Amygdala–prefrontal coupling underlies individual differences in emotion regulation

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ABSTRACT

Despite growing evidence on the neural bases of emotion regulation, little is known about the mechanisms underlying individual differences in cognitive regulation of negative emotion, and few studies have used objective measures to quantify regulatory success. Using a trait-like psychophysiological measure of emotion regulation, corrugator electromyography, we obtained an objective index of the ability to cognitively reappraise negative emotion in 56 healthy men (Session 1), who returned 1.3 years later to perform the same regulation task using fMRI (Session 2). Results indicated that the corrugator measure of regulatory skill predicted amygdala–prefrontal functional connectivity. Individuals with greater ability to down-regulate negative emotion as indexed by corrugator at Session 1 showed not only greater amygdala attenuation but also greater inverse connectivity between the amygdala and several sectors of the prefrontal cortex while down-regulating negative emotion at Session 2. Our results demonstrate that individual differences in emotion regulation are stable over time and underscore the important role of amygdala–prefrontal coupling for successful regulation of negative emotion.

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Introduction

The ability to regulate emotion according to one’s goals is a critical skill for psychological well-being and resilience. Among various forms of regulation, cognitive regulation of emotion using reappraisal as a strategy has received much scientific attention (Ochsner and Gross, 2005). Reappraisal involves reinterpreting the meaning of an emotional event; for example, creating an alternative scenario or adopting a different attitude (Gross, 2002; Ochsner et al., 2004). It is the basis of cognitive therapy (Freuden et al., 2008), has been found to be more beneficial than suppressing emotions (Ochsner et al., 2002), can be instructed or trained (Jackson et al., 2000), and varies widely across individuals (Gross and John, 2003).

Over the past decade, neuroimaging studies of reappraisal have revealed converging evidence that reappraisal engages sectors of the prefrontal cortex (PFC) and subcortical structures such as the amygdala (for a meta-analysis, see Kalisch, 2009). While the amygdala detects the significance of potentially emotion-eliciting situations and generates biobehavioral adjustments associated with that emotion (Phelps and LeDoux, 2005), the PFC provides top-down control, such as inhibiting proponent responses, maintaining affective goals, and recruiting further resources (Miller and Cohen, 2001), that steers and potentially modifies activation in subcortical circuitry including the amygdala. Supporting the PFC’s descending influence on the amygdala to achieve regulatory goals, recent studies have demonstrated a reciprocal PFC–amygdala relationship during successful reappraisal of negative emotion (Banks et al., 2007; Johnstone et al., 2007; Ochsner et al., 2002; Urry et al., 2006; Wager et al., 2008). This suggests that individuals with greater regulatory ability would be better able to engage the PFC–amygdala circuit during emotion regulation.

Most previous research on reappraisal, however, has reported group-mean findings in brain regions that are commonly activated across individuals. This approach rests on the assumption that all individuals regulate emotions in a similar way, and treats individual variation as statistical noise (Kosslyn et al., 2002). In the domain of emotion, however, variation across individuals is the rule rather than the exception (Hamann and Canli, 2004), and such individual differences in the capacity to regulate negative emotion may determine vulnerability and resilience in the face of adversity (Davidson, 2004). However, systemic investigation of the neural bases of individual differences in emotion regulation skills has been sparse, partly due to methodological issues, such as small sample sizes. Moreover, the extant neuroimaging literature has relied on self-reported negative affect as an index of regulatory success (Ochsner et al., 2002; Wager et al., 2008), or used measures reflecting non-specific arousal or effort such as eye-blink startle (Eippert et al., 2007), pupil dilation (Johnstone et al., 2007; Urry et al., 2009), and skin conductance response (Delgado et al., 2008). Arousal-dependent measures cannot differentiate between negative and positive emotions, and whether increased arousal and effort result from regulation success or failure. The use of subjective self-report measures of regulatory success can also be problematic because of demand characteristics and other biases such as inaccurate recall that plague the validity of such measures (Davidson, 1992). Furthermore, these measures were collected concurrently with the scan, which may be susceptible to state-
dependent factors such as mood, fatigue, motivation, etc., and thus may not reflect stable, trait-like differences (Braver et al., 2010).

One of the most widely used and well-validated measures to objectively index negative emotion is facial electromyography (EMG) over frowning muscles (corrugator supercili); cEMG. Activity in this muscle region reflects valence-specific negative affect (Bradley et al., 2001a), and is increased by direct intracerebral stimulation of the human amygdala (Lanteaume et al., 2007). Furthermore, cEMG activity has been shown to be systematically modulated by regulation instructions (Jackson et al., 2000; Lee et al., 2009; Ray et al., 2010), such that cEMG magnitude increases and decreases in accordance with instructions to amplify or attenuate negative emotion, respectively. In addition, these cEMG measures of emotion regulation exhibit high test–retest reliability over a four-week interval (Lee et al., 2009), suggesting that this measure may index trait-like emotion regulation ability. Regulation ability as measured by cEMG also predicts long-term adjustment in everyday life (Bonanno et al., 2004). Taken together, cEMG appears to be an objective and reliable measure to index trait-like individual differences in emotion regulatory ability.

To date, there has been no fMRI study that used cEMG to index individual differences underlying successful regulation of negative emotion. Although previous studies have found amygdala–PFC interactions important for regulation success, the findings widely diverge on which areas of the PFC critically impact regulatory success—for example, ventrolateral PFC (Wager et al., 2008), ventromedial PFC (Johnstone et al., 2007; Urry et al., 2006), dorsolateral/medial PFC and orbitofrontal cortex (Banks et al., 2007), and anterior cingulate cortex (Ochsner et al., 2002). The direction of the relationship that these PFC regions have with the amygdala has also been inconsistent across studies. While some report an inverse amygdala–PFC relation during the down-regulation of negative emotion (Johnstone et al., 2007; Ochsner et al., 2002; Urry et al., 2006; Wager et al., 2008), others find a positive coupling associated with regulation success (Banks et al., 2007).

Thus, in the current study, we aimed to independently assess trait-like regulatory ability in a large sample using the objective measure of cEMG, and to directly examine its neural network using functional connectivity analysis during emotion regulation. To this end, we conducted two laboratory sessions of emotion regulation in which 56 participants reappraised negative emotion while recording cEMG (Session 1) and BOLD fMRI (Session 2; see Fig. 1). We predicted that in both sessions participants would demonstrate an ability to regulate emotions according to instructions as evidenced by changes in cEMG activity and amygdala BOLD signal. We focused on the amygdala as a downstream target region of regulatory efforts for its activity has consistently been found to covary with regulatory goal (e.g., Eippert et al., 2007; Lapate et al., 2012; Ochsner et al., 2004; Urry et al., 2006; van Reekum et al., 2001a), and is increased by direct intracerebral stimulation of the amygdala (Chapman and Chapman, 1987) and free of psychiatric/neurological disorders. Only men were included because they showed more stable emotion regulation over time (Lee et al., 2009), as well as to eliminate variability due to sex differences in psychophysiological (Bradley et al., 2001) and neural (McRae et al., 2008) responses in emotion regulation. All participants were paid for participation and provided informed consent for the study procedures approved by the University of Wisconsin–Madison Social and Behavioral Health Sciences Institutional Review Boards.

Stimuli

Pictures were chosen from the International Affective Picture System (Center for the Study of Emotion and Attention [CSEA-NIMH], 1999). Two sets of 84 negative pictures (set 1: valence, 2.97 ± 0.66; arousal, 5.30 ± 0.93; set 2: valence, 2.98 ± 0.69; arousal, 5.29 ± 0.91) and 42 neutral pictures (set 1: valence, 5.02 ± 0.36; arousal, 2.75 ± 0.57; set 2: valence, 5.04 ± 0.47; arousal, 2.81 ± 0.50) were matched on valence and arousal ratings (Lang et al., 1999) with no repetition, and were counterbalanced across session for each participant.

Material and methods

Participants

Fifty-six male undergraduates (19.93 ± 1.81 years) were recruited from the University of Wisconsin–Madison, who were right-handed (Chapman and Chapman, 1987) and free of psychiatric/neurological

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Fig. 1. Trial schematics of emotion regulation task. In Session 1, following 4 s of picture viewing, one of three auditory regulation instructions was given (“enhance”, “suppress”, “maintain”); Participants used reappraisal strategies to regulate their emotional response until they saw “Relax”. Throughout the trial, cEMG was continuously recorded. Approximately one-year later, an fMRI-variant of Session 1 was conducted with a new matched set of pictures in Session 2. Red bars indicate the regulation period.
Participants underwent two sessions of emotion regulation in response to standardized affective pictures, one in which cEMG was measured (Session 1) and one wherein BOLD responses were collected (Session 2). In the first session, EMG sensors were placed on the corrugator supercili muscle (Tassinary et al., 1989), and 6 negative and 4 neutral pictures were presented to familiarize participants with the protocol. During the experiment, 126 pictures (1-s fixation; 8-s/picture; 12-s intertrial interval) were presented in 6 blocks. Four seconds after picture onset, one of three auditory regulation instructions was presented: “enhance” (increase intensity of emotional response), “suppress” (decrease intensity of emotional response), or “maintain” (sustain initial intensity of emotional response) (see below for more detail). Participants were instructed to continue regulating their emotional response for 12 s until the word “Relax” appeared on the screen (Fig. 1). Negative pictures were paired with each of the 3 regulation cues, whereas neutral pictures were paired only with the maintain instruction. Pictures were quasi-randomly presented with the constraint that no more than 3 trials of the same valence or instruction occurred consecutively. Following an average interval of 15.2 months (range: 11–19 months) participants returned to complete the fMRI-variant emotion regulation task. Prior to the experiment, participants completed a simulation scan to become familiar with the scanning environment and to practice emotion regulation. Using a non-repeating matched picture set, 126 pictures (1-s fixation; 12-s/picture; 5.1–9.9-s intertrial interval) were presented in 4 scan runs. After 4 s of un instructed picture viewing, participants received one of three regulation instructions: “enhance”, “suppress” or “maintain”.3 Participants were instructed to continue regulating for 8 s until they saw “Relax” (Fig. 1). In addition, pupil dilation was concurrently measured as an index of cognitive demand to ascertain the paradigm validity (Siegle et al., 2008).

Participants used cognitive reappraisal strategies to increase or decrease negative emotion, such as imagining a different outcome of the situation depicted in the picture or varying their level of personal involvement in the scene. For example, in order to reduce negative emotion to a picture of a child in surgery, participants might imagine that the outcome of the surgery turned out to be successful. In order to amplify negative emotion to a picture depicting mourning, participants could imagine themselves in place of the individual deceased (e.g., Larson et al., 2006), so as to obtain a more accurate estimate of the “regulation effect” which was our main interest. The AUC estimates were manually normalized to Talairach space for better alignment of limbic structures, particularly the amygdala (Nacewicz et al., 2006), and spatially blurred with a 6 mm full-width at half-maximum Gaussian filter. Mean AUC estimates were extracted from Talairach-defined region-of-interest (ROI) in the bilateral amygdala and entered into a paired t-test to test the effect of valence (negative—maintain vs. neutral—maintain) and into a repeated measures ANOVA to test the effects of regulation (negative pictures: enhance, maintain, suppress). To quantify regulation success, difference scores were computed as enhance — maintain and suppress — maintain for both cEMG activity and amygdala ROI estimates. A higher number in enhance — maintain indicates a better ability to up-regulate, whereas a lower number in suppress — maintain indicates a better ability to down-regulate negative emotion. To examine whether the cEMG index of regulatory success predicted amygdala index of regulatory success 1.3 years later, Pearson correlations were computed between cEMG and amygdala difference scores. Finally, functional connectivity analysis was performed using psychophysiological interaction (PPI) method (Friston et al., 1997). Timeseries from Talairach-defined bilateral amygdala (the same ROI as above) was extracted as a physiological seed, and regulation contrast (supress > maintain) was used as a psychological context, in order to create the psychophysiological interaction term (PPI). This interaction term was entered into a voxelwise regression, with the covariates of raw amygdala timeseries, six motion parameters and a second-order polynomial, and all original regressors of each regulation condition, in order to account for variance explained by the PPI over and above main effects of regulation conditions or amygdala activity. The resulting PPI parameter estimates (z-transformed betas) denoted the strength of functional coupling between the amygdala and the remainder of the brain during suppress relative to maintain trials. To examine the extent to which individual differences in down-regulation ability predicted this connectivity, cEMG difference scores (suppress — maintain) were entered...
into a voxelwise regression as a predictor of the PPI map. All statistical maps were thresholded at $P<0.01$, and corrected for multiple comparisons using cluster-size thresholding ($k>80$) based on whole-brain Monte Carlo simulation.

Horizontal pupil diameter was continuously acquired (60 Hz) using a remote eye-tracking device (SensoMotoric Instruments, Teltow, Germany). Pupil data from 14 participants were not usable due to technical problems. Data were processed using algorithms written by Siegle et al. (2002, unpublished Matlab code) and modified in our laboratory. Blinks were eliminated, missing points were linearly interpolated, and signals were smoothed with a 5-sample rolling average. Trials were removed for $>50\%$ interpolation during the regulation period and corrected for outliers ($\pm 3$ SD). Data were aggregated into 0.5-s bins, baseline-corrected (0.5-s pre-instruction), and computed for the mean proportional change averaged across 8-s of the regulation period. Pupil values were analyzed using GLM to test for the regulation effects.

### Results

First, we verified that the intended negative emotion was elicited by the pictures. In Session 1, cEMG activity was greater for negative versus neutral pictures during the initial 4-s period prior to regulation instructions ($t_{55}=6.66, P=0.001$). In Session 2, we confirmed the presence of picture-induced negative emotion by showing that amygdala activation was greater for negative—maintain versus neutral—maintain trials ($t_{55}=2.41, P=0.02$).\(^4\)

Next, we examined the effects of cognitive regulation of negative emotion. In Session 1, replicating previous findings (Jackson et al., 2000; Lee et al., 2009; Ray et al., 2010), cEMG activity was modulated according to the regulation instructions (enhance $>$ maintain $>$ suppress; $F_{2,110}=51.54, P<0.001$, pair-wise $P<0.001$; Fig. 2A). In Session 2, consistent with prior reports (Eippert et al., 2007; Ochsner et al., 2004; Urry et al., 2006; van Reekum et al., 2007), amygdala activation was modulated by the regulation instructions (enhance $>$ maintain $>$ suppress; $F_{2,110}=10.63, P<0.001$, pair-wise $P_{	ext{Bonferroni}}<0.045$; Fig. 2B). We additionally confirmed that the intended effort was expended following regulation attempts as evidenced by pupil dilation (enhance $>$ suppress $>$ maintain; $F_{2,82}=51.75, P<0.001$, pair-wise $P<0.001$). Thus, in both sessions, participants as a group were able to regulate negative emotion as instructed.

Further, to quantify regulation success difference scores were computed (i.e., suppress $-$ maintain; enhance $-$ maintain) for both cEMG and amygdala. The ability to down-regulate negative emotion (i.e., suppress $-$ maintain) as measured by cEMG in Session 1 was predictive of the amygdala BOLD signal in Session 2 about 1.3 years later ($r=0.39, P=0.003$). The ability to up-regulate negative emotion (i.e., enhance $-$ maintain) was positively correlated across sessions but not statistically significant ($r=0.21, P=0.12$; Fig. 3).\(^5\)

Finally, to determine the extent to which individual differences in down-regulatory ability predicted amygdala–PFC connectivity, cEMG difference scores of suppress $-$ maintain were regressed voxel-wise on the functional connectivity of the amygdala during suppress versus maintain trials (Friston et al., 1997). Results suggested that individuals who showed a stronger ability to down-regulate negative emotion were more likely to show a stronger gPFC connectivity to the amygdala (Ray et al., 2010).

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4 The correlation with amygdala and cEMG activity was $r=.26, P=.05$, suggesting that the level of negative emotion elicited by the pictures, in the absence of active regulation, was positively related across sessions.

5 We found similar results using the statistical amygdala ROI. The ability to down-regulate negative emotion was still significant, $r=.33, P=.01$, and the ability to up-regulate negative emotion was not significant, $r=.18, P=.18$.\(^5\)

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**Fig. 2.** Effects of emotion regulation on (A) cEMG activity (Session 1) and (B) amygdala BOLD signal (Session 2). For both sessions, participants regulated their responses to negative pictures according to instructions. Error bars indicate the SEM difference. Inset figures illustrate the time series of cEMG and amygdala activity.

**Fig. 3.** Stability of emotion regulatory success across sessions. The ability to down-regulate negative emotion assessed using cEMG ($\mu V^2/Hz$) and amygdala activity (% signal change) was moderately correlated over the 1.3-year interval. However, the ability to up-regulate negative emotion was not significantly correlated.
with greater capacity for reducing negative emotion (as measured with cEMG) exhibited greater inverse functional coupling between the amygdala and several regions of the PFC including the pregenual anterior cingulate cortex (pgACC), orbitofrontal cortex (OFC), and dorsomedial/lateral PFC (dm/dlPFC) when down-regulating negative emotion (Fig. 4A; see Table 1 for the complete list of regions). Conversely, unsuccessful regulators showed more positive coupling between the amygdala and these PFC regions. Among these PFC regions, when examining the main effects of regulatory goal, OFC was not modulated by regulation instructions ($F_{(2,110)}=1.81, P=0.17$) whereas pgACC and dm/dlPFC showed significant regulation effects ($F_{(2,110)}>21.75, Ps<0.001$; enhance=suppress $Ps>0.01$, suppress>maintain $Ps<0.001$) (Fig. 4B).

**Discussion**

The present study adds to the growing literature on emotion regulation by having an independent assessment of an objective and trait-like index of emotion regulatory ability from a large number of individuals. To our knowledge, our study is the first to correlate individual differences in BOLD response and functional connectivity during emotion regulation with a cEMG measure of regulatory ability. Our data provide new evidence that the trait-like ability to regulate negative emotion is associated with modulation of the amygdala activity as well as with amygdala–PFC functional connectivity. Specifically, we found that individuals who were better able to down-

**Table 1**

<table>
<thead>
<tr>
<th>Brain region (Brodmann area)</th>
<th>Size (mm$^3$)</th>
<th>Max T</th>
<th>Location of max T</th>
</tr>
</thead>
<tbody>
<tr>
<td>Middle, superior, medial frontal gyrus (BA 8, 9, 6)</td>
<td>17784</td>
<td>5.06</td>
<td>−15 27 46</td>
</tr>
<tr>
<td>Caudate, thalamus</td>
<td>3304</td>
<td>4.64</td>
<td>−11 15 18</td>
</tr>
<tr>
<td>Culmen</td>
<td>2424</td>
<td>3.56</td>
<td>−27 −45 −28</td>
</tr>
<tr>
<td>Inferior, middle temporal gyrus (BA 20, 37)</td>
<td>2248</td>
<td>3.96</td>
<td>−53 −33 −18</td>
</tr>
<tr>
<td>Anterior cingulate, medial/superior frontal gyrus (BA 32, 9, 10)</td>
<td>1768</td>
<td>3.48</td>
<td>−17 49 24</td>
</tr>
<tr>
<td>Cuneus (BA 19, 18)</td>
<td>1600</td>
<td>4.30</td>
<td>−23 −81 24</td>
</tr>
<tr>
<td>Lentiform nucleus</td>
<td>1352</td>
<td>4.39</td>
<td>−23 −9 −8</td>
</tr>
<tr>
<td>Inferior semi-lunar lobule, cerebellar tonsil, pyramis</td>
<td>944</td>
<td>3.49</td>
<td>35 −67 −34</td>
</tr>
<tr>
<td>Inferior frontal gyrus (BA 47, 11)</td>
<td>688</td>
<td>3.37</td>
<td>27 31 −4</td>
</tr>
</tbody>
</table>

Note: Corrected cluster for multiple comparisons at $P<0.01$. Coordinates of the location of the cluster's maximum $T$ are in Talairach space.
regulate negative emotion as indexed by cEMG at Session 1 showed not only more attenuated amygdala signal but also greater inverse functional connectivity between the amygdala and specific areas of the PFC, notably pgACC, OFC and dm/dIPFC, while down-regulating negative emotion at Session 2. These PFC regions have previously been shown to exert regulatory influences on the amygdala—pgACC inhibits amygdala activity in the resolution of emotional conflicts (Egner et al., 2008; Etkin et al., 2006); OFC, through its extensive an-atomical connections (Ghashghaie and Barbas, 2002; Ongur and Price, 2000), modulates the amygdala in the reappraisal of contextual value (Dolan, 2007); and lateral and dorsal PFC regions have also been found to influence amygdala function, possibly mediated via the OFC/vmPFC, in reducing negative emotion (Johnstone et al., 2007; Ochsner et al., 2002; Urry et al., 2006; Wager et al., 2008). Accord-ingly, our results suggest that the individual variations in emotion regulation skills are reflected in this amygdala–PFC circuit, in which PFC regions have an inverse functional connectivity with the amygdala in promoting adaptive regulation of negative emotion.

While previous research has primarily focused on between-subject findings, our individual difference analyses using cEMG and functional connectivity revealed a new set of large prefrontal clusters that do not overlap with the previously-reported ventromedial/lateral areas tied to amygdala activation (Johnstone et al., 2007; Ochsner et al., 2002; Urry et al., 2006; Wager et al., 2008). It is also notable that our OFC region as identified by the individual difference connectivity analysis, unlike pgACC and dm/dIPFC regions, did not reveal statistically significant regulation effects by the group-mean analysis. This finding suggests that the OFC was recu-rried in individuals who were particularly successful in decreasing both amygdala and cEMG activities, and contrasts a more generic circuity of reappraisal comprising lateral and dorsomedial regions (Kalisch, 2009) with the OFC-like regions usually found in individual difference analyses (Banks et al., 2007; Johnstone et al., 2007; Urry et al., 2006; Wager et al., 2008). Our finding, however, was in the op-posite direction from the only published study conducting the same type of connectivity analysis during reappraisal (Banks et al., 2007). Although Banks et al. located similar prefrontal regions, such as sub- genual ACC, OFC, dmPFC, and dIPFC, better regulators showed the more positive amygdala–PFC connectivity. The reason for conflicting results can be in part attributable to the fact that Banks et al. used self-reported intensity of negative emotion on a restricted-range scale of 1–5 to index regulatory success in a small sample (N=14), whereas we used cEMG to capture a continuous and a much wider range of regulation ability in a large sample (N=56). Furthermore, Banks et al. did not include the self-reported index of regulatory suc-cess in their connectivity analysis, which may have biased the find-ings towards positive PFC–amygdala coupling. This discrepancy between the results showcases the impact of methodology in individ-ual differences research, and could be resolved in future work by di-rectly comparing the psychophysiological and self-report measures in the effectiveness and validity of representing trait-like regulatory ability.

Our results also showed that despite the long temporal interval between assessments individual differences in the capacity to volitionally down-regulate negative emotion were stable. Given the imperfect coherence among different emotional systems (Mauss et al., 2005), it is notable that this stability was observed across pe-ripheral and central output systems. Moreover, for a subset of the current participants (n=17), the ability to regulate emotion predicted the ability to regulate pain three years later (Lapate et al., 2012). These findings underscore the trait-like quality of individual differ-ences in emotion regulation and suggest that such individual differ-ences may be an important target for understanding normal variation in temperament (Thompson, 1994) and for determining risk for psychopathology (Davidson, 2004; Phillips et al., 2008; Taylor and Liberzon, 2007).

In contrast to our findings for down-regulation, we did not find a sta-ble association between cEMG and amygdala activation during the up-regulation of negative emotion. There are two plausible explanations: first, given prior finding that men show lower cEMG activity to negative pictures as compared to women (Bradley et al., 2001), our male-only sample might have a more limited range to increase cEMG activity above and beyond that already activated in response to negative stimuli, which subsequently constrained our ability to detect the significant cor-relation with amygdala activity. Indeed, our participants showed signific-antly less mean changes in cEMG activity when increasing (M=.20, SD=.22) as compared to decreasing negative emotion (M=.24, SD=.21), f59=3.93, P<.001. Second, differences in stimulus duration between sessions might have differentially affected our ability to detect the predicted correlation; for example, increasing negative emotion might have become easier in the fMRI session as participants had more time to elaborate on the negative pictures. In fact, previous work suggests that regulating emotion while the picture is on produce more pronounced effects of regulation as compared to regulating emotion be-yond the picture offset (Dichter et al., 2002; Jackson et al., 2000; Lee et al., 2009). It should also be noted that while relations with amygdala activity might not be present, there may well be associations with other regions such as those in the PFC for the ability to increase negative emotion.

Two limitations of the current study warrant future research. First, the causal influence of prefrontal regions on the amygdala, or vice versa, cannot be determined with the functional connectivity analy-sis. This is a shortcoming of all correlative neuroimaging research and more invasive techniques would be required to examine the causative nature of these relationships. Second, given prior evidence that men are less emotionally reactive in expressive measure to aver-se stimulation (e.g., Bradley et al., 2001) and based on our finding that men showed a truncation of range for increasing negative emo-tion, caution is warranted when generalizing our results to women. Given gender differences in the prevalence of affective disorders (Kessler et al., 2004), future research with adequate sample sizes of each gender would be required to systematically address this issue.

In sum, this study complements and extends the extant group-based research by adopting a rigorous individual-difference approach (Braver et al., 2010; Kosslyn et al., 2002) and integrating psychophysiology and neuroimaging (Davidson, 2003). Our data suggest that successful emotion regulators exhibit inverse functional connectivity between the amygdala and PFC during down-regulating negative emotion. Such connectivity patterns have been implicated in affective disorders (Phillips et al., 2008), and could be targeted for clinical assessment of reappraisal success or training. More broadly, our data underscore the importance of examining stable individual differences to provide fur-ther insights into the neural bases of emotion regulation. Future re-search should examine the extent to which regulatory ability is plastic and the extent to which interventions designed to reduce nega-tive emotion and promote well-being modulate amygdala–prefrontal circuitry.

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