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The cognitive control of emotion

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The capacity to control emotion is important for human adaptation. Questions about the neural bases of emotion regulation have recently taken on new importance, as functional imaging studies in humans have permitted direct investigation of control strategies that draw upon higher cognitive processes difficult to study in nonhumans. Such studies have examined (1) controlling attention to, and (2) cognitively changing the meaning of, emotionally evocative stimuli. These two forms of emotion regulation depend upon interactions between prefrontal and cingulate control systems and cortical and subcortical emotion-generative systems. Taken together, the results suggest a functional architecture for the cognitive control of emotion that dovetails with findings from other human and nonhuman research on emotion.

> If you are distressed by anything external, the pain is not due to the thing itself, but to your estimate of it; and this you have the power to revoke at any moment.
>
> Marcus Aurelius (Meditations)

Introduction

Conflicts, failures, and losses at times seem to conspire to ruin us. Yet, as Marcus Aurelius observed nearly two millennia ago, we humans have an extraordinary capacity to regulate the emotions occasioned by such travails. Importantly, these regulatory efforts largely determine the impact such difficulties will have on our mental and physical well-being [1–3]. Given its importance to adaptive functioning, it is not surprising that research on emotion regulation has a long history (Box 1). Past work has investigated the cellular responses to stress, the behavioral consequences of adopting specific regulatory strategies, and the neural systems involved in simple forms of affective learning and social behavior in rodents and nonhuman primates [1,4–7]. In recent years, research on emotion regulation has entered a new phase as functional imaging studies of regulatory phenomena in humans have developed rapidly. This growth has facilitated investigation of human analogs to affective behaviors studied in animals, but, perhaps more importantly, has allowed study of the emotion regulatory power of higher cognitive control processes that are difficult to study in animal models. In so doing, current work on the 'hot' control of emotion draws on rapidly developing cognitive neuroscience models of the 'cold' control of attention and memory (e.g. [8,9]). The aim of this review is to evaluate recent imaging studies that, in the context of evidence from allied human and animal work, help to elucidate the functional architecture underlying the cognitive control of emotion.

Emotion and emotion regulation

An essential part of understanding emotion regulatory mechanisms is characterizing the processes that generate emotions. Current models posit that emotions are valenced responses to external stimuli and/or internal mental representations that (i) involve changes across multiple response systems (e.g. experiential, behavioral, peripheral physiological [10]), (ii) are distinct from moods, in that they often have identifiable objects or triggers, (iii) can be either unlearned responses to stimuli with intrinsic affective properties (e.g. an unconditioned response to an aversive shock) or learned responses to stimuli with acquired emotional value (e.g. a conditioned response or stimulus–reward association), (iv) and can involve multiple types of appraisal processes that assess the significance of stimuli to current goals [11], that (v) depend upon different neural systems [3,12,13].

Emotion regulation involves the initiation of new, or the alteration of ongoing, emotional responses through the

Box 1. A brief history of psychological research on emotion regulation

Study of the cognitive control of emotion has three major historical antecedents within psychology [1]. The first antecedent is the psychodynamic study of defense, which was initiated by Freud a century ago. This line of work has examined the regulation of anxiety and other negative emotions using clinical descriptions and individual difference studies of so-called perceptual defenses against processing negatively arousing stimuli, and specific defenses such as repressive coping [68,69]. The second antecedent is the stress and coping tradition that grew out of the psychodynamic approach in the 1960s. This line of work has focused on the management of situations that 'tax or exceed the resources of the person' ([70], p. 141), and generated an early classic study of reappraisal showing that subjective and physiological responses decreased when a film of a potentially upsetting surgical procedure was viewed in analytical and detached terms [71]. The third antecedent is the developmental study of self-regulation, which had its roots in the study of socioemotional development. This work showed that children could obtain a preferred but delayed reward by thinking about available treats in abstract ways (e.g. putting a mental 'picture-frame' around a cookie) that decreased their immediate impulse to eat them [72]. Contemporary research builds on this foundation using both behavioral and neuroscience methods to describe when, how, and with what consequences individuals regulate their emotions.

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Box 2. From basic mechanisms to individual differences

Characterizing the nature and operating characteristics of basic emotion regulatory mechanisms in healthy participants might help to establish a normative model for explaining the successful regulation of emotion. It might also lead to a greater understanding of individual differences, clinical conditions and lifespan development, by describing them in terms of variation and change in the function of a basic functional architecture for the cognitive control of emotion.

Among healthy adults, there is considerable variability in the nature and strength of emotional responses, and also in the capacity to regulate them. Behavioral studies have begun to explore the experiential and behavioral consequences of these differences [73], and characteristic patterns of resting and/or emotional stimulus-related neural activity in prefrontal and emotional appraisal systems are now being associated with gender, personality, negative affectivity [3,74] and regulatory ability. For example, Jackson *et al.* found that greater left PFC electrical activity at rest predicted dampened physiological reactivity to aversive stimuli, which might reflect automatic regulatory processes [75], and Ray *et al.* [76] found that the tendency to cognitively ruminate about emotional events predicted enhanced ability to increase or decrease amygdala responses through reappraisal, which itself depends upon cognitively reexamining the meaning of emotional events.

Many forms of psychopathology revolve around failures to adaptively regulate emotional responses, with consequences ranging from personal distress to socially maladaptive and self-destructive behaviors [2,3,5]. Resting and symptom provocation studies have begun to identify abnormal patterns of neural response in psychiatric illness [3,6,13] and substance abuse (e.g. [77]) that might be related to emotion regulation failures. However, very few studies have examined directly the neural mechanisms mediating successful or unsuccessful regulation in clinical populations using methods like those described in this review (see, however, [78]). Building knowledge of dysregulatory mechanisms from a basic model of effective regulation could elucidate the nature of these disorders and suggest avenues for cognitive and pharmacological treatment.

Basic models of emotion regulation might also help to explain the development of regulatory capacities across the lifespan. It is possible, for example, that structural and functional changes in control and appraisal systems underlie normal and abnormal emotional responses in children [79], and the positivity of emotional experience in older adults [80].

action of regulatory processes. Current work examines the processes that individuals use to influence which emotions they generate, when they do so, and how these emotions are experienced or expressed [1]. Several schemes have been proposed for organizing regulatory strategies (e.g. [14]). One distinction suggested by Gross and colleagues contrasts behavioral (e.g. suppressing expressive behavior) and cognitive (e.g. attending to or interpreting emotion-eliciting situations in ways that limit emotional responding) regulation. Behavioral regulation of negative emotions might limit expressive action but does not dampen unpleasant experience, worsens memory, and increases sympathetic nervous system activation. By contrast, cognitive regulation neutralizes negative experience without impairing memory and might decrease physiological arousal [15,16]. Individual differences in emotional responsivity and/or cognitive control capacity might be related to both normal and pathological variation in well-being and social behavior (Box 2).

Recent imaging work has investigated two types of cognitive regulation, attentional control and cognitive change, which are the focus of this review. Figure 1 uses a hypothetical continuum to illustrate relationships between regulatory strategies tapping these two types of control. These strategies might differ in: (1) their targets – impacting different types of emotional appraisal processes and associated neural systems [17,18]; (2) their effects – serving to initiate (amplify) or block (diminish) perception of our responses to stimuli; (3) their relative reliance on the overlapping neural systems supporting attentional control and cognitive change, as indicated by their placement along the continuum; and (4) whether emotion change is their explicit goal (‘*I want to feel better*!’), or occurs as a by-product of pursuing some other learning or judgment-related goal (e.g. ‘*I want to learn which judgment is correct*’).

Attentional control

Attention is often referred to as the selective aspect of information processing, enabling us to focus on goal-relevant (e.g. our writing) and ignore goal-irrelevant (e.g. loud music next door) information. In general, studies have indicated that behavioral and neural responses to attended as compared with unattended stimuli (or stimulus features) are either facilitated or inhibited, respectively (e.g. [19]). When responses to attended and unattended inputs do not differ, processing is considered to be relatively automatic. In the context of emotion, researchers have begun asking how paying less attention

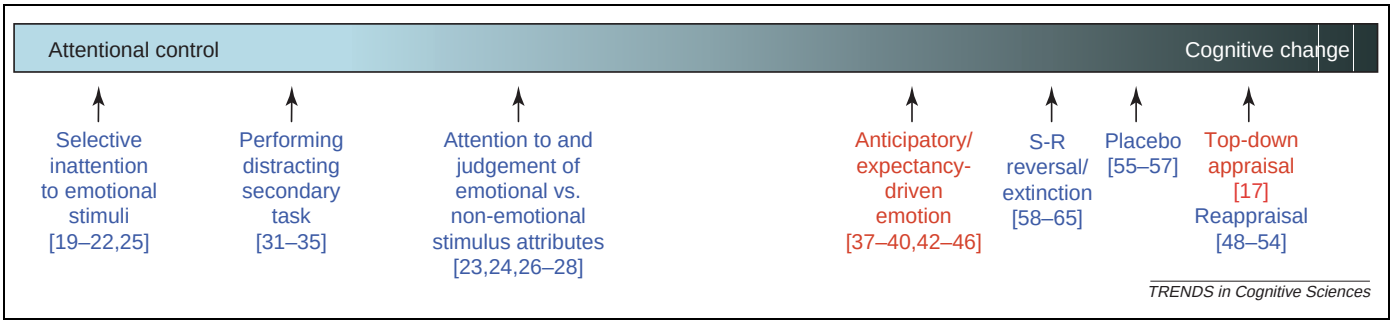

Figure 1. Hypothetical continuum illustrating relationships among the forms of cognitive control of emotion described in this review. The left and right anchors for the continuum represent the exclusive use of attentional control or cognitive change, respectively, to modulate emotion perception and/or responses. Red and blue text denote strategies for controlled emotion generation and regulation, respectively. Relevant citations for each strategy are shown in brackets. This continuum is intended to serve a heuristic function, helping the reader to visualize relationships among control strategies (see text).

to emotional stimuli or their features modulates processing in emotional appraisal systems such as the amygdala.

Selective attention

Several studies have manipulated the amount of attention paid to emotional stimuli by asking participants to selectively judge either their emotional or their perceptual features. These studies, which have focused particularly on modulation of amygdala responses, have produced strikingly discrepant results.

On one hand, some studies have shown that amygdala activation decreases when participants attend to and evaluate emotional features, including matching emotional faces or scenes based on emotional labels rather than perceptual features [20,21], viewing supra- as compared with subliminal presentations of (presumably negative) African American faces [22], judging the expression rather than the gender of fearful, angry, or happy faces [23], or rating their emotional response to aversive scenes rather than viewing them passively [24].

On the other hand, studies have shown amygdala activity to be invariant with respect to attention to emotional features when participants judged the gender of fearful faces rather than judging aspects of simultaneously presented houses [19,25], judged the gender as compared with expression of happy and disgusted [26], or happy, sad, disgusted and fearful faces [27], judged the age or trustworthiness of normatively untrustworthy faces [28], or the age or goodness of normatively 'bad' famous people (e.g. Hitler) [29].

The reasons for these discrepant findings are not yet clear, but two possibilities stand out (see [18], and Critical Summary below). First, some judgments might impose a greater attentional load, which more strongly limits processing of perceptual inputs and as a consequence also limits amygdala responses (cf. [30]). Second, participants might in some cases actively regulate their responses. In keeping with the latter suggestion, when making good/bad evaluations of valenced concepts (e.g. abortion), right ventral lateral prefrontal cortex (LPFC) activation was found on trials for which participants indicated in postscan ratings that they had exerted control [22]. Right ventral LPFC activity is also found in combination with amygdala deactivation during cognitive change, as discussed below. These results could explain why similar reciprocal PFC–amygdala relationships have been observed when participants judged emotional compared with perceptual properties of stimuli [20,21].

Attentional distraction

A second approach to interactions between attention and emotion uses a distracting secondary task to limit attention to emotional stimuli. These studies have focused primarily on responses to pain (however, see [31]), and have found that performance of a verbal fluency task [32], the Stroop task [33,34], or simply being asked to 'think of something else' [35] diminishes the aversiveness of pain, reduces activity in cortical and subcortical pain-related regions, including midcingulate cortex, insula, thalamus and periacqueductal gray, and activates orbitofrontal cortex (OFC), anterior cingulate cortex (ACC) and medial and lateral PFC regions related to cognitive control. It is not yet clear, however, whether these activations reflect (i) deliberate attempts to regulate pain in order to facilitate performance of the distractor task and/or (ii) processes supporting performance of that task directly.

Critical summary

Studies of attentional control have shown that limiting attention to emotional stimuli can limit responses in appraisal systems, but the contexts and mechanisms governing this regulatory effect are not clear. For example, studies of selective attention have used primarily emotional face stimuli whereas studies of distraction have used painful stimuli, confounding type of attentional control and type of stimulus. Furthermore, there has been lack of clarity concerning the underlying processing demands – whether conceived as attentional load or some other type of cognitive operation – imposed by specific judgments or tasks. For studies of selective attention, however, a more important problem might be an over-reliance on brain activation changes – in the absence of corroborating behavioral or physiological measures – to support the inference that emotion regulation has taken place. That fact (coupled with the use of low arousal, face stimuli) has made it difficult to determine whether amygdala modulation reflects regulatory success and/or the failure to elicit a strong response. Although studies of attentional distraction have avoided these pitfalls by using highly arousing (painful) stimuli, questions remain about precisely what processes are being carried out by control systems.

Cognitive change

The use of higher cognitive abilities such as working memory, long-term memory and mental imagery to support learning, judgment and reasoning has been a primary focus of research in cognitive neuroscience. In general, these abilities have been shown to depend upon interactions between prefrontal systems that support control processes and posterior cortical and subcortical systems that represent different types of modality specific (e.g. visual, spatial, auditory) information [8,36]. In the context of emotion, researchers have begun asking how these abilities can be used to construct expectations for, select alternative interpretations of, and/or make different judgments about emotional stimuli [18,36] that can change both behavioral and neural responses to them. Cognitive change might be used either to generate an emotional response when none was ongoing or to regulate an already triggered response.

Controlled generation

The use of cognitive change to generate an emotional response has been studied in three ways.

The first approach has examined the neural correlates of anticipatory responses that precede expected emotional events. Such anticipation has been associated with activation of dorsal medial PFC (MPFC) regions [37–40] implicated in mental state attribution [41], which might reflect cognitive expectations for pleasant or unpleasant experiences, in combination with activation of regions

important for appraising the aversive or rewarding (as compared with neutral) properties of stimuli. Thus, anticipating a painful shock [37,42,43], heat [38] or injection [39] activates cingulate, insula and amygdala; anticipating pleasant or aversive tastes activates amygdala, nucleus accumbens (NAcc) and/or OFC [44]; and anticipating monetary reward activates NAcc, amygdala, insula and cingulate [40].

The second approach has examined how expectations about how a stimulus might feel influence neural responses to it. Studies have shown that nonpainful stimuli are perceived as painful when participants expect pain, and that this expectation leads to activation of midcingulate regions [45] as well as medial temporal and rostral cingulate regions [46], which might be involved in pain affect and cognitive expectations about pain, respectively.

A third approach has directly contrasted top-down responses generated by beliefs about a stimulus with bottom-up responses driven by direct perception of aversive stimuli. To date, only one study has addressed this issue by asking participants either to look at aversive images (bottom-up) or to think about neutral images in negative ways (top-down). Amygdala activation was observed in both conditions. However, only top-down generation activated ACC, LPFC and MPFC systems [17], which might be involved in cognitively generating an aversive appraisal of an otherwise innocuous image.

Controlled regulation

The use of cognitive change to regulate an existing or ongoing emotional response has also been studied in the context of three different forms of higher cognition and learning.

The first type of cognitive regulation is known as reappraisal, and involves reinterpreting the meaning of a stimulus to change one's emotional response to it [47]. In general, studies have found that reappraisal of negative emotion activates dorsal ACC and PFC systems that support the selection and application of reappraisal strategies, and decreases, increases or maintains activity in appraisal systems such as the amygdala or insula in accordance with the goal of reappraisal [48–54]. There has been variability in the precise prefrontal and appraisal systems recruited across studies, however, which might be attributable to differences in the nature of the stimuli used and the goal or content of reappraisal strategies (see below, and [18,51]).

The second type of controlled regulation is implicated in placebo responses to situations that involve no active drug compounds that could impact appraisal systems. Two studies have shown that if participants believe that placebo creams or drugs blunt pain, then painful stimuli elicit less pain and produce (i) decreased activation of amygdala and pain-related cingulate, insula and thalamic regions in combination, with (ii) increased activation of lateral and medial prefrontal regions related to cognitive control, including rostral cingulate cortex and dorsal and right ventral LPFC [55–57]. Although the precise nature of the cognitive processes mediating placebo effects is not yet clear, placebo-related interactions between prefrontal and appraisal systems are strikingly similar to those supporting reappraisal, suggesting that placebo effects are mediated by the active maintenance of beliefs about placebo compounds that change the way in which stimuli are appraised [57].

The third type of cognitive regulation builds on animal models of emotion regulation (e.g. [6,7]) by examining the ways in which simple stimulus–reinforcer associations are formed and altered. Although the precise systems recruited and the nature of interactions among them have differed across studies and paradigms, instrumental avoidance of aversive stimuli [42], extinction of classically conditioned fear responses [58,59] and reversal of stimulus–reward associations [60–63] have been shown to depend upon interactions between similar cognitive control and emotional appraisal systems. On the control side, findings of activation in ventral lateral and medial PFC, OFC and/or ACC have been observed consistently, supported by neuropsychological studies showing impairments of reversal learning in patients with lesions of ventral and orbital but not dorsolateral PFC [64,65]. On the appraisal side, however, findings have been less consistent. For example, amygdala activation has been reported to either decrease [59] or increase [58] during extinction, and during reversal learning both striatal [60] and amygdala activation have been observed, with separate regions of the amygdala tracking previously as compared with currently reinforced stimuli [62]. These discrepancies across studies might be connected with differences in stimulus characteristics, and also how emotional associations are learned and altered.

Critical summary

In general, studies of cognitive change have shown consistently that emotional appraisal systems can be modulated by PFC, OFC and cingulate control systems activated either (i) by high-level expectations for beliefs about, and interpretations of, stimuli, or (ii) by learning to associate new emotional responses with stimuli. These findings are strikingly similar to control dynamics observed for 'cold' forms of control that involve prefrontal and cingulate systems [8,9]. The consistency of these findings (relative to inconsistent results for studies of attentional control) might be attributable to two factors: the use of stimuli that generate strong emotional responses and the use of regulatory strategies that clearly and strongly engage regulatory processes. That being said, questions remain about when and how specific control and appraisal systems interact, including working out exactly why specific control strategies recruit specific control systems and determining the extent to which different strategies modulate appraisal systems in different ways.

Towards a functional architecture of cognitive control of emotion

The goal of this review was to evaluate recent imaging studies whose results can help to elucidate the functional architecture underlying the cognitive control of emotion.

Work using animal models of affective learning and imaging studies of either cognitive control or emotional

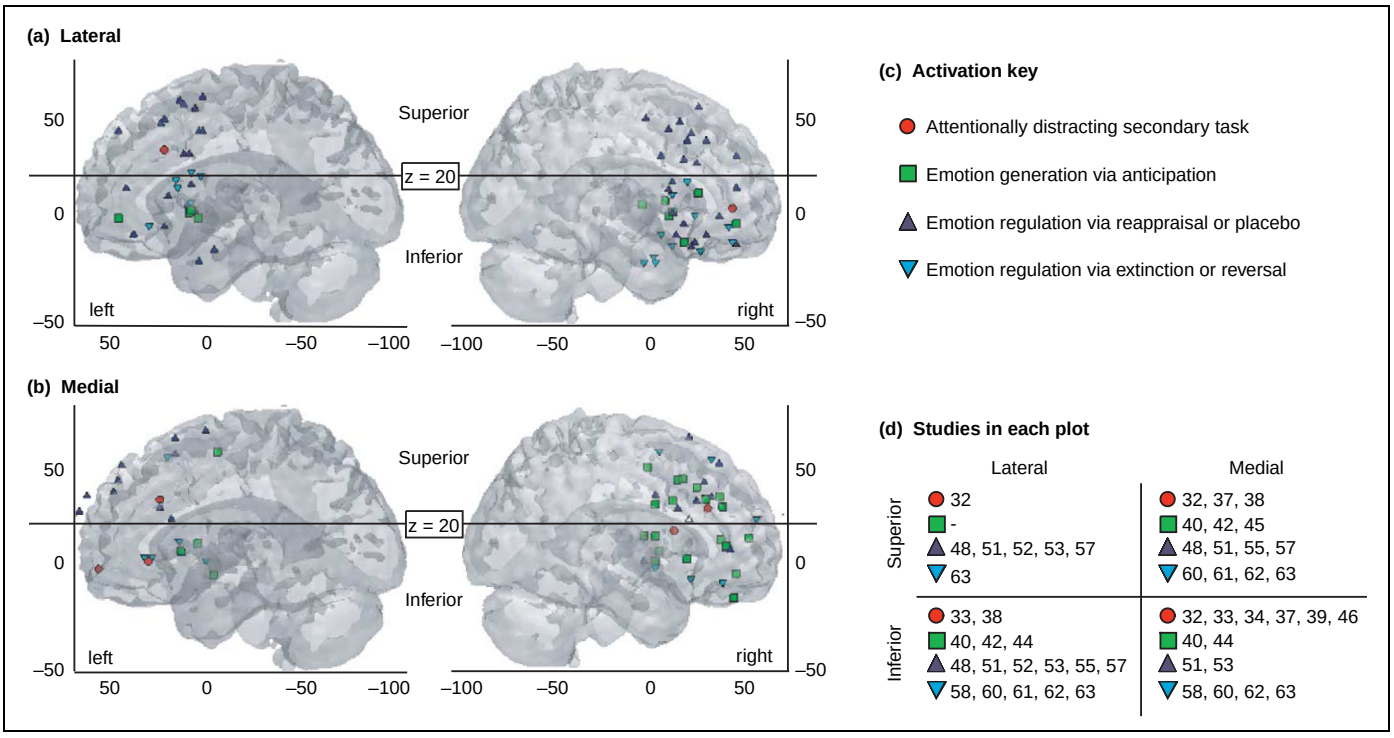

Figure 2. Activations in **(a)** LPFC and **(b)** MPFC associated with different forms of cognitive control over emotional responding located dorsal and ventral to $z=20$ (roughly the median z-coordinate). Each point corresponds to an activation focus representing the results of a contrast isolating regions related to control, shape- and color-coded according to the type of strategy used. **(c)** Activation key indicating which shapes correspond to which types of cognitive control. As described in the text, regulation strategies differ in the extent to which they draw upon dorsal PFC systems supporting redescription of emotional associations or ventral PFC systems supporting alteration of these associations through choice and learning. As is illustrated in **(a)** and **(b)** and listed (by reference number) in **(d)**, reappraisal and placebo recruit dorsal MPFC and both dorsal and ventral LPFC whereas extinction and reversal primarily recruit dorsal and ventral MPFC and only ventral LPFC. Fewer studies have examined attentional distraction and emotion generation, which recruit ventral LPFC and both dorsal and ventral MPFC.

responding in both healthy and psychiatric populations have implicated regions of PFC, OFC and ACC in specific types of control processes and subcortical regions, such as the amygdala, in different types of emotional appraisal [3,5–7,13]. Current imaging work on attentional deployment and cognitive change builds on this work by examining the ways in which these control systems regulate appraisal system activation. The consistent involvement of control–appraisal system dynamics in various forms of regulation suggests a common functional architecture that might be flexibly deployed to support multiple types of control strategies that regulate multiple types of emotional responses.

Furthermore, current imaging work is beginning to identify patterns of functional specificity in cognitive control mechanisms and their impact on emotion-generative systems. For example, it seems that relationships between types of cognitive change might be understood in terms of the extent to which they depend upon two types of control processes (Figure 2). The first type involves ventral PFC and OFC systems used to evaluate the context-appropriate emotional value of stimuli and select actions on the basis of those evaluations. Maintaining representations of these values might *directly* affect emotional associations through direct reciprocal connections with appraisal systems such as the amygdala and NAcc. Through these reciprocal connections, appraisal systems could also affect representation of goal-relevant information in PFC and OFC regions. The second type involves dorsal PFC systems that have few, if any, direct connections with emotional appraisal systems, and are used to explicitly reason about, and describe, how associations between stimuli and emotional responses can be changed. Maintaining representations of these descriptions might provide a task context that *indirectly* affects emotional associations by biasing processing either in the ventral control system or in perceptual and associative memory systems that represent alternative interpretations of events, which in turn send inputs to appraisal systems. Against this backdrop, it can be seen that forms of cognitive change group into those that recruit only ventral systems (stimulus–reward reversal learning and extinction) and those that might recruit both ventral and dorsal systems (reappraisal, placebo and anticipation). A key benefit of this type of classification scheme is that it could help to relate simple forms of affective learning – of the sort studied in animal models – to the use of higher cognitive processes to regulate emotion.

Future directions

Although current research provides converging evidence for a functional architecture for emotion control, it is important to note that for each type of control examined here, limited data and/or variability in activations across studies make it difficult to draw firm and highly specific inferences concerning which control computations are carried out by specific systems, and how they configure for different strategies in different contexts. To address these issues, future work will need to: (1) make use of experiential, behavioral and/or physiological indices that

Box 3. Questions for future research

- When are specific control systems involved in different types of cognitive emotion regulation and what computations does each carry out? How do these control systems relate to those involved in 'cold' forms of cognitive control, such as working memory or attention switching? All recruit LPFC, MPFC and ACC, but are the regions recruited the same?
- When and in what way are specific appraisal systems modulated by different types of cognitive control? Are regulatory effects short-lived or long-lasting? Are different systems involved for positive and negative emotion, discrete emotions such as sadness or disgust, or other affective states such as pleasure and pain?
- To what extent do individual, group, or cultural differences in emotion-response tendencies and/or emotion regulation practices or abilities influence the dynamics underlying cognitive emotion control (see Box 2)?

can provide evidence of emotion modulation *independent of* brain activation; and (2) characterize the precise attentional and cognitive demands for a given regulatory strategy and why they theoretically would be expected to impact specific components of emotional appraisal and response. Addressing both points is crucial to moving beyond general claims that 'emotion processing' has been modulated by 'control systems' to more specific claims about how particular types of cognitive operations can influence particular appraisal processes and channels of emotional response.

As methodological and conceptual clarity increases, future work will be required to address at least three kinds of questions about emotion regulation (see also Box 3). First, the specific regulatory functions carried out by particular control systems are not yet clear. For example, it seems that recruitment of systems might vary as the goal (and/or effect) of control changes from increasing to decreasing emotional responding, and as the operations involved in a given type of strategy are implemented in different ways. Thus far, these two goals or effects have been contrasted directly only in the context of reappraisal (Figure 3). Second, the way in which appraisal systems are modulated by control is also not yet clear. For example, questions about the neural dynamics underlying the regulation of positive compared with negative emotion [49], and the extent to which these effects are durable, remain to be addressed. Third, the relationship of emotion regulatory mechanisms to the mechanisms supporting related behaviors should be examined. For example, future work could compare 'hot' emotion control with 'cold' control of attention and memory, which seem to recruit similar prefrontal and cingulate systems. Systems associated with cognitive emotion control have also been observed in imaging studies of social [66] and reward-related (e.g. [67]) decision making, and with lesion studies of social and emotional behavior (e.g. [36,64,65]). Future work could examine the roles that selective attention to the emotional properties of choice alternatives, anticipation of expected outcomes and reappraisal of disappointing or unexpected outcomes play in these behaviors.

Progress on these exciting questions will take time, of course. Research on these topics is comparatively new, and precise functional descriptions of neural systems will emerge gradually from systematic research programs that target specific types of cognitive control and their emotional impacts. With this in mind, current research can be seen as providing some initial answers – but stimulating many interesting questions for future work – about the neural bases of the cognitive control of emotion.

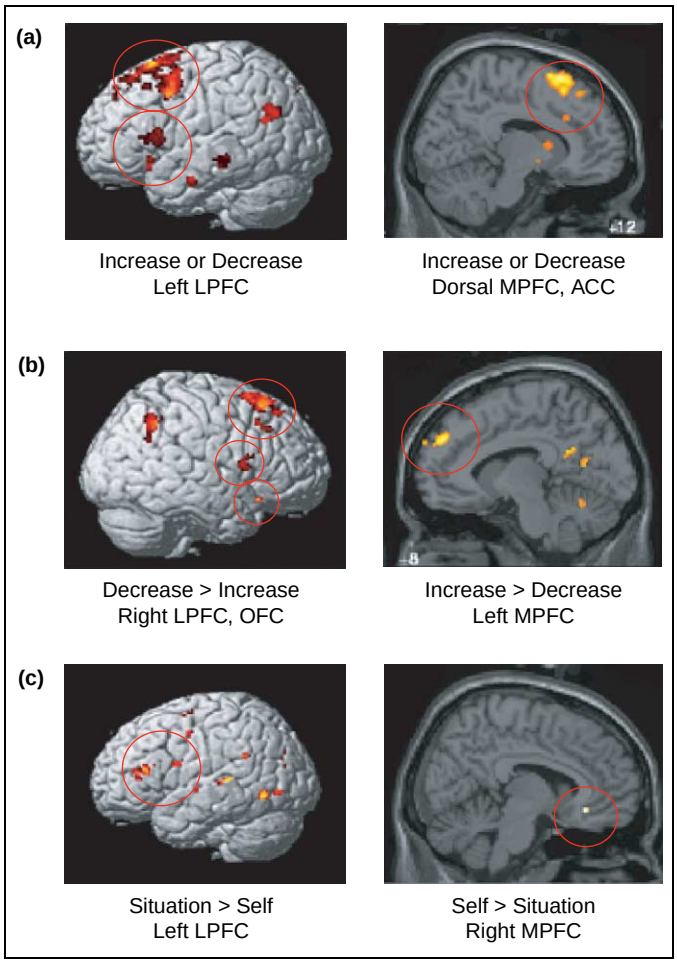

Figure 3. Results from a study examining the effects on brain activation and emotion of systematic variations in the goal and content of reappraisal strategies. Adapted with permission from [51]. **(a)** Regardless of the goal to increase or decrease emotion, common regions of (primarily left) LPFC and ACC were recruited. **(b)** When the goal was to decrease emotion, right ventral LPFC and OFC regions implicated in altering negative associations to stimuli were more active than when the goal was to increase emotion, which differentially recruited left lateral and dorsomedial PFC regions involved in imagining worsening experiences and outcomes. **(c)** When strategies for decreasing emotion involved reinterpreting situations depicted in photos as compared with distancing the self, left lateral as opposed to medial PFC regions were activated, which have been implicated in retrieval of semantic information about context and self-reference, respectively [41,51].

Acknowledgements

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References

1 Gross, J.J. (1998) The emerging field of emotion regulation: an integrative review. *Rev. Gen. Psychol.* 2, 271–299
2 Gross, J.J. and Munoz, R.F. (1995) Emotion regulation and mental health. *Clin. Psychol. Sci. Pract.* 2, 151–164
3 Davidson, R.J. (2000) Affective style, psychopathology, and resilience: brain mechanisms and plasticity. *Am. Psychol.* 55, 1196–1214
4 Charney, D.S. (2004) Psychobiological mechanisms of resilience and vulnerability: implications for successful adaptation to extreme stress. *Am. J. Psychiatry* 161, 195–216

5 Kalin, N.H. and Shelton, S.E. (2003) Nonhuman primate models to study anxiety, emotion regulation, and psychopathology. *Ann. N. Y. Acad. Sci.* 1008, 189–200
6 Quirk, G.J. and Gehlert, D.R. (2003) Inhibition of the amygdala: key to pathological states? In *The Amygdala in Brain Function: Basic and Clinical Approaches: Vol. 985* (Shinnick-Gallagher, P. and Pitkänen, A., eds), pp. 263–325, New York Academy of Sciences
7 Holland, P.C. and Gallagher, M. (2004) Amygdala–frontal interactions and reward expectancy. *Curr. Opin. Neurobiol.* 14, 148–155
8 Botvinick, M.M. *et al*. (2004) Conflict monitoring and anterior cingulate cortex: an update. *Trends Cogn. Sci.* 8, 539–546
9 D'Esposito, M. *et al*. (2000) Prefrontal cortical contributions to working memory: evidence from event-related fMRI studies. *Exp. Brain Res.* 133, 3–11
10 Cacioppo, J.T. *et al*. (2000) The psychophysiology of emotion. In *The Handbook of Emotion* (Lewis, R. and Haviland-Jones, J.M., eds), pp. 173–191, Guilford Press
11 Scherer, K.R. *et al.*, eds (2001) *Appraisal Processes in Emotion: Theory, Methods, Research*, Oxford University Press
12 Ochsner, K.N. *et al*. (2001) A multiprocess perspective on the neuroscience of emotion. In *Emotions: Current Issues and Future Directions* (Mayne, T.J. and Bonanno, G.A., eds), pp. 38–81, Guilford Press
13 Phillips, M.L. *et al*. (2003) Neurobiology of emotion perception II: Implications for major psychiatric disorders. *Biol. Psychiatry* 54, 515–528
14 Parkinson, B. and Totterdell, P. (1999) Classifying affect-regulation strategies. *Cogn. Emot.* 13, 277–303
15 Gross, J.J. (2002) Emotion regulation: affective, cognitive, and social consequences. *Psychophysiology* 39, 281–291
16 Jackson, D.C. *et al*. (2000) Suppression and enhancement of emotional responses to unpleasant pictures. *Psychophysiology* 37, 515–522
17 Ochsner, K.N. and Gross, J.J. (2004) Thinking makes it so: a social cognitive neuroscience approach to emotion regulation. In *Handbook of Self-Regulation: Research, Theory, and Applications* (Baumeister, R.F. and Vohs, K.D., eds), pp. 229–255
18 Ochsner, K.N. Characterizing the functional architecture of affect regulation: emerging answers and outstanding questions. In *Social Neuroscience* (Cacioppo, J.T., ed.), MIT Press (in press)
19 Anderson, A.K. *et al*. (2003) Neural correlates of the automatic processing of threat facial signals. *J. Neurosci.* 23, 5627–5633
20 Hariri, A.R. *et al*. (2000) Modulating emotional responses: effects of a neocortical network on the limbic system. *Neuroreport* 11, 43–48
21 Hariri, A.R. *et al*. (2003) Neocortical modulation of the amygdala response to fearful stimuli. *Biol. Psychiatry* 53, 494–501
22 Cunningham, W.A. *et al*. Separable neural components in the processing of black and white faces. J. *Neurosci.* (in press)
23 Critchley, H. *et al*. (2000) Explicit and implicit neural mechanisms for processing of social information from facial expressions: a functional magnetic resonance imaging study. *Hum. Brain Mapp.* 9, 93–105
24 Taylor, S.F. *et al*. (2003) Subjective rating of emotionally salient stimuli modulates neural activity. *Neuroimage* 18, 650–659
25 Vuilleumier, P. *et al*. (2001) Effects of attention and emotion on face processing in the human brain: an event-related fMRI study. *Neuron* 30, 829–841
26 Gorno-Tempini, M.L. *et al*. (2001) Explicit and incidental facial expression processing: an fMRI study. *Neuroimage* 14, 465–473
27 Winston, J.S. *et al*. (2003) Common and distinct neural responses during direct and incidental processing of multiple facial emotions. *Neuroimage* 20, 84–97
28 Winston, J.S. *et al*. (2002) Automatic and intentional brain responses during evaluation of trustworthiness of faces. *Nat. Neurosci.* 5, 277–283
29 Cunningham, W.A. *et al*. (2003) Neural components of social evaluation. *J. Pers. Soc. Psychol.* 85, 639–649
30 Bishop, S. *et al*. (2004) Prefrontal cortical function and anxiety: controlling attention to threat-related stimuli. *Nat. Neurosci.* 7, 184–188
31 Pessoa, L. *et al*. (2002) Neural processing of emotional faces requires attention. *Proc. Natl. Acad. Sci. U. S. A.* 99, 11458–11463
32 Frankenstein, U.N. *et al*. (2001) Distraction modulates anterior cingulate gyrus activations during the cold pressor test. *Neuroimage* 14, 827–836
33 Bantick, S.J. *et al*. (2002) Imaging how attention modulates pain in humans using functional MRI. *Brain* 125, 310–319
34 Valet, M. *et al*. (2004) Distraction modulates connectivity of the cingulo-frontal cortex and the midbrain during pain – an fMRI analysis. *Pain* 109, 399–408
35 Tracey, I. *et al*. (2002) Imaging attentional modulation of pain in the periaqueductal gray in humans. *J. Neurosci.* 22, 2748–2752
36 Beer, J.S. *et al*. (2004) Frontal lobe contributions to executive control of cognitive and social behavior. In *The Cognitive Neurosciences: III* (Gazzaniga, M.S., ed.), pp. 1091–1104, MIT Press
37 Hsieh, J.C. *et al*. (1999) PET study on central processing of pain in trigeminal neuropathy. *Eur. J. Pain* 3, 51–65
38 Ploghaus, A. *et al*. (1999) Dissociating pain from its anticipation in the human brain. *Science* 284, 1979–1981
39 Porro, C.A. *et al*. (2002) Does anticipation of pain affect cortical nociceptive systems? *J. Neurosci.* 22, 3206–3214
40 Knutson, B. *et al*. (2001) Dissociation of reward anticipation and outcome with event-related fMRI. *Neuroreport* 12, 3683–3687
41 Ochsner, K.N. *et al.* Reflecting upon feelings: an fMRI study of neural systems supporting the attribution of emotion to self and other. *J. Cogn. Neurosci.* (in press)
42 Jensen, J. *et al*. (2003) Direct activation of the ventral striatum in anticipation of aversive stimuli. *Neuron* 40, 1251–1257
43 Phelps, E.A. *et al*. (2001) Activation of the left amygdala to a cognitive representation of fear. *Nat. Neurosci.* 4, 437–441
44 O'Doherty, J.P. *et al*. (2002) Neural responses during anticipation of a primary taste reward. *Neuron* 33, 815–826
45 Sawamoto, N. *et al*. (2000) Expectation of pain enhances responses to nonpainful somatosensory stimulation in the anterior cingulate cortex and parietal operculum/posterior insula: an event-related functional magnetic resonance imaging study. *J. Neurosci.* 20, 7438–7445
46 Ploghaus, A. *et al*. (2001) Exacerbation of pain by anxiety is associated with activity in a hippocampal network. *J. Neurosci.* 21, 9896–9903
47 Gross, J.J. (1998) Antecedent- and response-focused emotion regulation: divergent consequences for experience, expression, and physiology. *J. Pers. Soc. Psychol.* 74, 224–237
48 Ochsner, K.N. *et al*. (2002) Rethinking feelings: an FMRI study of the cognitive regulation of emotion. *J. Cogn. Neurosci.* 14, 1215–1229
49 Kim, S.H. and Hamann, S.B. (2004) Voluntarily increasing and decreasing emotional responses to positive and negative emotional pictures modulates amygdala activity and subsequent memory. In *Annu. Meeting Cogn. Neurosci. Soc.*, p. 15, Cognitive Neuroscience Society
50 Phan, K.L. *et al*. (2005) Neural substrates for voluntary suppression of negative affect: a functional magnetic resonance imaging study. *Biol. Psychiatry* 57, 210–219
51 Ochsner, K.N. *et al*. (2004) For better or for worse: neural systems supporting the cognitive down- and up-regulation of negative emotion. *Neuroimage* 23, 483–499
52 Levesque, J. *et al*. (2003) Neural circuitry underlying voluntary suppression of sadness. *Biol. Psychiatry* 53, 502–510
53 Beauregard, M. *et al*. (2001) Neural correlates of conscious self-regulation of emotion. *J. Neurosci.* 21, RC165
54 Schaefer, S.M. *et al*. (2002) Modulation of amygdalar activity by the conscious regulation of negative emotion. *J. Cogn. Neurosci.* 14, 913–921
55 Lieberman, M.D. *et al*. (2004) The neural correlates of placebo effects: a disruption account. *Neuroimage* 22, 447–455
56 Petrovic, P. *et al*. (2002) Placebo and opioid analgesia – imaging a shared neuronal network. *Science* 295, 1737–1740
57 Wager, T.D. *et al*. (2004) Placebo-induced changes in FMRI in the anticipation and experience of pain. *Science* 303, 1162–1167
58 Gottfried, J.A. and Dolan, R.J. (2004) Human orbitofrontal cortex mediates extinction learning while accessing conditioned representations of value. *Nat. Neurosci.* 7, 1144–1152
59 Phelps, E.A. *et al*. (2004) Extinction learning in humans: role of the amygdala and vmPFC. *Neuron* 43, 897–905
60 Cools, R. *et al*. (2002) Defining the neural mechanisms of probabilistic reversal learning using event-related functional magnetic resonance imaging. *J. Neurosci.* 22, 4563–4567
61 Kringelbach, M.L. and Rolls, E.T. (2003) Neural correlates of rapid reversal learning in a simple model of human social interaction. *Neuroimage* 20, 1371–1383

62 Morris, J.S. and Dolan, R.J. (2004) Dissociable amygdala and orbitofrontal responses during reversal fear conditioning. *Neuroimage* 22, 372–380
63 Rogers, R.D. *et al*. (2000) Contrasting cortical and subcortical activations produced by attentional-set shifting and reversal learning in humans. *J. Cogn. Neurosci.* 12, 142–162
64 Fellows, L.K. and Farah, M.J. (2005) Different underlying impairments in decision-making following ventromedial and dorsolateral frontal lobe damage in humans. *Cereb. Cortex* 15, 58–63
65 Hornak, J. *et al*. (2004) Reward-related reversal learning after surgical excisions in orbito-frontal or dorsolateral prefrontal cortex in humans. *J. Cogn. Neurosci.* 16, 463–478
66 Rilling, J. *et al*. (2002) A neural basis for social cooperation. *Neuron* 35, 395–405
67 McClure, S.M. *et al*. (2004) Separate neural systems value immediate and delayed monetary rewards. *Science* 306, 503–507
68 Erdelyi, M.H. (1974) A new look at the New Look: perceptual defense and vigilance. *Psychol. Rev.* 81, 1–25
69 Paulhus, D.L. *et al*. (1997) Psychological defense: contemporary theory and research. In *Handbook of Personality Psychology* (Hogan, R. *et al*., eds), pp. 543–579, Academic Press
70 Lazarus, R.S. and Folkman, S. (1984) *Stress, Appraisal and Coping*, Springer
71 Lazarus, R.S. and Alfert, E. (1964) Short-circuiting of threat by experimentally altering cognitive appraisal. *J. Abnorm. Psychol.* 69, 195–205
72 Mischel, W. *et al*. (1989) Delay of gratification in children. *Science* 244, 933–938
73 Gross, J.J. and John, O.P. (2003) Individual differences in two emotion regulation processes: implications for affect, relationships, and well-being. *J. Pers. Soc. Psychol.* 85, 348–362
74 Hamann, S. and Canli, T. (2004) Individual differences in emotion processing. *Curr. Opin. Neurobiol.* 14, 233–238
75 Jackson, D.C. *et al*. (2003) Now you feel it, now you don't: frontal brain electrical asymmetry and individual differences in emotion regulation. *Psychol. Sci.* 14, 612–617
76 Ray, R.D. *et al*. Individual differences in trait rumination modulate neural systems supporting the cognitive regulation of emotion. *Cogn. Affect. Behav. Neurosci.* (in press)
77 Volkow, N.D. and Fowler, J.S. (2000) Addiction, a disease of compulsion and drive: involvement of the orbitofrontal cortex. *Cereb. Cortex* 10, 318–325
78 Mayberg, H.S. (2003) Modulating dysfunctional limbic-cortical circuits in depression: towards development of brain-based algorithms for diagnosis and optimised treatment. *Br. Med. Bull.* 65, 193–207
79 Levesque, J. *et al*. (2004) Neural basis of emotional self-regulation in childhood. *Neuroscience* 129, 361–369
80 Mather, M. *et al*. (2004) Amygdala responses to emotionally valenced stimuli in older and younger adults. *Psychol. Sci.* 15, 259–263