Emotion regulation reduces loss aversion and decreases amygdala responses to losses

Peter Sokol-Hessner, 1 Colin F. Camerer, 2 and Elizabeth A. Phelps 3

1 Division of the Humanities and Social Sciences (HSS), California Institute of Technology, 2 Division of the Humanities and Social Sciences (HSS) and Computational and Neural Systems (CNS), California Institute of Technology, 1200 E. California Blvd., Pasadena, CA 91125, USA, and 3 Department of Psychology, New York University, 6 Washington Place, New York, NY 10003, USA

Emotion regulation strategies can alter behavioral and physiological responses to emotional stimuli and the neural correlates of those responses in regions such as the amygdala or striatum. The current study investigates the brain systems engaged when using an emotion regulation technique during financial decisions. In decision making, regulating emotion with reappraisal-focused strategies that encourage taking a different perspective has been shown to reduce loss aversion as observed both in choices and in the relative arousal responses to actual loss and gain outcomes. In the current study, we find using fMRI that behavioral loss aversion correlates with amygdala activity in response to losses relative to gains. Success in regulating loss aversion also correlates with the reduction in amygdala responses to losses but not to gains. Furthermore, across both decisions and outcomes, we find the reappraisal strategy increases baseline activity in dorsolateral and ventromedial prefrontal cortex and the striatum. The similarity of the neural circuitry observed to that seen in emotion regulation, despite divergent tasks, serves as further evidence for a role of emotion in decision making, and for the power of reappraisal to change assessments of value and thereby choices.

Keywords: emotion; reappraisal; emotion regulation; decision making; loss aversion

INTRODUCTION

Decision making is not dispassionate but is instead fundamentally supported by emotions (Loewenstein, 1996; Frank et al., 2009; Phelps, 2009). Evidence of this comes from two sources. First, decision-making studies, measuring components of emotion such as arousal, have shown that physiological responses correlate with information like imminent losses (Bechara et al., 1997), volatility (Lo and Repin, 2002), anger during interpersonal interactions (van’t Wout et al., 2006) and deception (Wang et al., 2010). The second source of evidence for the involvement of emotion in decision making comes from studies manipulating emotion during choices, demonstrating the causality of the relationship (Lerner et al., 2004; Winkelman et al., 2005; Harlé and Sanfey, 2007; Andrade and Ariely, 2009). The neural mechanisms of decision making also include regions like the amygdala and insula (Bechara et al., 1997; Gottfried et al., 2003; Paulus et al., 2003; Hsu et al., 2005; Shiv et al., 2005; Murray, 2007; Clark et al., 2008), traditionally associated with emotion and physiological responses (Morris et al., 1996; Whalen et al., 1998; LeDoux, 2000; Critchley et al., 2004). This connection between emotion and choices is further supported by studies of patients with damage to those same regions (Shiv et al., 2005; De Martino et al., 2010), providing compelling evidence that the mechanisms underlying emotions and choice overlap.

Emotions are also known to be actively generated in part by our thoughts or appraisals (Schacter and Singer, 1962), suggesting that by changing appraisals, we can change emotions. This concept led to a large body of research on regulating emotions with reappraisal (Gross, 1998), demonstrating its power to alter emotional responses (Ochsner et al., 2002; Eippert et al., 2007; Delgado et al., 2008a). Neurally, dorsolateral and ventromedial prefrontal cortex (DLPFC and VMPFC) are consistently identified in the regulation of emotion, and the amygdala and striatum in representing value and processing emotion (Ochsner and Gross, 2008; Hartley and Phelps, 2010).

The behavioral and physiological consequences of the reappraisal of decision making were demonstrated in a recent study (Sokol-Hessner et al., 2009). Participants showed greater skin conductance responses to losses compared to gains in a baseline condition, and this differential arousal correlated with behavioral estimates of loss aversion (the relative decision weight of losses to gains). In a second condition, participants reappraised choices from a broader perspective, reducing loss aversion behaviorally and eliminating over-arousal to losses relative to gains by decreasing loss responses.

Studies of the neural correlates of loss aversion have implicated both the striatum and the amygdala. One functional magnetic resonance imaging (fMRI) study found striatal activity at decision reflected the overall expected utility of the choice, including loss aversion (Tom et al., 2007). However, others linked amygdala activity to the endowment effect (Weber et al., 2007), often interpreted as a behavioral consequence of loss aversion (Tversky and Kahneman, 1991). Similarly, patients with amygdala damage show reduced loss aversion (De Martino et al., 2010). Though these latter findings linking the amygdala to loss aversion conflict with the quantitative fMRI exploration of loss aversion with functional imaging, we note that Tom et al. (2007) had only decisions and no outcomes. Consequently, one possible model is that the amygdala may mediate loss aversion in responses to outcomes, accounting for Weber et al. (2007) and De Martino et al. (2010), and that the weights in those outcome responses may be passed on to the striatum, leading it to represent loss aversion at the time of decision as in Tom et al. (2007). As reappraisal modulates loss aversion behaviorally, we would therefore expect to see changes in the striatum and amygdala as a result of regulation.

In the present study, we ask whether the neural mechanisms underlying reappraisal in decision making are similar to those known to support intentional emotion regulation. Such similarity would suggest a central role for emotions and their regulation in decision making. It would also close the distance between emotions and valuation, and...
emotion regulation and reappraisal in choice, arguing that they are simply instantiations of the same processes and mechanisms in different contexts.

In a within-subjects design, participants were scanned while making risky monetary choices in each of two conditions. In the ‘Attend’ condition, participants considered each choice on its own merit, as if it was the only choice they were making. In the ‘Regulate’ condition, participants took a broader perspective and considered each choice in its greater context, as one of many. We estimated participants’ loss aversion, risk attitudes and consistency over choices in each condition. Analyses of the EMRI data focused on correlates of loss aversion and its reduction, as well as the application of the ‘Regulate’ intentional strategy.

**EXPERIMENTAL PROCEDURES**

**Participants**

Sixty-three participants (34 female, mean age 19.8 ± 3.1 years) took part in the study. Of those, nine were removed for excess motion, and seven were excluded for other reasons (See ‘Detailed Participant Exclusions’ in Supplementary Data for more details). The remaining 47 participants (27 female, mean age 20.1 ± 1.7 years) were entered into the decision analysis. For analysis of outcomes, seven participants were excluded for not having enough outcome trials [exclusion criterion: fewer than 10 trials in 2 or more outcome categories (e.g. ‘Attend’ win, ‘Regulate’ loss)]. The outcomes analyses include the remaining 40 participants (24 female, mean age 20.2 ± 1.7 years). One participant was excluded from the loss aversion coefficient (represented by \( \lambda \)) correlational analyses for having an ‘Attend’ \( \log(\lambda) \) value greater than three standard deviations above the mean. All correlations with \( \lambda \) were performed on the remaining 39 participants. All participants gave informed consent as approved by the University Committee on Activities Involving Human Subjects at New York University, and in accordance with the Declaration of Helsinki.

**Behavioral procedure**

Participants were thoroughly instructed in all details and contingencies of the task, successfully completed a brief quiz assessing comprehension of the instructions, practiced use of the strategies with the experimenter and did a practice block of trials before scanning.

Immediately after giving informed consent, participants were endowed with $30, told the money was theirs to risk during the study, and asked to place it in their wallets or purses. After the task, 10% of all the trials in which feedback was presented (30 out of 300 trials) were randomly selected. Participants were paid the $30 plus the sum of their actual outcomes in those trials. Participants could lose up to the entire $30 endowment, and could gain up to a theoretical maximum of an additional $662. All participants also received a $75 ($25/h) subject fee upon completion of the study.

Participants completed two identical sets of choices (Figure 1) with different intentional cognitive strategies. For the ‘Attend’ strategy, participants were asked to consider each choice in isolation from any context, as if it was the only choice in the entire study. For the ‘Regulate’ strategy, participants were asked to consider each choice in a greater context, remembering that each choice was one of many or part of a portfolio. The conceptual nature of these strategies was emphasized by asking participants not to keep a running tally of their previous outcomes or overall earnings. Similar to typical studies of emotion regulation (e.g. Ochsner et al., 2002), detailed strategy instructions were read aloud, and participants were given a chance to ask questions about the strategies. They were then encouraged to repeat the strategies back to the experimenter in their own words. After a brief set of practice trials completed with the experimenter, participants completed a longer set of practice trials alone, after which they could again ask questions about the strategies. The full text of the strategies is in the Supplementary Data.

The choices presented to participants were identical for the two instructed strategies, but the win/loss outcomes varied randomly across trials. Each ‘set’ of 150 choices was designed to allow the dissociation of several aspects of behavior (see below, ‘Behavioral Model’ section). Out of the 150 choices, 120 were between a mixed gamble (positive and negative possible outcomes) and a guaranteed amount of zero, and 30 were between gain-only gambles (positive and zero possible outcomes) and positive guaranteed amounts (see Supplementary Data for exact monetary amounts). Participants had to either accept the gamble, in which case they won or lost with equal probability, or reject it for the guaranteed amount. Each decision was immediately followed by its outcome, prior to the next trial beginning. See Figure 1 for trial details and timing. In addition to the 300 ‘full’ trials (consisting of choice, inter-stimulus interval (ISI), outcome, and inter-trial interval (ITI); 150 in each ‘Attend’ and ‘Regulate’), there were 64 partial trials (consisting only of choice, partial trial indicator and ITI; 32 in each ‘Attend’ and ‘Regulate’) to allow accurate separation of decision and outcome blood oxygenation level-dependent (BOLD) activity (Ollinger et al., 2001a, 2001b). The partial trial indicator following the
response period consisted of 1s of normal (white) fixation and 1s of a red fixation cross.

Each block was completed using one of the two strategies (14 blocks each for ‘Attend’ and ‘Regulate’, for 28 blocks total; 4 blocks per functional run). Blocks were preceded by a cue (4s; followed by 2s fixation) indicating which strategy to use for the following trials. There were no gaps between blocks, other than the ITI following the last trial of the block, after which the cue for the next block was displayed. Blocks were pseudorandomly ordered such that no strategy occurred more than four times in a row at any point (including across runs). Participants completed one of four counterbalanced task orders which were independently randomized along the following dimensions: order of condition blocks (‘Attend’, ‘Regulate’), gamble outcomes (‘win’, ‘lose’), and gamble order within each condition block.

**Behavioral model**

Choice behavior was estimated with a prospect theory-inspired (Kahneman and Tversky, 1979) three parameter model (Sokol-Hessner et al., 2009). Gains and losses were modeled with Equations (1) and (2), respectively. The resulting subjective utility estimates were used in the softmax function in Equation (3), translating the subjective difference between the gamble and the guaranteed amount into a probability of gamble acceptance.

\[ u(x^+) = x^\lambda \]  
\[ u(x^-) = -\lambda \times (-x)^\lambda \]  
\[ p(\text{gamble acceptance}) = \frac{1}{1 + \exp[-\mu (u(\text{gamble}) - u(\text{guaranteed}))]} \]  

Lambda (\( \lambda \), the loss aversion coefficient) appears only in the utility function for losses [Equation (2)]. It represents the multiplicative weighting of the subjective value of losses relative to gains. When \( \lambda > 1 \), losses are overvalued relative to gains of the same size (‘loss averse’). When \( \lambda = 1 \), gains and losses are valued equally (‘gain-loss neutral’). When \( \lambda < 1 \), gains are overvalued relative to losses (‘gain-seeking’). Rho (\( \rho \), the curvature of the function) represents risk attitudes and diminishing sensitivity to changes in value as the absolute value increases. Mu (\( \mu \), the logit sensitivity) represents the consistency of participants’ decisions across multiple choices.

The model was estimated for each individual participant separately in the ‘Attend’ and ‘Regulate’ conditions with a Nelder–Mead simplex maximum likelihood procedure in Mathematica (Wolfram Research, Champaign, IL, USA). Overall model significance for each participant in each condition was evaluated against a random-choice model with a likelihood ratio test, determining whether the probability of the data observed was significantly higher given the estimated parameters. Within-subject changes in parameters were similarly evaluated with likelihood ratio tests of the full model (containing all ‘Attend’ and ‘Regulate’ parameters) against reduced models which restricted parameter values to be identical in ‘Attend’ and ‘Regulate’.

**Scanning parameters**

Scanning was performed at NYU’s Center for Brain Imaging with a 3T Siemens Allegra head-only scanner and a Nova Medical head coil (model NM011). High-resolution anatomical images were acquired using a T1-weighted protocol (Field of view (FOV) = 256, 176 slices, 1 × 1 × 1 mm). Functional imaging used a single-shot gradient echo echo-planar imaging (EPI) sequence (TR = 2000 ms, TE = 30 ms, FOV = 192, flip angle = 90°), acquiring 7 functional runs of 421 volumes, each with 36 contiguous 3 mm isovoxel oblique axial slices aligned parallel to the AC–PC plane. Data were preprocessed with BrainVoyager software (Brain Innovation, Maastricht, The Netherlands), including motion correction, slice-time correction and spatial smoothing (4 mm FWHM Gaussian kernel). Functional runs were coregistered to the high-resolution anatomical after which all scans were transformed to Talairach space (Talairach and Tournoux, 1988). Data used in general linear models (GLMs) were temporally high-pass filtered (period = 128 s) with SPM8’s filtering tool (Statistical Parametric Mapping 8, http://www.fil.ion.ucl.ac.uk/spm/) prior to analysis. Trial-triggered averages were taken from data that had not been temporally high-pass filtered. Analysis was performed with BrainVoyager and custom Matlab scripts (Mathworks, Natick, MA, USA).

GLMs were estimated on a fixed-effects level for each participant. The results were then subjected to random effects group-level contrasts in order to functionally define regions of interest (ROIs) for use in other analyses.

Analyses of decisions included all 47 participants. The GLM used for these analyses included, separately for ‘Attend’ and ‘Regulate’, a main effect (indicator) regressor for mixed valence decisions and a parametric regressor with the subjective expected utility of the decision (calculated using the participant’s estimated value functions) at the time of gamble presentation in the mixed-valence trials. For completeness, the GLM also included main effect regressors (separately for ‘Attend’ and ‘Regulate’) for mixed-valence win outcomes, mixed-valence loss outcomes, mixed-valence guaranteed outcomes, gain-only decisions, gain-only outcomes and block cues.

Analyses of outcomes were performed with the 40 participants with enough trials to support estimation (see Experimental Procedures: Participants). In order to eliminate statistical issues associated with having few trials of a given type in a single functional run, participants’ functional runs were temporally concatenated to form one long run. The GLM used for these analyses included, separately for ‘Attend’ and ‘Regulate’, a main effect (indicator) regressor for mixed-valence win outcomes, another for mixed-valence loss outcomes and parametric regressors with the win amount in dollars, and similarly with the loss amount. For completeness, the GLM also included main effect regressors (separately for ‘Attend’ and ‘Regulate’) for mixed valence decisions, mixed-valence guaranteed outcomes, gain-only decisions, gain-only outcomes and block cues. Additional parametric regressors included the subjective expected utility of the decision (calculated using the participant’s estimated value functions) at the time of gamble presentation, and the prediction error associated with an outcome at the time of outcome presentation, calculated as the difference between the subjective utility of the outcome and the subjective expected utility of the gamble.

All parametric outcome predictors were orthogonalized against the relevant main effect regressors, and the prediction error regressors were subsequently orthogonalized against the main effect and parametric regressors.

In all GLMs, nuisance predictors included indicators for the decision and outcome periods when participants failed to respond in time, the occurrence of partial trial indicators, and motion estimates (six total: translation and rotation in each of three orthogonal planes). The outcome analyses (performed on the concatenated data) also included a constant factor for each functional run. All predictors except the motion estimates and run constants were convolved with the BrainVoyager hemodynamic response function (HRF).

The blocked nature of our task made it possible for two different patterns to be observed in BOLD activity as a result of the perspective-taking regulation strategy. First, it is possible that transient responses to events (e.g. loss outcomes) were altered by regulation,
such that the observed \( \beta \)-values from a general linear model would be different between conditions ('Attend' or 'Regulate'). However, because the conditions occurred in blocks, it is additionally possible that there were longer lasting changes consistent with this perspective shift, independent of individual trial events (e.g. Donaldson et al., 2001; Visscher et al., 2003). That is, regulation in this setting could include not just altering an event-specific response, but also maintaining a perspective across any particular type of event, manifesting in a baseline shift in BOLD activity (in contrast to the transient responses to events). To get at any potential baseline shifts across blocks, we used the technique of trial-triggered averaging (e.g. Buckner et al., 1996), which is not constrained by assumptions about the shape of the hemodynamic response. Beginning with the timepoint at the start of the event of interest (e.g. decision), we calculated individuals’ average BOLD signal at each timepoint during that event, and then combined across individuals to produce a group-level average of the mean BOLD signal at each timepoint. We then compared these average BOLD signal time courses across conditions ('Attend' or 'Regulate'). The data used for this analysis was non-high-pass filtered BOLD activity extracted from ROIs.

RESULTS

Behavioral results

Estimates of the behavioral model parameters across 47 participants were consistent with previous results using this choice paradigm (Sokol-Hessner et al., 2009), and in many other paradigms including naturally occurring field data (Camerer et al., 1997; Genesove and Mayer, 2001; Haigh and List, 2005). Mean ‘Attend’ parameter values (with standard errors of the mean) were \( \lambda = 1.62 \) (0.14; loss aversion), \( \rho = 0.88 \) (0.04; risk attitudes), and \( \mu = 3.26 \) (0.61; consistency over choices). Since the distribution of the loss aversion coefficient \( \lambda \) across people is typically right-skewed, taking its log is more conducive to standard statistical testing. The mean log(\( \lambda \)) value in the ‘Attend’ condition was 0.34 (0.08), corresponding to \( \lambda = 1.41 \). In the ‘Regulate’ condition, mean parameter values were \( \lambda = 1.33 \) (0.11), \( \rho = 0.91 \) (0.04) and \( \mu = 2.93 \) (0.38). The mean log(\( \lambda \)) value was 0.17 (0.07), corresponding to \( \lambda = 1.19 \).

To compare estimates between ‘Attend’ and ‘Regulate’ at the group level, we performed two-tailed paired \( t \)-tests of participants’ parameter estimates in ‘Attend’ vs those in ‘Regulate’. These tests indicated a significant difference for \( \lambda \) (loss aversion; \( t(46) = 4.52, P < 5 \times 10^{-5} \)), using log(\( \lambda \)); \( t(46) = 4.94, P < 2 \times 10^{-5} \)), but no difference in either risk-aversion \( \mu \); \( t(46) = 0.90, P < 0.38 \) or consistency \( \rho \); \( t(46) = 0.77, P < 0.45 \). Thus, there is a consistent, selective effect of the ‘Regulate’ strategy in decreasing loss aversion from ‘Attend’ to ‘Regulate’ \( (\lambda_{\text{Attend}} - \lambda_{\text{Regulate}}) \) by an average of 14% (Figure 2).

We found that 37 individuals (out of 47 participants; 79%) showed some decrease in \( \lambda \) from ‘Attend’ to ‘Regulate’, comparable to previous findings (86% in Sokol-Hessner et al., 2009). Because of the unique, quantitative nature of our task, we were able to characterize individual differences in the strength of that effect. We examined individuals’ behavior for significant differences in value parameters between ‘Attend’ and ‘Regulate’ using likelihood ratio tests (LRTs; see Supplementary Data for more details). Out of 47 participants, 16 had significant shifts in \( \lambda \) (all ‘Regulate’ < ‘Attend’) at a threshold of \( P < 0.05 \). We identified these participants as ‘Regulators’. The remaining 31 participants who did not show a significant decrease in loss aversion we identified as ‘Non-Regulators’. LRTs performed on the other parameters found 8 out of 47 significant shifts in \( \rho \) (two ‘Regulate’ < ‘Attend’), and 9 out of 47 in \( \mu \) (6 ‘Regulate’ < ‘Attend’).

Since these differences were neither large nor systematic, the remainder of the analysis focuses on \( \lambda \), the loss aversion coefficient.

LRTs assessing the overall significance of the estimated models in both ‘Attend’ and ‘Regulate’ were overwhelmingly significant (all \( P s < 10^{-5} \)). We also calculated the mean predicted likelihood of participants’ choices given the estimated parameters. Across participants, the average predicted likelihood of a choice given the estimated was 0.75 (s.e. = 0.01) in ‘Attend’ and 0.73 (s.e. = 0.01) in ‘Regulate’, demonstrating that choices were predicted imperfectly, but much better than chance (See Supplementary Data for more details).

Imaging results

We performed two types of analysis on the fMRI data. First, we examined event-specific, transient responses by analyzing parameter estimates (\( \beta \)‘s) of activity across the brain. Second, because of the
Reappraisal and amygdala loss responses

Correlates of loss aversion at decision and outcome

In our analysis of $\beta$'s, we looked for correlates of loss aversion separately at decision and outcome. A previous study of loss aversion examined activity at the time of decision and found that activity in the ventral striatum and other regions reflected the overall expected utility (including the estimated degree of loss aversion) of the potential losses and gains being considered (Tom et al., 2007). Since gain and loss values were presented simultaneously at the time of decision and were correlated (see Methods in Supplementary Data), it was not possible to independently analyze the BOLD response to losses or to gains at decision. Instead, we calculated the expected utility of the mixed valence gambles in the baseline ‘Attend’ condition using participants’ individually estimated value functions, including their unique $\lambda$ estimate. This parametric regressor was then entered into a whole-brain analysis, identifying regions of the brain whose activity correlated with the expected utility (including loss aversion) of the gamble being considered. Among the regions whose activity at decision correlated with this regressor was the bilateral striatum (Table 1; see also Supplementary Data), consistent with previous results suggesting a unified representation in that region of expected utility at decision (e.g. Hsu et al., 2005; Preuschoff et al., 2006).

In analyzing outcome-related activity for correlates of behavioral loss aversion, we were able to separately estimate the BOLD responses to loss outcomes and gain outcomes (since only one outcome occurred at any given time), and then do second-level correlations and contrasts with those estimates. We performed a correlation of the log of individuals’ $\lambda$ values (loss aversion) in ‘Attend’ with the voxelwise contrast of ‘Attend’ Loss > ‘Attend’ Win across the entire brain (both main effect, or binary predictors). This analysis revealed a region of the left amygdala as the sole neural correlate at outcome of behavioral loss aversion under these conditions (Figure 3A; Figure 3B is a replotting of the correlation for illustrative purposes only; see Table 1 for whole-brain correlation details).

Previous findings related behavioral loss aversion to physiological arousal responses to loss vs gain outcomes in a parallel fashion (Sokol-Hessner et al., 2009). Since the amygdala is known to mediate arousal responses across a variety of contexts (Phelps et al., 1998; Garavan et al., 2001; Williams et al., 2001; Glauscher and Adolphs, 2003; McGaugh, 2004), these findings serve as further evidence that the aforementioned arousal-loss aversion relationship may be amygdala mediated. A similar whole-brain correlation of the behavioral loss

Table 1 Contrast and correlation activations

<table>
<thead>
<tr>
<th>Brain region (± or -- is the contrast value sign)</th>
<th>Brodmann’s area (BA)</th>
<th>Talairach coordinates (x, y, z)</th>
<th>Number of voxels (1 mm³)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Loss aversion at decision: 'Attend' decision subjective expected value &gt; + (n = 47); P &lt; 0.001 (unc); cluster threshold = 81 mm³;</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>-- Right striatum</td>
<td>8, 5, 6</td>
<td>567</td>
<td></td>
</tr>
<tr>
<td>-- Left striatum</td>
<td>-8, 4, 5</td>
<td>131</td>
<td></td>
</tr>
<tr>
<td>-- Lingual gyrus</td>
<td>19</td>
<td>478</td>
<td></td>
</tr>
<tr>
<td>-- Lingual gyrus</td>
<td>-13, -69, -5</td>
<td>2671</td>
<td></td>
</tr>
<tr>
<td>-- Midbrain</td>
<td>-8, -24, -6</td>
<td>82</td>
<td></td>
</tr>
<tr>
<td>-- Middle frontal gyrus</td>
<td>46</td>
<td>98</td>
<td></td>
</tr>
<tr>
<td>-- Precentral gyrus</td>
<td>-46, -1, 32</td>
<td>85</td>
<td></td>
</tr>
<tr>
<td>Loss aversion at outcome ('Attend'): Correlation: 'Attend' $\lambda$ with (Attend Lose &gt; Win) (n = 39); P &lt; 0.005 (unc); cluster threshold = 81 mm³;</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>-- Left amygdala</td>
<td>-22, 2, -19</td>
<td>163</td>
<td></td>
</tr>
<tr>
<td>Loss aversion at outcome ('Regulate'): Correlation: 'Regulate' $\lambda$ with (Regulate Lose &gt; Win) (n = 39); P &lt; 0.005 (unc); cluster threshold = 81 mm³;</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>-- Inferior frontal gyrus</td>
<td>38, 27, 14</td>
<td>109</td>
<td></td>
</tr>
<tr>
<td>-- Para hippocampal gyrus</td>
<td>34, -15, -23</td>
<td>430</td>
<td></td>
</tr>
<tr>
<td>-- Cerebellum</td>
<td>38, -39, -25</td>
<td>99</td>
<td></td>
</tr>
<tr>
<td>-- Corona</td>
<td>18</td>
<td>255</td>
<td></td>
</tr>
<tr>
<td>-- Inferior parietal lobe</td>
<td>-64, -33, 20</td>
<td>132</td>
<td></td>
</tr>
<tr>
<td>Regions related to application of the regulation strategy:</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Attend Win &gt; Attend Lose (n = 40); P &lt; 0.001 (unc); cluster threshold = 81 mm³;</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>-- Ventromedial prefrontal cortex</td>
<td>-8, 12, 3</td>
<td>213</td>
<td></td>
</tr>
<tr>
<td>-- Right Caudate head</td>
<td>8, 4, 1</td>
<td>924</td>
<td></td>
</tr>
<tr>
<td>-- Left Caudate head</td>
<td>-12, 2, -2</td>
<td>897</td>
<td></td>
</tr>
<tr>
<td>-- Anterior cingulate</td>
<td>15, 29, 11</td>
<td>615</td>
<td></td>
</tr>
<tr>
<td>-- Medial frontal gyrus</td>
<td>-6, 51, 11</td>
<td>342</td>
<td></td>
</tr>
<tr>
<td>-- Supplementary motor area</td>
<td>-8, -26, 54</td>
<td>182</td>
<td></td>
</tr>
<tr>
<td>-- Right parietal lobe</td>
<td>29, -26, 33</td>
<td>828</td>
<td></td>
</tr>
<tr>
<td>-- Right parietal lobe</td>
<td>30, -28, 31</td>
<td>113</td>
<td></td>
</tr>
<tr>
<td>-- Right frontal lobe</td>
<td>23, -1, 28</td>
<td>340</td>
<td></td>
</tr>
<tr>
<td>-- Left frontal lobe</td>
<td>-28, -12, 28</td>
<td>178</td>
<td></td>
</tr>
<tr>
<td>-- Thalamus</td>
<td>-6, -33, 16</td>
<td>105</td>
<td></td>
</tr>
<tr>
<td>Regions related to application of the regulation strategy: Regulate decision ME &gt; Attend decision ME (n = 16); P &lt; 0.005 (unc); cluster threshold = 81 mm³;</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>-- Right dorsolateral prefrontal cortex</td>
<td>9</td>
<td>31, 37, 30</td>
<td>92</td>
</tr>
<tr>
<td>-- Supplementary motor area</td>
<td>6</td>
<td>6, -12, 59</td>
<td>196</td>
</tr>
<tr>
<td>-- Motor cortex</td>
<td>4</td>
<td>-3, -35, 59</td>
<td>86</td>
</tr>
<tr>
<td>-- Right thalamus</td>
<td>6, -22, 7</td>
<td>99</td>
<td></td>
</tr>
</tbody>
</table>

The blocked nature of the reappraisal task, we thought there might be baseline shifts in activity as a function of condition (i.e. ‘Attend’ or ‘Regulate’), in addition to any event-specific effects. In order to investigate this possibility, we analyzed trial-triggered averages of fMRI BOLD signal to look for baseline shifts in activity across decision and outcome. See Table 1 for detailed contrast results.
The voxelwise correlation between individuals’ estimated $\beta_{\text{ATTEND/WIN}}$ and the contrast for ‘Attend’ minus that in ‘Attend’ for either wins (greens) or losses (reds). Positive numbers indicate increased activity in ‘Regulate’, negative numbers indicate reduced activity in ‘Regulate’. Asterisks indicate significantly different from zero at $P<0.05$. "T" indicates a significant group difference (one-tailed two-sample unequal variance $t$-test, $P=0.05$).

Using the first, group-based approach, we looked at changes in activity to losses and to gains for the ‘Regulators’. Similar to our previous study’s arousal results, the ‘Regulator’ participants showed a strong reduction in their amygdala responses to losses [mean ‘Regulate’ Loss $\beta$ − ‘Attend’ Loss $\beta=-0.26$; $t(13)=2.21$, $P<0.05$], while ‘Non-Regulators’ showed no such change [mean ‘Regulate’ Loss $\beta$ − ‘Attend’ Loss $\beta=-0.01$; $t(24)=0.08$, $P>0.95$], a difference that was significant between groups (one-tailed two-sample unequal variance $t$-test; $t(36.25)=1.67$, $P=0.05$). In the case of win outcomes, both ‘Non-Regulators’ and ‘Regulators’ showed similarly sized increases in their left amygdala responses (‘Regulators’ mean ‘Regulate’ Win $\beta$ − ‘Attend’ Win $\beta=0.35$; $t(13)=1.62$, $P>0.13$; ‘Non-Regulators’ mean ‘Regulate’ Win $\beta$ − ‘Attend’ Win $\beta=0.34$; $t(24)=2.90$, $P<0.008$) (Figure 4).

Taking the second, correlational approach to examining the relationship between regulation success and outcome processing, we performed correlations across all participants (both ‘Regulators’ and ‘Non-Regulators’) of individuals’ percent reduction in $\lambda$ (loss aversion) with the reduction in left amygdala responses to loss outcomes, and to win outcomes. Regulation success was marginally significantly correlated in the left amygdala ROI with reduction in BOLD responses to loss outcomes (Percent reduction in $\lambda$ correlated with ‘Attend’ Loss $\beta$ − ‘Regulate’ Loss $\beta$; $r(37)=0.26$, $P<0.08$), but was not correlated with changes in responses to gain outcomes (Percent reduction in $\lambda$, correlated with ‘Attend’ Win $\beta$ − ‘Regulate’ Win$\beta$; $r(37)=-0.11$, $P<0.49$), though the difference between the Loss $\beta$ and Gain $\beta$ correlations was not significant (Fisher’s r-to-z transformation, $z=1.6$, $P=0.11$).

The findings from both group-level and correlational approaches implicate the regulation of loss outcome responses in the successful attenuation of behavioral loss aversion, directly echoing previous results with physiological arousal (Sokol-Hessner et al., 2009).

**Baseline effects of regulation across decision and outcome**

Two final sets of contrasts were used to examine responses in regions of the brain related to regulation and choice behavior, in keeping with...
the expectation of regulation-related effects at decision and changes in the processing of outcomes as a consequence of regulation. The regions of interest were defined as follows. First, the contrast of the main effect of a ‘Regulate’ decision is that of an ‘Attend’ decision (‘Regulate’ Decision > ‘Attend’ Decision) was performed on the ‘Regulators’, identifying a region of the right DLPFC related to the application of the regulation strategy. The DLPFC has been repeatedly implicated in studies of emotion regulation (Ochsner et al., 2002, 2004; Banks et al., 2007; Eippert et al., 2007; Delgado et al., 2008a, 2008b; Ochsner and Gross, 2008; Hartley and Phelps, 2010). Second, areas related to value computation, including the bilateral striatum and VMPFC, were identified by contrasting estimates of BOLD activity to gain outcomes with that to loss outcomes (‘Attend’ Win > ‘Attend’ Lose; see Table 1 for full list of regions identified in both contrasts).

Since the regulation task was blocked (choices were in sets of 25; see Experimental Procedures section), we were able to examine whether any changes might have occurred in baseline levels of BOLD activity as a function of regulation by using trial-triggered averaging (see ‘Methods’ section). In contrast to the previously discussed event-specific transient responses represented by β estimates of activity, we expected to observe baseline increases in activity over the entire time course as a result of the condition (‘Attend’ or ‘Regulate’). For each of the aforementioned regions of interest, trial-triggered averages were calculated for all participants with sufficient data for outcome analyses (n = 40).

As mentioned above, the first contrast (‘Regulate’ Decision > ‘Attend’ Decision) identified voxels in the DLPFC. Confirming the transient-focused contrast of β’s used to define this region of DLPFC, the trial-triggered averages across the decision period exhibited greater activity in the ‘Regulate’ condition as opposed to the ‘Attend’ condition [t(6) = 8.2, P < 0.0002]. Interestingly, that increase in baseline activity persisted throughout the outcome phase [during wins, t(6) = 4.9, P < 0.003; during losses, t(6) = 6.2, P < 0.0008].

Trial-triggered averages from the striatum and VMPFC, identified in the second contrast (‘Attend’ Win > ‘Attend’ Lose), exhibited a parallel pattern to DLPFC activity. Separate ANOVAs performed on the left striatum and the VMPFC also showed increased activity in the ‘Regulate’ condition as compared to ‘Attend’ at decision [repeated measures ANOVA, condition (2) x TR (7); Left striatum main effect of condition F(1,39) = 4.1, P < 0.05; VMPFC main effect of condition F(1,39) = 5.1, P < 0.03], as well as outcome [repeated measures ANOVA, condition (2) x outcome type (2) x TR (7); Left striatum main effect of condition F(1,39) = 7.9, P < 0.008; VMPFC main effect of condition F(1,39) = 14.8, P < 0.001] (Right striatum results were similar to the left striatum; See Supplementary Table S2 for full ANOVA results). This result was further confirmed by planned paired t-tests for both the striatum [left striatum at decision t(6) = 4.5, P < 0.004; during wins, t(6) = 14.5, P < 7 × 10⁻⁶; and during losses, t(6) = 6.6, P < 6 × 10⁻⁴], and for the VMPFC [at decision t(6) = 5.5, P < 0.002; during wins, t(6) = 22.6, P < 5 × 10⁻⁷; and during losses, t(6) = 4.5, P < 0.005] (Figure 5). The data from both striatum and VMPFC suggest that baseline shifts, like those observed in DLPFC above, may also extend into regions more generally associated with value representation.

Fig. 5 Trial-triggered averages for activity in (A) right DLPFC, (B) VMPFC and (C) left striatum. Brown diamonds represent ‘Attend’ activity, and blue circles represent ‘Regulate’ activity. Decision activity is indicated with filled-in markers, outcome activity with outlined markers, and wins and losses with solid and dotted lines, respectively. The contrasts used to define the ROIs are indicated on the right, and their respective lines are plotted in gray on the graph. Decision activity is locked to the time of decision presentation (TRs 0 and 1, indicated by gray block on X-axis). Outcome activity is locked to the presentation of the outcome (labeled TR 7 on the graph, indicated by gray block on X-axis). Error bars are standard error of the mean.
DISCUSSION

In the present study, we examine the BOLD correlates of loss aversion during a risky decision-making task and the effect of intentionally reappraising the meaning of the choice on decisions and brain activity. We find striking similarities both to previous studies directly assessing emotional responses in decision making, as well as to studies of emotion regulation in non-choice domains. Our three main results are: (i) amygdala activity to losses vs gains correlates with estimates of behavioral loss aversion; (ii) individuals’ degree of success in regulating their choices is correlated with changes in amygdala responses to losses only—a pattern that directly echoes previous findings with physiological emotional responses (Sokol-Hessner et al., 2009); and (iii) baseline increases in BOLD activity during reappraisal in regions of the brain, including DLPCF, VMPFC and striatum, that mirror regulation-related activity found in other studies of emotion regulation (Ochsner et al., 2002, 2004; Banks et al., 2007; Eippert et al., 2007; Delgado et al., 2008a, 2008b). Together, these results suggest that the observed BOLD correlates of reappraisal during decision-making overlap with those from emotion regulation. As perspective taking, by definition, alters appraisals, a component of emotion (Scherer, 2005) and emotion regulation (Gross, 1998), these findings confirm the importance of emotion regulation in decision making. Specifically, they suggest value computation includes emotional components that can be intentionally shifted, just like other emotional responses.

Behaviorally, other studies on emotion regulation differ from the current study in that their quantitative analyses of regulation success focused on the group level (Ochsner et al., 2002, 2004; Banks et al., 2007; Eippert et al., 2007; Delgado et al., 2008a, 2008b; Wager et al., 2008; Urry et al., 2009). While we performed group-level analyses, the nature of our task also enabled a second level of analysis, in which we were able to quantitatively investigate the strength or significance of our effect on an individual-participant basis. This approach was as unique to the study of emotion regulation as it was critical in characterizing some of our effects.

In addition to this study, we are aware of two others that have quantitatively estimated individuals’ loss aversion, and examined the relationship to neural function. First, Tom et al. (2007), found a unified representation of expected utility (which included loss aversion) in VMPFC and striatal responses (among other regions; but not the amygdala) at the time of decision over mixed valence gambles. We also found bilateral striatal activity at decision representing expected utility, implicating the striatum in loss aversion. The second study, De Martino et al. (2010), observed two patients with bilateral amygdala lesions as they made choices in a task nearly identical to that in Tom et al. (2007), and found that neither patient was loss averse (λ’s of 0.76 and 1.06; both less than matched controls). In our study, amygdala responses to loss outcomes relative to gain outcomes correlated with loss aversion, implicating the amygdala in its representation. Despite the fact that these findings indicate different regions of the brain in loss aversion (the striatum, Tom et al., 2007; the amygdala, De Martino et al., 2010), we believe our findings and those of the previous two studies can be accounted for in a unified model, proposed below.

The current study, in combination with Sokol-Hessner et al. (2009), links responses (neurally and physiologically) to outcomes with estimates of behavior from decisions. It is possible that responses at outcome may be incidentally linked to decisions, without influencing them. Alternatively, and we think more likely, our findings are consistent with a model in which anticipated responses to outcomes might guide behavior at decision. This latter model, in line with extant theories (LeDoux and Gorman, 2001; Delgado et al., 2008c; Seymour and Dolan, 2008; Talmi et al., 2008; De Martino et al., 2010), suggests that amygdala responses to outcomes may signal emotional salience. These signals modulate activity in regions including the striatum and VMPFC, which have well documented connections with the amygdala (Amaral et al., 1992). This modulation would lead activity in those regions at decision to reflect loss aversion.

Though further testing of this model will clearly be necessary, it can account for the conclusions of previous studies. As Tom et al. (2007) had decisions without outcomes, BOLD activity could have reflected stored values or weights, perhaps from previous experience. De Martino et al. (2010) might then have observed no loss aversion in patients in the same task because of a complete absence of an amygdala signal, during or prior to the study. By imaging decisions and outcomes in the present study, we could confirm Tom et al. (2007), and observe the hypothesized amygdala signal from De Martino et al. (2010) both in baseline behavior and as it changed during reappraisal.

It is additionally notable that the proposed model is similar to modulatory models of the role of emotion and the amygdala in capturing attention (Taylor and Frangopanagos, 2005; Pourotis and Vuilleumier, 2006; Stanley et al., 2009), altering memory ( Phelps et al., 1998; McGaugh, 2004; Kensinger and Schacter, 2008), and learning contingencies ( Phelps et al., 2004; Schiller et al., 2008). The present study suggests a similar modulatory role in decision making.

Further supporting this hypothesis that affective salience plays a role in loss aversion, the pattern of amygdala responses to outcomes exhibits striking similarities to the pattern previously found for arousal responses (Sokol-Hessner et al., 2009). In that study, skin conductance responses to losses (relative to gains) correlated with loss aversion. Individuals who reduced their loss aversion reduced their arousal responses to losses relative to gains, driven by a reduction in the response to losses. The present study showed a similar correlation between loss aversion and the response to losses vs gains in the amygdala, and linked the reduction in loss aversion with reduction in amygdala activity to losses. Though neural data are not evidence of emotion ( Phelps, 2009), it is compelling that amygdala activity exhibits the same pattern observed in research with emotional responses (Sokol-Hessner et al., 2009), consistent with amygdala mediation of physiological arousal responses (Garavan et al., 2001; Glascher and Adolphs, 2003). These parallels suggest that relative emotional responses to outcomes may support loss aversion and its regulation.

In addition to the effects discussed above of reappraisal on participants’ responses to events, like winning or losing money, our blocked design also allowed us to observe consequences of reappraisal on longer timescales. The shifts we found in baseline BOLD activity using trial-triggered averaging suggest that taking a different perspective may also involve maintaining a tonic mindset over the course of a block. Such shifts indicate that perspective taking might be similar to regulatory fit ( Higgins, 2005; Avnet and Higgins, 2006) or manipulations of mood (Lerner et al., 2004; Harlé and Sanfey, 2007; Andrade and Ariely, 2009).

That the DLPCF showed a baseline shift is consistent with a putative role in strategic representation and control (Miller and Cohen, 2001; Badre and D’Esposito, 2007), the (non-intentional) regulation of value and decision making (Knoch et al., 2006a, 2006b; Hare et al., 2009; Bhatt et al., 2010; Figner et al., 2010), and the neural mechanisms supporting emotion regulation (Ochsner and Gross, 2008; Hartley and Phelps, 2010). This suggests that similar neural mechanisms may support regulation in financial situations. Of course, DLPCF localization varies (Ochsner and Gross, 2008; Hartley and Phelps, 2010), and there is currently no consensus on its functional organization (Miller and Cohen, 2001; Badre and D’Esposito, 2007; for more discussion, see Supplementary Data).

Baseline shifts were also found in other brain regions. These include the VMPFC, associated with expected utility (Hampton et al., 2006; Tom et al., 2007; Weber et al., 2007; Hare et al., 2008, 2009), extinction...
Reappraisal and amygdala loss responses

SCANN (2012) 9 of 10


