gross et al., 1997), several experimental studies have suggested that they are actually less able to decrease negative emotion (Opitz et al., in press; Shiota & Levenson, 2009; Tucker, Feuerstein, Mende-Siedlecki, Ochsner, & Stern, in press; Winecoff et al., 2010). However, this reduced ability may be a function of the reappraisal tactic used. While older adults show deficits in reappraising when using a neutralizing tactic, in which one attempts to think about stimuli in unemotional terms, they are just as good as young adults when using a positivizing tactic (Shiota & Levenson, 2009), where one seeks positive ways to reframe a stimulus's meaning (McRae, Ciesielski, & Gross, in press). Interestingly, another study

found that while older adults were unable to effectively use reappraisal to *decrease* their negative affect, they were able to use reappraisal to *increase* negative affect (Opitz, Rauch, Terry, & Urry, 2011). Given that anterior dmPFC has been implicated in upregulation of emotion in prior work (Ochsner et al., 2004), this suggests that older adults may be better at reappraising when the reappraisal goals rely on dmPFC-supported processes in comparison to when reappraisal goals rely on processes supported by dorsal and lateral PFC. Indeed, both older and younger adults recruit dmPFC to a similar degree in several tasks known to involve this area, including making self-referential (Gutchess, Kensinger, & Schacter, 2007) or semantic (Ritchey, Bessette-Symons, Hayes, & Cabeza, 2011) judgments about valenced stimuli, and more generally when viewing positive stimuli (Kensinger & Schacter, 2008; Leclerc & Kensinger, 2008, 2011). Consistent with these hypotheses, two neuroimaging studies of reappraisal in older adults have shown weaker vIPFC activity accompanied by impaired ability to decrease negative emotion (Opitz et al., in press; Winecoff et al., 2010). Furthermore, one of these studies found that activity in a dACC region adjacent to anterior dmPFC mediated older adults greater success at increasing negative emotion (Opitz et al., in press).

Taken together, this work suggests that older adults have preserved reappraisal abilities when reappraising using a positivizing tactic or when the reappraisal goal is to increase emotion. However, it may also be the case that older adults struggle to reappraise when asked to use tactics and follow goals that are not consistent with their chronic regulatory tendencies or to draw upon cognitive processes that decline with age, such as working memory and response selection processes involved in neutralizing negative emotion that are supported in lateral PFC.

Emotion regulation in psychopathology

A second important goal for translational research will be to understand how potential dysfunction in the mechanisms of emotion generation and regulation may underlie various forms of psychopathology. This translational direction is being pursued in studies of reappraisal across various disorders, ranging from borderline personality disorder (Koenigsberg et al., 2010; S. Lang et al., 2011; Schulze et al., 2010), to depression (Erk, Mikschl, et al., 2010; Heller et al., 2009; Johnstone et al., 2007) to anxiety disorders (Goldin, Manber, Hakimi, Canli, & Gross, 2009; Goldin, Manber-Ball, Werner, Heimberg, & Gross, 2009), including phobia (Hermann et al., 2009) and posttraumatic stress disorder (S. Lang et al., 2011; New, Fan, et al., 2009). In the next section of this chapter, we will highlight the research findings to date on the neural bases of emotion regulation in each of these disorders.

Borderline Personality Disorder Emotional instability, particularly in the context of interpersonal relationships, is one of the hallmarks of Borderline Personality Disorder (BPD) (Gunderson, 2007; Gunderson & Lyons-Ruth, 2008). Not only is this instability a trademark feature of BPD, it is also one of the most destructive aspects of this disorder in that it is associated with suicidality, extreme anger and feelings of emptiness (Koenigsberg et al., 2001; Linehan, 1993). Structural MRI research has linked this tendency to experience heightened emotional reactivity to decreased amygdala and

insula volumes, regions associated with emotional responding, as well as reduced ACC volumes, a region associated with emotion regulation (Nunes et al., 2009; Rusch et al., 2003; Schmahl, Vermetten, Elzinga, & Douglas Bremner, 2003; Soloff et al., 2012; Tebartz van Elst et al., 2003). Building on these findings, fMRI studies have found that individuals with BPD show exaggerated amygdala responses during passive viewing of emotional stimuli (Donegan et al., 2003; Herpertz et al., 2001; Koenigsberg, Siever, et al., 2009; Minzenberg, Fan, New, Tang, & Siever, 2007; Schnell, Dietrich, Schnitker, Daumann, & Herpertz, 2007) and atypical prefrontal recruitment during cognitive control tasks (New, Hazlett, et al., 2009; Silbersweig et al., 2007; Vollm et al., 2004; Wingenfeld et al., 2009).

At present, three studies have assessed the neural correlates of reappraisal in individuals with BPD. While these studies differed in terms of stimulus type (pictures vs. emotional scripts), reappraisal goal (increase vs. decrease), and reappraisal tactic (reinterpretation vs. distancing), taken together they suggest two basic patterns of results. First, individuals with BPD did not differ from healthy controls on behavioral measures of baseline emotional reactivity or reappraisal success (Koenigsberg, Fan, et al., 2009; S. Lang et al., 2011). The second, somewhat contradictory pattern, is that in comparison to healthy controls, during reappraisal individuals with BPD show both heightened amygdala responses and diminished recruitment of cortical regions involved in cognitive control, including dlPFC, vlPFC and, most consistently, dACC and anterior dmPFC (Koenigsberg, Fan, et al., 2009; S. Lang et al., 2011; Schulze et al., 2010). Prior work has suggested that high alexithymic traits, the tendency to have difficulty in recognizing and expressing emotions, exist in BPD (Berenbaum, 1996; Domes, Grabe, Czeschnek, Heinrichs, & Herpertz, 2011) and that high alexithymic traits are also associated with diminished activity in dACC and anterior and posterior dmPFC during mentalizing and emotional interference tasks (McRae, Reiman, Fort, Chen, & Lane, 2008; Moriguchi et al., 2007; Moriguchi et al., 2006). Taken together, these behavioral and imaging findings suggest that individuals with BPD are less adept than healthy controls at self-monitoring and making online evaluations of reappraisal success, and perhaps their own emotions more generally.

Major Depressive Disorder Major depressive disorder (MDD) is characterized by prolonged dysphoric mood as well as disrupted motivation, thought and behavior (Drevets & Todd, 1997). To date, a central focus of neuroimaging research on MDD has been evaluating whether MDD symptomology is caused by a bottom-up enhancement of responses to negative stimuli, diminished responses to positive stimuli, impaired top-down regulatory ability, or some combination of these three. In keeping with these ideas, fMRI work has suggested that individuals with MDD exhibit 1) atypical resting state activity in anterior dmPFC and ventral striatum (Kuhn & Gallinat, 2011), as well as 2) enhanced amygdala, and 3) diminished striatal responses to emotional stimuli (Delvecchio et al., 2012; J. P. Hamilton et al., 2012). Structural studies also are generally consistent, with two recent meta-analyses showing that individuals with MDD show diminished hippocampal, prefrontal and orbitofrontal volumes in comparison to healthy controls (Arnone, McIntosh, Ebmeier, Munafo, & Anderson, 2012; Kempton et al., 2011). Somewhat confusing, however, is a separate meta-analysis focusing

specifically on the amygdala, which found that amygdala volumes were enlarged for individuals with MDD on medication relative to healthy controls but diminished for un-medicated individuals with MDD relative to healthy controls (J. Hamilton, Siemer, & Gotlib, 2008). Together, these data raise questions about the relationships between structural volumes and functional responsivity that have yet to be resolved.

With this somewhat cloudy picture as a backdrop, to date five studies have examined the neural mechanisms of reappraisal in depression. While these studies examined different reappraisal tactics (reinterpretation vs. distancing), goals (increase vs. decrease emotion) and emotional valence (positive vs. negative), three general trends have emerged. First, individuals with MDD do not differ from healthy controls on behavioral measures of reappraisal success (Beauregard, Paquette, & Levesque, 2006; Erk, Mikschl, et al., 2010), although in one study individuals with MDD reported that it was more difficult to reappraise than did healthy controls (Beauregard et al., 2006). Second, regulation of subcortical responses to affective stimuli is impacted by MDD, with one study finding that individuals with MDD fail to sustain ventral striatal responses during upregulation of positive emotion (Heller et al., 2009), another finding that they show enhanced amygdala responses during downregulation of negative affect (Beauregard et al., 2006) and yet another finding that individuals with MDD do not show enduring reappraisal-related modulation of the amygdala (Erk, Mikschl, et al., 2010). Third, in three out of the four studies that compared neural responses in a reappraise>"respond naturally" contrast in individuals with MDD relative to healthy controls, depressed individuals recruited larger swaths of prefrontal cortex during reappraisal than did healthy controls. This suggests lesser efficiency during emotion regulation in individuals with MDD (Beauregard et al., 2006; Johnstone et al., 2007; Light et al., 2012). Fourth, all studies that examined prefrontal-subcortical dynamics found that functional connectivity between the PFC and the amygdala (Erk, Mikschl, et al., 2010; Johnstone et al., 2007) or ventral striatum (Heller et al., 2009) in individuals with MDD was either diminished or showed an opposite pattern of what was observed in healthy controls.

Phobias, Anxiety Disorders and PTSD State anxiety may be defined as a feeling of agitation or arousal caused by the perception of a real or imagined threat (Amstadter, 2008). In anxiety disorders (AD), specific (e.g., social anxiety disorder, SAD; phobias; and posttraumatic stress disorder, PTSD) or varied (e.g., generalized anxiety disorder, GAD) triggers chronically activate this anxious state (DSM-IV, 2005). Within the context of our model of emotion regulation, AD may represent an inability to accurately appraise what is threatening, an inability to reappraise threat, or both.

In support of the appraisal possibility, relatively greater activation of the insula and amygdala has been shown in response to negative or threatening social stimuli across different types of AD (Etkin & Wager, 2007). These relative hyperactivations have been observed in response to negative emotional facial expressions (Blair et al., 2008; Evans et al., 2008; Goldin, Manber, et al., 2009; Klumpp, Angstadt, & Phan, 2012; Labuschagne et al., 2010), during a speech preparation task in individuals with SAD (Lorberbaum et al., 2004), to trauma-themed pictures and scripts for individuals with

PTSD (Simmons & Matthews, 2012) and to phobogenic stimuli for individuals with spider phobia (Alpers et al., 2009; Caseras et al., 2010; Dilger et al., 2003; Goossens, Schruers, Peeters, Griez, & Sunaert, 2007; C. L. Larson et al., 2006; Lipka, Miltner, & Straube, 2011; Schienle, Schafer, Walter, Stark, & Vaitl, 2005; Schweckendiek et al., 2011; Straube, Mentzel, & Miltner, 2006; Wendt, Lotze, Weike, Hosten, & Hamm, 2008). It should be noted, however, that appraisal-related activity is somewhat different for PTSD than for other ADs in that most ADs are associated with hyperactivation of the insula while PTSD is not, nor is PTSD associated with hyperactivation of the entire amygdala, but rather only more ventral portions (Etkin & Wager, 2007).

GAD is unusual in its lack of specificity for what produces anxious feelings, and it is perhaps for this reason that some neuroimaging studies have found anticipation of or viewing of fear-related stimuli to elicit greater amygdala responses in GAD (Etkin & Schatzberg, 2011; McClure et al., 2007; Nitschke et al., 2009), while others have found no differences in amygdala responses between individuals with GAD and healthy controls (Palm, Elliott, McKie, Deakin, & Anderson, 2011), and yet others have observed hypoactive amygdala responses in GAD (Blair et al., 2008).

In summary, inappropriate threat appraisals in AD appear to be linked to abnormal activity in structures involved in perceiving, responding to and remembering fear-inducing stimuli, such as the amygdala and insula (Etkin & Wager, 2007).

In addition to the hyperactivations observed in targets of emotion regulation described above, a number of abnormalities associated with AD have been noted in prefrontal control regions. However, the specific pattern of atypical brain recruitment appears to vary across disorders. While most fMRI studies in AD samples report typical or enhanced recruitment of prefrontal regions, particularly dorsal ACC and anterior dmPFC, most fMRI studies of PTSD report diminished recruitment of both dorsal and ventral mPFC (Damsa, Kosel, & Moussally, 2009; Etkin & Wager, 2007).

In healthy adults, using strategies like “reality checking”, which involves using a type of emotional distancing (i.e., thinking about participating in a fMRI experiment on emotion from a more objective perspective), or attentional deployment strategies such as self-distraction to regulate state anxiety elicits activity in anterior and posterior dmPFC and dlPFC and reduces activity in the amygdala and insula (Herwig et al., 2007; Kalisch et al., 2005). To date, two fMRI studies have examined reappraisal in individuals with SAD (Goldin, Manber, et al., 2009; Goldin, Manber-Ball, et al., 2009), one study has examined reappraisal in spider phobia (Hermann et al., 2009), and one study has examined reappraisal in PTSD (New, Fan, et al., 2009). Across these studies of different subtypes of AD, three patterns have emerged. First, none of these studies observed behavioral differences between individuals with AD and healthy controls. Second, none of these studies found group differences with regards to amygdala modulation during reappraisal, although other types of effects have been reported. For example, one study found that spider phobics were less effective at downregulating insula activity to phobogenic stimuli than to generally aversive stimuli (Hermann et al., 2009) and another study found less functional connectivity between prefrontal control

regions and the amygdala during downregulation for individuals with SAD in comparison to healthy controls (Goldin, Manber-Ball, et al., 2009). Third, though the precise set of prefrontal regions associated with reappraisal varied from study to study, all four studies found evidence to suggest that individuals with AD recruit anterior and posterior dmPFC, dlPFC and vlPFC to a lesser degree than do healthy controls (Goldin, Manber, et al., 2009; Goldin, Manber-Ball, et al., 2009; New, Fan, et al., 2009) or they recruit these regions to a lesser degree when regulating responses to phobogenic stimuli than other types of emotional stimuli (Hermann et al., 2009).

Taken together, the translational work to date emphasizes the need to examine emotion regulation in development, aging and psychopathology using converging evidence from the behavioral and fMRI literatures. In doing so, we have already made great strides toward identifying how emotional reactivity and regulation abilities interact to predict emotional stability, yet much more work must still be done. While it is clear that breakdowns in prefrontal control systems and subcortical affective appraisal systems contribute to regulatory failures in the young, the elderly and the mentally ill, there is still a need to carefully and precisely characterize the nature of these breakdowns on a population-by-population basis. Additionally, when looking between those populations that *are* well-characterized, there is a need to understand why they differ. For example, why does more prefrontal recruitment result in diminished regulatory success in some cases (e.g., MDD) while less recruitment results in diminished regulatory success in other cases (e.g., BPD)? This is just one of many questions that have yet to be fully addressed by translational emotion regulation work.

Summary and Future Directions

The overarching goal of this paper has been to review and synthesize current functional imaging research on emotion regulation and to apply it to development, aging and psychopathology. In the first part of this chapter, we outlined a basic model of the processes and neural systems that support emotion generation and regulation. At its core, this model specifies how prefrontal, cingulate and parietal control systems modulate activity in affective appraisal regions as well as occipito-temporal regions involved in semantic and perceptual representations. Such dynamics may differ across regulatory contexts as a function of one's goal, tactic, and the nature of the stimuli and emotions being regulated (Ochsner et al., Submitted).

With this model in place, the second part of this chapter sought to translate the model to constrain and interpret findings on emotion regulation in developmental, aging and clinical populations. This approach is critical both for understanding the mechanisms underlying normal variability and for testing the boundaries of our basic model of emotion regulation.

While this chapter sought to clarify and synthesize our existing knowledge about basic and applied aspects of emotion regulation, it is important to note the limitations of our current knowledge. On the basic side, four questions stand out. First, more direct comparisons between paradigms that differ in terms of goals, tactics and stimulus types

are needed to clarify the basic mechanisms underlying reappraisal. Second, additional work is needed to determine what roles the brain systems supporting reappraisal play in related phenomena such as attentional deployment and situational selection/modification. Third, it will be essential to not only refine our understanding of the distinctions between different regulatory processes, but also to address new questions about how emotion regulation operates. For example, while it is critical that regulation strategies have immediate effects on emotional responses, it is equally important, if not more so, to determine what their long-term effects are. Fourth, assessing whether regulatory effects are enduring is significant for both everyday and clinical contexts where one might repeatedly re-encounter an emotionally evocative stimulus (e.g., having daily interactions with a difficult co-worker). At present, this issue has only been addressed in three studies. In one study, reappraisal diminished arousal-related ERP activity for up to 30 minutes (Macnamara, Ochsner, & Hajcak, 2011). In a second study, reappraisal diminished amygdala responses for up to 40 minutes in healthy adults, but not those with major depression (Erk, Mikschl, et al., 2010). In a third study, reappraisal differentially impacted amygdala responses as a function of successful recall one year later (Erk, von Kalckreuth, & Walter, 2010). However, reappraisal did not affect whether or not stimuli were remembered. Taken together, these studies suggest that reappraisal may result in long-term changes in the neural response to emotional stimuli, but more work is clearly needed to characterize the extent of this duration and the mediating psychological processes.

On the translational side, as discussed in this article, two major goals will prove important for guiding future research. The first is to understand from a neural and cognitive perspective how and why emotional behavior changes as we transition from childhood to adolescence, from adolescence to adulthood, and from adulthood to old age. Evaluating the neural bases of emotional development has the potential to enhance mental health and wellbeing in at least two ways. First, neuroimaging data may allow us to better parse emotional reactivity and regulation than self-report data alone. For example, when questionnaire or diary studies compare self-reports of emotion across individuals at different ages, it can be difficult to determine whether bottom-up or top-down processes drive age effects. By using converging evidence from both fMRI and behavioral data, however, we may elucidate what drives emotional changes across development (i.e., is activity in sources or targets of reappraisal more strongly linked to affective ratings?). This will be critical for achieving a second goal for this line of work, developing appropriate interventions for individuals at different points in the lifespan. For example, if vIPFC develops relatively late in adolescence, and deteriorates relatively early in old age, then the young and the old ought to be better at using reappraisal subtypes that rely less strongly on vIPFC, such as distancing, than those that rely more strongly on vIPFC, such as reinterpretation. If this is the case, it may be more effective to teach these populations distancing strategies in interventions than reinterpretation strategies.

A second important goal for translational research is to clarify how potential dysfunction in the mechanisms of emotion generation and regulation may underlie psychopathology. While the present review discussed this solely in the context of depression (Erk,

Mikschl, et al., 2010; Heller et al., 2009; Johnstone et al., 2007), borderline personality disorder (Koenigsberg et al., 2010; S. Lang et al., 2011; Schulze et al., 2010) and anxiety disorders (Goldin, Manber, et al., 2009), including phobia (Hermann et al., 2009) and posttraumatic stress disorder (S. Lang et al., 2011; New, Fan, et al., 2009), this line of work may also extend to other clinical populations as well as to addicted populations (Kober et al., 2010; Volkow et al., 2010). Examining the neural bases of emotion generation and regulation in psychopathology has at least two potential benefits. First, doing so may elucidate disorder-specific patterns of altered function in emotion generation and regulation systems. Second, neuroimaging of emotion regulation processes may be used before and after treatment regimes as a biomarker to predict and assess treatment response. While such studies are only beginning to emerge, they hold great promise for understanding why some individuals improve and others do not, as well as whether different treatments (e.g. pharmacological vs. cognitive behavioral therapy) have different mechanisms of action.

By integrating basic and translational perspectives on emotion regulation, we may be better suited to identify which individuals are at greatest risk for maladaptive health behaviors and emotional outcomes, at what ages this risk is greatest, and which regulatory mechanisms could be targeted in future interventions during particular points in the life course. While this remains a far-off goal, establishing basic and applied models of emotion regulation is a necessary stepping stone for ultimately achieving it.

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Figure captions

Figure 1. A model of the cognitive control of emotion. **A.** Diagram of the processing steps involved in generating an emotion and the ways in which cognitive control processes (blue box) might be used to regulate them. As described in the text, the effects of different emotion regulation strategies (the red arrows descending from the cognitive control processes box) can be understood in terms of the stages of of the emotion generation sequence that they impact. **B.** Neural systems involve in using cognitive strategies, such as reappraisal, to regulate emotion (left, blue boxes) and systems involved in generating those responses (left, pink boxes).

Figure 2. Plots of activation foci from the reappraisal studies described in the text and Table 1. Contrasts in which the goal was to increase emotion were excluded from these plots, as such contrasts combine both source and target activity. **A.** Plots of foci for contrasts identifying sources of reappraisal (e.g., reappraise>look). **B.** Plots of foci for contrasts identifying targets of reappraisal (e.g., look>reappraise). A mask was used to limit foci to the amygdala and striatum.

Figure 3. Three theoretical trajectories for emotional responsivity in development.