OPINION

The neural bases of emotion regulation

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Abstract | Emotions are powerful determinants of behaviour, thought and experience, and they may be regulated in various ways. Neuroimaging studies have implicated several brain regions in emotion regulation, including the ventral anterior cingulate and ventromedial prefrontal cortices, as well as the lateral prefrontal and parietal cortices. Drawing on computational approaches to value-based decision-making and reinforcement learning, we propose a unifying conceptual framework for understanding the neural bases of diverse forms of emotion regulation.

Emotions shape how we think, feel and behave, and they may be regulated in various ways[1]. When emotion regulation is absent, deficient or poorly matched to situational demands, emotional responses may be excessive, inappropriate or insufficient, as is seen in the context of various psychiatric disorders[2]. Unfortunately, despite the importance of such regulation, we lack a mechanistic framework for its analysis and for offering testable hypotheses regarding its underlying computations and neurobiological processes. In this Opinion article, we provide a framework for understanding emotion regulation that emerges from neuroimaging studies involving various emotion-regulation paradigms and conceptual and computational advances in other domains of self-regulation. Our hope is that this new framework will encourage the development of novel computational and experimental approaches to investigate emotion regulation.

Emotion and emotion regulation

Emotions consist of sets of cognitive, subjective, physiological and motor changes that arise from an individual's conscious or non-conscious determination that a stimulus has a positive or negative value in a particular context and with respect to that individual's currently active goals[3]. Stimuli that drive this 'good versus bad' determination may be internal or external to the individual, and they may have an innate or acquired value. Hence, the process of valuation is at the core of emotion. Emotions unfold over time, and the cognitive, subjective, physiological and motor components of emotion may furthermore be discordant with each other with respect to timing, magnitude and duration[4,5]. Importantly, emotions alter the state of the individual and/or the environment (that is, through the actions of the individual), often leading to the achievement of a more 'good for me' or less 'bad for me' state (that is, an 'action' output). For example, fear might motivate avoidance of a dangerous situation for the individual, and happiness may lead to an individual repeating an action. Seen in this way, an emotion can be described as a perception–valuation–action (PVA) sequence[6–8] (the emotional-reactivity PVA sequence), in which input from the external or internal world is perceived, valued and then triggers an action that alters the external or internal world (FIG. 1).

At the neural level, emotions engage highly evolutionarily conserved subcortical systems, such as the amygdala, ventral striatum and periaqueductal grey (PAG), as well as a set of cortical regions (more elaborated in primates) that include the anterior insula and dorsal anterior cingulate[9–21] (dACC; FIG. 2). Thus, multiple anatomical locations are associated with emotion. The variety of information coding that occurs in these structures accounts in part for the multi-component features of emotion (that is, the cognitive, subjective, physiological and motor changes). Moreover, each structure processes information to varying levels of abstraction or incorporation of contextual information. For example, core limbic regions, such as the amygdala, ventral striatum and PAG, may extract simple motivational features of a stimulus (for example, a snake being a potential threat); cortical regions, such as the insula, may provide additional interoceptive information; the hippocampus may provide temporal and spatial context related to memory; and the dACC may relate the stimulus to other motivational demands on the individual[8].

Emotion regulation refers broadly to implementation of a conscious or non-conscious goal to start, stop or otherwise modulate the trajectory of an emotion[7]. Although emotion regulation is conceptually distinct from the unfolding of the emotion itself, it nonetheless also involves a PVA sequence, such as that described above for an emotion[7]. Emotion regulation is triggered when the emotional reaction itself (that is, the action output of an emotional-reactivity PVA sequence) becomes the target of valuation (FIG. 1) or when there is conflict between different emotional-reactivity PVAs in determining behaviour. As in emotions, 'good for me–bad for me' valuations of emotional reactions may be innate or learned through experience, and they may be driven by contextual factors or the individual's goals. The action output of the emotion-regulation PVA sequence is the process of emotion regulation itself, which may be carried out consciously or non-consciously and may potentially target any component of the emotional-reactivity PVA sequence (FIG. 1).

Glossary

Computational modelling
The application of algorithms representing functions computed by the brain to explain observed behaviour through latent variables.

Conditioned stimulus
(CS). A previously neutral stimulus that takes on aversive or rewarding properties after being associated with an unconditioned stimulus.

Limbic regions
Deep brain structures (for example, the amygdala, ventral striatum and brain stem nuclei) involved in emotional and motivational processes.

Prediction errors
Discrepancies between experienced stimuli and expectations about them.

Reinforcement learning
An area of study describing changes in behaviour driven by the experience of rewards or punishments.

Transcranial magnetic stimulation
(TMS). A method for non-invasive stimulation of the brain using a focal pulsed magnetic field, which can be used to excite or inhibit brain activity.

Unconditioned stimulus
(US). A naturally aversive or rewarding stimulus.

Value
A dimensionless 'universal currency' that denotes the relative 'good for me' or 'bad for me' motivational relevance of a stimulus or action.

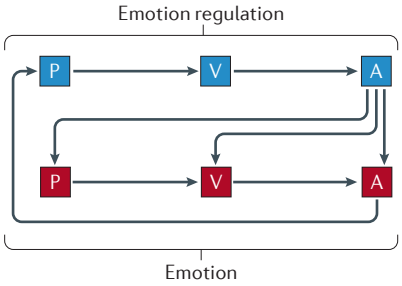

Figure 1 | **A valuation perspective on emotional reactivity and regulation.** Emotional reactivity is a perception–valuation–action (PVA) sequence (red boxes). The valuation reflects a 'good for me–bad for me' judgment about a stimulus, which triggers a multi-componential set of actions (for example, physiological, cognitive, motoric and subjective actions). Emotion regulation also involves a PVA sequence that is driven by valuation of the emotion itself, with its action being regulation of the emotional response (blue boxes). Such regulation may affect any component of the emotional-reactivity PVA sequence.

Based on behavioural and neuroimaging findings, two broad types of emotion regulation have been distinguished: 'explicit' and 'implicit' regulation[22]. Explicit regulation of emotion requires conscious effort for initiation and demands some level of active monitoring of emotion during implementation, and thus is associated with some level of insight and awareness. The most-commonly studied explicit regulation strategy is reappraisal, which entails explicit alteration of the self-relevant meaning (an appraisal) of an emotion-inducing stimulus. Meta-analyses of neuroimaging studies have found that reappraisal is associated with activation of various brain regions, namely the frontoparietal executive network — including the dorsolateral prefrontal cortex (dlPFC), the ventrolateral PFC (vlPFC) and the parietal cortex — as well as the insula, supplemental motor area (SMA) and pre-SMA[23,24] (FIG. 2).

Implicit regulation is characterized by the absence of an explicit instruction, is evoked automatically by the stimulus itself, runs to completion without conscious monitoring, and can happen without insight and awareness[22]. Examples of this type of regulation are inhibition of fear and regulation of emotional conflict[25–36]. In these paradigms, neural activation is consistently observed in the ventral ACC (vACC) and the ventromedial PFC (vmPFC)[25–36] (FIG. 2).

We currently lack a unified computational and mechanistic framework within which we can understand these two types of emotion regulation. We propose that such a framework can be derived by considering another domain of self-regulation that deals with predictions, valuation and prediction errors, and for which a large amount of experimentally well-developed computational literature exists: reinforcement learning, a component of value-based decision-making[37–41].

The reinforcement learning view

The field of reinforcement learning has developed and validated computational models of choice behaviour. Individuals continually make choices based on predictions about the 'good for me–bad for me' value of stimuli or actions. Value predictions that are discrepant with the rewards or punishments that are experienced after a choice yield prediction errors, which are used to update the values of potential choices and thereby drive future behaviour.

Here, we consider emotion regulation as a set of decisions about actions that are aimed at achieving a desired emotional state, which is specified within the multi-componential space of emotion. This state is therefore the predicted outcome of the emotion-regulatory actions. Thus, an emotion-regulatory action with a particularly 'good for me' predicted outcome will have a high value when deciding whether to engage in emotion regulation and which strategy to use to do so. Although we refer to a decision-making process, the computations that determine and execute emotion-regulatory actions may occur consciously or non-consciously (and thus may be conserved to differing degrees between humans and other animals). A discrepancy between the predicted emotional state and the actual emotion can therefore be considered a prediction error, indicating that the regulatory action has not attained its expected outcome. When the emotion has fallen short of the target emotional state, there is a negative prediction error (that is, an expected event in the intended direction failed to occur). When the emotion has exceeded the target emotional state, there is a positive prediction error. These emotion regulation-prediction errors update the decisional value of the emotion-regulatory action, supporting the ability to continue making decisions about whether to engage in emotion regulation and which strategy to use (or switch to).

Emotion-regulatory actions also have costs that are associated with their execution. This means that the decisional value of a regulatory action reflects both the predicted outcome and the cost of execution. There are probably situations in which a decision is made to not regulate because the benefits of such regulation are small or the costs are large. Although empirical research has primarily focused on downregulation of negative emotion, the same conceptual and computational structure can equally apply to up- or downregulation of negative or positive emotions.

Computational accounts of reinforcement learning distinguish between two types of decisional control — model-free control and model-based control — which can both update predicted stimulus or action values and drive choice behaviour. Model-free control is characterized by its responsiveness to environmental events within a limited set of potential stimuli and responses. In such control, behaviour is guided solely based on experienced prediction errors (that is, assuming no a priori knowledge) and is therefore computationally efficient but not very flexible. Model-based control is characterized by application of rule-based decision-making and dynamic computation of optimal actions (based on an internal model that represents the individual's a priori knowledge of the context) but is less computationally efficient. Although learning often shapes the internal model, application of model-based control does not require new experiences to occur for a model to be built. It is particularly useful when a priori knowledge can create a shortcut to a decision or when adaptive decisions simply cannot be made in a timely manner through model-free control alone. Models may be built in a 'supervised' manner by instruction or through observing the actions of others.

Across many decision-making paradigms and stimulus modalities, the choice with greater positive value is associated with greater vACC–vmPFC activation[39,40,42–44]. Association of reward with a chosen stimulus or action will result, in a model-free manner, in a further increase in the decisional value of that stimulus or action, leading to that stimulus or action being more readily chosen next time — a process encoded in vACC–vmPFC activity[40]. During model-based control, when decision-making has to consider both external stimuli and internal a priori models, activation of the vACC–vmPFC is insufficient to guide choice by itself[37,45]. In this case, cognitive control systems (such as the frontoparietal executive network)[46] and the core cognitive capacities they mediate (for example, working memory) are needed for making use of an internal model[47,48]. For example, cognitively taxing individuals by having them perform a second task concurrently with reinforcement learning leads to greater use of model-free

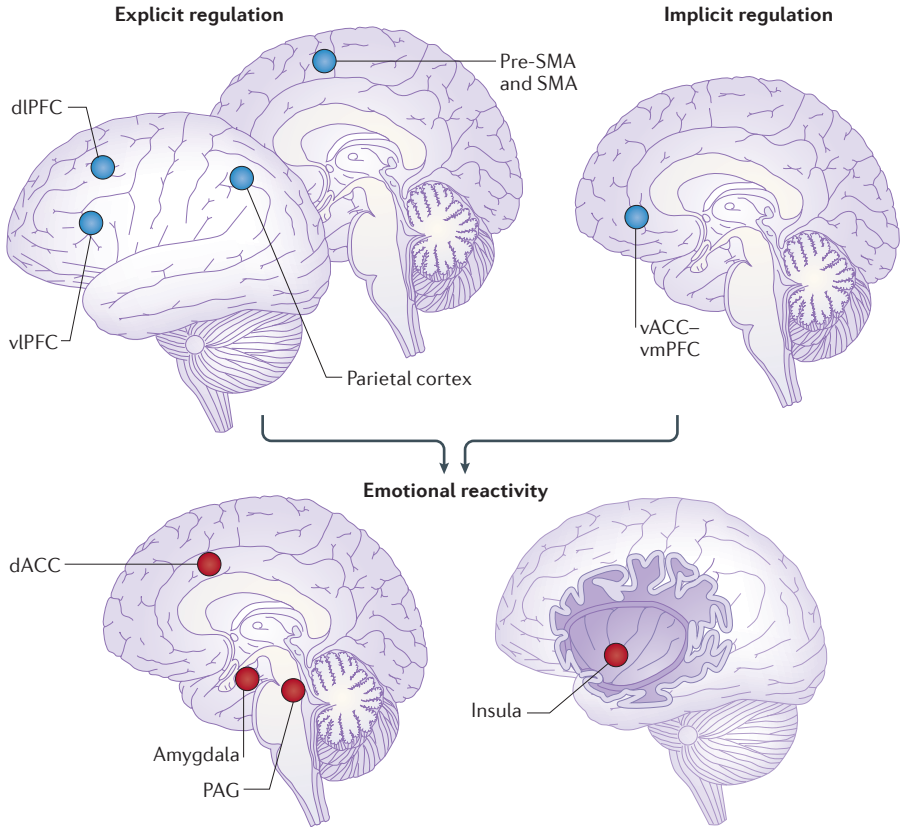

Figure 2 | **Regions implicated in emotion regulation.** The dorsal anterior cingulate (dACC), insula, amygdala and periaqueductal grey (PAG) (shown in red) have been implicated in emotional reactivity. By contrast, the dorsolateral prefrontal cortex (dlPFC), ventrolateral PFC (vlPFC), supplementary motor area (SMA), pre-SMA and parietal cortex (shown in blue) have been implicated in 'explicit' emotion regulation, and the ventral ACC (vACC)–ventromedial PFC (vmPFC; also shown in blue) has been implicated in 'implicit' emotion regulation.

over model-based control[47]. Likewise, application of inhibitory repetitive transcranial magnetic stimulation (rTMS) to the dlPFC biases individuals towards model-free from model-based control[46], as does experimentally induced stress (which also impairs working memory); interestingly, in both the stress and the rTMS studies, individuals with greater working memory capacity experienced less disruption of model-based control. The lateral PFC may also itself encode value signals[49,50].

We discuss in more detail below several emotion-regulatory paradigms and examine how they may map onto concepts of model-free and model-based control.

Model-free emotion regulation

We propose that, in model-free emotion regulation, activity in the vACC–vmPFC reflects experience-dependent alteration in the value of emotion-regulatory behaviour (FIG. 3a). Thus, model-free emotion regulation would be able to proceed entirely based on prediction error feedback. Various forms of fear inhibition and regulation of emotional conflict exemplify this type of regulation, wherein an increase in vACC–vmPFC activity drives the individual into a more 'good for me' state (a lower level of fear or less reaction-time slowing) entirely in response to environmental contingencies. Although prior ideas about implicit emotion regulation align heavily with the concept of model-free emotion regulation, the key operational principle is to best capture the mechanisms of control (that is, model-free) rather than whether those mechanisms require conscious awareness (which may itself shift across time).

In a typical fear-inhibition experiment in humans, an individual first learns to associate a previously neutral conditioned stimulus (CS) with an aversive unconditioned stimulus (US). Conditioned responses to the CS can be extinguished if the CS is repeatedly presented without the US. From a valuation perspective on emotion regulation, mounting a fear response when it is unnecessary given the changed conditioning context is costly and undesirable to the individual. This cost–benefit analysis thus favours regulating the fear response over not regulating it, and the cost–benefit can be tracked and adjusted simply based on experienced CS-driven emotional prediction errors.

At the neural level, a fear-conditioned CS activates regions such as the amygdala, insula and dACC, as well as brainstem regions like the PAG[10,14–18,51,52]. By contrast, fear extinction engages the vACC–vmPFC[51]. These results are consistent with findings from lesion and inactivation studies in animals, which demonstrate that fear inhibition as a consequence of extinction is an active process and not a decay of the original fear memory[25–27]. Similarly, acute reversal in humans of the CS–US contingency that was established during conditioning (that is, when a previously non-reinforced CS now predicts the US and the previously reinforced CS does not) is associated with vACC–vmPFC activation[28]. This activation is greater in magnitude than the activation associated with acquisition of the non-reinforced CS, suggesting that the vACC–vmPFC signal during reversal reflects an active 'safety signal' or positive value that is related to omission of the US rather than lack of a fear response per se.

During tests of fear generalization in which one CS is paired to a US and a second CS is unpaired (and thus becomes a safety-signalling stimulus), stimuli that are the most similar to the unpaired CS are associated with greatest activation in the vACC–vmPFC[29,30]. By contrast, activation in the dACC and insula is greatest for stimuli that are the most similar to the paired CS[29,30]. Occasions in which an individual overcomes an acute threat (for example, they decide to approach a threat, such as allowing a snake to be advanced towards them in the MRI scanner) are associated with vACC–vmPFC activation and with reduced activation in the insula and dACC[31]. Likewise, exposure to a distant threat is associated with greater vACC–vmPFC activation than is exposure to an imminent threat[32]. In all of these cases, vACC–vmPFC activation is seen when fear is inhibited, putting the individual in a more 'good for me' state. Concomitantly, decreased activation is seen in emotional-reactivity regions, such as the amygdala, insula, dACC and PAG.

A second example of model-free emotion regulation comes from the emotional conflict task[33,34] — a version of the classic Stroop paradigm that involves assessment of emotions[53]. Participants are presented with photographs of fearful or happy faces

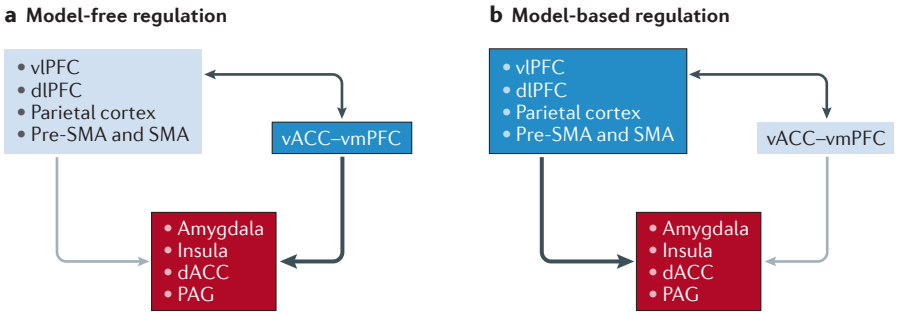

Figure 3 | **Schematic of model-based and model-free emotion regulation. a** | We propose that model-free emotion regulation involves the primary action of the ventral anterior cingulate (vACC)–ventromedial prefrontal cortex (vmPFC) in encoding the experience-dependent value of regulatory actions, which modulate activity in emotional-reactivity regions (for example, the amygdala, insula, dorsal ACC (dACC) and periaqueductal grey (PAG)). **b** | By contrast, we propose that model-based emotion regulation involves the primary action of the frontoparietal and dorsal midline (supplementary motor area (SMA) and pre-SMA) cortices, which are used in the implementation of an internal model to compute the value of emotion-regulatory actions and guide behaviour. The arrow between model-free and model-based systems indicates that the systems are likely to communicate which each other and that both types of regulation may occur to differing degrees at the same time. dlPFC, dorsolateral PFC; vlPFC, ventrolateral PFC.

with the word 'fear' or 'happy' written over them and are asked to indicate whether the facial expression is fearful or happy. The word either matches the facial expression (congruent trials) or does not match the facial expression (incongruent trials). The emotional conflict during incongruent trials induces reliable reaction-time slowing, as well as activation in emotional-reactivity regions, such as the amygdala, dACC and insula[33–35].

Less reaction-time slowing is seen in incongruent trials if they were preceded by another incongruent trial, when compared with incongruent trials that had been preceded by a congruent trial, thus indicating that regulation occurred. The slowing associated with prior incongruent trials (compared with prior congruent trials) is therefore valued as 'bad for me,' triggering regulation, which can proceed in a model-free manner. Indeed, post-incongruent incongruent trials are associated with increased activity in the vACC–vmPFC and decreased activity in the amygdala, dACC and insula[33–35]. Furthermore, individuals with lesions in the vACC–vmPFC are unable to regulate emotional conflict[54].

Model-based emotion regulation

We propose that, in model-based emotion regulation, frontoparietal regions are recruited for implementation of an internal model to guide behaviour, and that these regions may also encode value (FIG. 3b). Consistent with this formulation, we argue below that model-based emotion regulation requires intact working memory capacity to construct and/or make use of internal models. Crucially, we conceptualize model-based regulation as being particularly helpful in situations in which model-free regulation would not be selected, despite its lower implementation cost, because doing so would take too long to effectively regulate emotion or because prediction error-based adjustments alone could not achieve the desired regulation outcome.

Reappraisal exemplifies the model-based category of emotion regulation. As such, although previous concepts about explicit emotion regulation align heavily with model-based control, here we shift the emphasis to computational mechanisms rather than to whether conscious awareness is required. When an individual uses reappraisal to decrease negative emotion, they seek to alter the meaning of the stimulus (that is, the emotional valuation phase)[1,7]. For example, the individual may search for alternative subdominant meanings (such as someone's tears reflecting joy rather than sadness) until the stimulus no longer induces an emotional response[1]. Model-based emotion regulation is characterized by its ability to flexibly change in response to contextual and environmental demands by using the internal model of the individual's external environment and internal state (that is, an 'internal simulation'). Such an internal model is explicitly invoked when teaching participants how to reappraise in a task[55,56]. In fact, an a priori internal model is referenced in all forms of explicit emotion regulation, even though the specific strategies may differ (for example, distancing or distraction). Arguably, it would be difficult to effectively regulate emotion in complex, often socially relevant, contexts without an internal model.

Meta-analyses show that, when reappraisal is used to decrease negative emotion, activation is seen in the dlPFC, vlPFC, dACC–dorsomedial PFC (dmPFC), pre-SMA, SMA, insula and parietal cortex, and decreased activation is seen in the amygdala[23,24] (FIG. 2). One study found that the relationship between reappraisal success and vlPFC engagement was separately mediated by ventral striatal and amygdalar activity[57]. Another study found that better emotion-regulatory success, as determined by an electromyography measure, was associated with greater amygdala inhibition and stronger negative connectivity between the amygdala and the dlPFC and dACC–dmPFC[58]. Interestingly, greater amygdala inhibition during emotion regulation was also associated with a better ability to decrease pain through a similar reappraisal strategy[59]. Indeed, up- or downregulation of pain through a related strategy was associated with activation in the inferior frontal junction and SMA as well[60]. Although this finding with respect to overlapping neural substrates supports a potential similarity between regulation of pain as one type of stimulus and negative emotions more broadly, specific work comparing regulation of negative emotions induced with typical visual stimuli and regulation of pain is needed to determine precise points of neural and mechanistic overlap and divergence.

Consistent with predictions from work on model-based control during reinforcement learning and at least some overlap between frontoparietal brain systems involved in cognitive control and working memory with those involved in model-based emotion regulation[23,24,61–63], individual differences in reappraisal ability have been related to individuals' working memory capacity[22,56,64]. A recent study showed that enhancing dlPFC excitability through the use of transcranial direct-current stimulation improved participants' ability to both downregulate and upregulate negative emotions using reappraisal, as assessed by self-report and physiological measures[65]. In another study, the effect of experimentally induced stress was tested on participants' ability to use reappraisal to decrease physiological responses to a fear-conditioned CS[66]. Stress, which impairs working memory and dlPFC function[67–69], disrupted the ability of reappraisal to reduce

autonomic responses to the CS. Although relatively few imaging studies have examined forms of model-based regulation other than reappraisal, studies of both distraction and expressive suppression (that is, prevention of emotional expressions) found that both strategies engage dlPFC, vlPFC and dACC circuitry[56,70,71]. Ultimately, the precise overlap and differences in circuits used for cognitive control and different types of model-based emotion regulation may benefit from sophisticated multi-voxel pattern-analysis methods to clarify neural subcomponents[72] or methods such as representational similarity analyses (which incorporate computational modelling)[73]. Regardless, conceptual and computational approaches from reinforcement learning can still be applied to understand emotion regulation.

Recent work has also found that, although greater vACC–vmPFC activity encodes positive subjective value of emotional pictures when participants are asked to experience the emotion without regulating, this is not the case when participants engage in reappraisal[74]. Another study examined the effect of a cognitive regulation strategy aimed at up- or downregulating food cravings on value coding of these stimuli[50]. They found positive subjective value signals in both the vACC–vmPFC and dlPFC when participants experienced food stimuli. During downregulation, activity increased in the dlPFC but not the vmPFC, whereas during upregulation, activation increased in the vmPFC but not the dlPFC. This is consistent with non-human primate work that found coding of positive and negative value by dlPFC neurons during reinforcement learning[49]. Reappraisal of fear-conditioned stimuli also diminishes autonomic reactivity to the CS and recruits both dlPFC and vACC–vmPFC activity[75].

Computational implementation

The classic model for reinforcement learning is that described by Rescorla and Wagner[76]. In its simplest form, the model can be described as follows:

$$V_t = V_{t-1} + \alpha\delta \quad (1)$$

In this equation, V reflects the decision value, t is trial or time point, δ is the prediction error and α is the learning rate. A higher learning rate would indicate a greater impact of the prediction error on value updating. The Rescorla–Wagner model and its adaptations rely on signed prediction errors, whereas other related formulations rely on unsigned prediction errors. The Pearce–Hall model[77] exemplifies the latter case, replacing the Rescorla–Wagner learning rate with two variables: associability (the absolute value of the prediction error) and the salience of one or multiple stimuli. These models can also be effectively combined[78,79].

With respect to emotion regulation, we propose that the decision to pursue emotion regulation (or to do nothing and let the emotional reaction play out), as well as the choice among different regulatory strategies, involves a comparison of the predicted value of each action, accounting also for the cost (C) of each action (for example, see FIG. 4), and can be written as follows:

$$V(n)_t = V(n)_{t-1} + \alpha\delta - C(n) \quad (2)$$

Hence, a general formulation of the decisional value of the n^{th} potential emotion-regulatory action ($V(n)_t$) would reflect the predicted emotional state on trial or time point *t*. This value would be calculated by updating the prior value of this action ($V(n)_{t-1}$) based on the prediction error (δ_n), multiplied by a learning rate (α). The prediction error is the discrepancy between the measured emotional reactions minus the predicted emotional state (in multicomponential space). The learning rate thus determines the relative importance of the emotional reactivity discrepancy on regulation. Given that many emotion-regulatory actions may be possible, we propose that selecting a particular emotion-regulatory action at time point *t–1* would lead to its value being updated by the prediction error for time *t*. The value of alternative unselected strategies may either not be updated or be decreased based on the magnitude of the prediction error. Finally, the value of each emotion-regulatory action must also take into consideration its specific implementation cost, C(n).

Evidence already exists for the impact of emotion or regulation on computational parameters during reinforcement learning. Exposure to fearful faces before a predictive cue in a reward-based reinforcement-learning task leads to faster acquisition of the rule, a higher learning rate and increased amygdala–striatal connectivity[80]. Similarly, reappraisal and related model-based emotion-regulation strategies modulate prediction error and expected-value signals in the striatum[81], as well as counterfactual prediction errors (that is, to non-experienced outcomes) in the insula[82].

However, to adapt computational models derived from reinforcement learning to emotional regulation, it is also necessary to incorporate measures of an individual's cognitive, subjective, physiological and motoric responses to emotional stimuli. Although limited in scope, evidence suggests that this is indeed possible. In studies of the reversal of learned fear, reinforcement learning-related models were fit to skin-conductance data, finding that the skin-conductance signal correlates independently with both value and

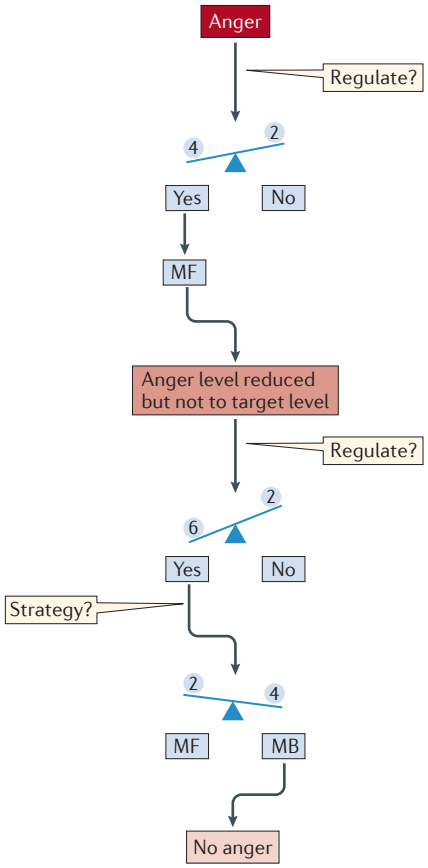

Figure 4 | **Example of emotion regulation as a decision-making process.** Consider a situation in which a hot-tempered manager shouts at his employees. The employee's initial reaction, borne out of evaluating this situation as 'bad for me', is to react with anger. However, as this response would be detrimental to their job security, the emotional reaction is also valued as 'bad for me', and hence is targeted for potential regulation. The decision to engage in regulation involves a comparison of the value (accounting for cost) of regulating versus that of not regulating, here favouring regulation (four arbitrary units versus two). We argue that, in many instances, model-free (MF) regulation will be the default strategy used, which in this case reduces the emotional reaction. Partial success, at least in this example, with MF regulation increases the value of the regulatory action (six arbitrary units versus two). However, the fact that the emotional reaction was not sufficiently eliminated then leads to another decision. In this example, comparison of the value (and cost) of model-based (MB) versus MF regulation (four arbitrary units versus two) favours selection of a MB regulation strategy, which finally achieves the original goal of regulation.

Box 1 | **Emotion regulation abnormalities in psychopathology**

Studies examining emotion-regulation abnormalities in individuals with psychiatric conditions often consist of single reports for a particular regulation strategy or disorder, mostly with anxiety and depressive disorders. Nonetheless, these studies point to the clinical relevance of understanding the computational mechanisms underlying emotion regulation. As examples of impairments in model-free forms of emotion regulation, patients with either post-traumatic stress disorder or obsessive-compulsive disorder were impaired in their ability to express fear-extinction memories acquired 24 hours earlier[87,88] and showed impaired activation of the ventral anterior cingulate (vACC)–ventromedial prefrontal cortex (vmPFC) during fear extinction. In a fear-generalization task, patients with general anxiety disorder (GAD) failed to activate the vACC–vmPFC in response to a conditioned stimulus unpaired with an unconditioned stimulus (that is, in response to a 'safety signal')[30]. Likewise, patients with GAD or depression failed to activate the vACC–vmPFC or deactivate the amygdala during emotional conflict regulation[35,89].

Studies on model-based emotion regulation in psychiatric disorders have primarily focused on reappraisal. Findings in patients with depression have been inconsistent, with reports of patients showing a failure in dampening amygdala activity[90,91] and hypoactivation of the dorsolateral PFC (dlPFC)[92], but also showing hyperactivation of the dlPFC and related structures[91–93], and showing no changes in activity[94]. Although limited, findings from studies in patients with anxiety disorders have been less variable, finding underactivation of the dlPFC and dorsomedial PFC[95–99].

It is not yet clear which components of the computations underlying emotion regulation are most affected in any particular disorder or task (for example, prediction error signalling, value coding and updating, or strategy selection and implementation). Likewise, it is not clear whether emotion-regulatory impairments are primarily due to a failure to activate key regions or to abnormalities in the connectivity and coordination between regions. Nonetheless, the presence of abnormalities in conventional analyses of these paradigms encourages development of a computational modelling approach to emotion regulation.

associability[28,79]. Moreover, prediction error signals were found in the dACC, striatum, insula and thalamus[28], consistent with our formulation above. Activity in the amygdala was found to correlate with associability[79]. Within the realm of cognitive phenomena such as errors, conflict and contextual variability, two computational models closely related to reinforcement learning have been proposed to explain the function of the dACC (and presumably related structures)[83]. In these models, prediction errors are calculated based on violations of expectations of accuracy and reaction times in a task. As these behavioural measures also reflect cognitive components of emotional reactivity, they could be used when modelling emotion regulation. Finally, momentary self-reported positive emotion in response to outcomes during a reinforcement-learning task could be predicted by computationally derived parameters for expected value and prediction error, rather than by simply winnings in the task[84].

From a model implementation perspective, we propose that the prediction error term can be calculated in multi-componential space by contrasting the actual emotion on each trial (for example, through measuring the skin-conductance responses, startle responses, reaction times and/or subjective ratings) with the predicted emotion. In this way, the predicted emotion would also be expressed in units similar to those for the multi-componential emotional response. We anticipate that optimal fitting of computational models may also require inclusion of different simultaneous recordings of emotional responses, such as heart rate, skin conductance and startle, that each reflect (potentially discordant) components of emotional reactivity[4,5]. In fact, the discordance between these response channels may be one reason why learning effective emotion regulation is more difficult than acquiring stimulus–reward associations in typical reinforcement-learning tasks. This problem may be further accentuated in psychopathological states (BOX 1).

The process by which the brain selects between potential emotion-regulation strategies is also important to understand. Drawing on computational and neuroimaging work in reinforcement learning[37], we propose that the emotion-regulatory strategy with the greatest value (accounting for cost) will be applied first (FIG. 4). In new situations in which no a priori internal model readily applies, this would mean a preference for model-free strategies, transitioning to model-based strategies if the initial attempts fail. However, in situations in which a well-informed internal model applies and would more-easily result in effective emotion regulation, individuals may begin with a model-based strategy rather than transitioning after a model-free strategy fails. A similar logic would apply to the choice between different forms of model-based regulation[85,86]. Recent findings in a sequential two-choice decision task probing both model-free and model-based control during reinforcement learning found that activity in the vlPFC and frontopolar cortex may reflect the arbitration between both types of strategies[45]. Negative connectivity between these regions and value-encoding regions emerged during transition between model-free and model-based control, consistent with model-based control requiring inhibition of aspects of a default model-free control path. In a similar manner, the vlPFC was more-negatively coupled with the vmPFC when using a model-based cognitive strategy to down-regulate food cravings — a condition under which the dlPFC coded value more strongly than did the vmPFC[50].

Ultimately, it is unlikely that any individual adopts an exclusively model-free or model-based form of emotion regulation. Rather, one type of strategy may predominate, and the relative weight of each strategy may be continually adjusted; alternatively, both strategies may proceed at the same time, with one predominating (FIG. 3). Although we draw heavily on insights from reinforcement learning, this also does not mean that learned changes in predictive valuation occur at the same timescale in emotion regulation as in reinforcement learning. There is little doubt that effective emotion regulation is a learned process, but it may take a few attempts or even years of repeated attempts to achieve success. Patterns of brain activation during different emotion-regulation paradigms may therefore reflect either short-term learning processes observed in real-time or the consequences of many years of practice.

Conclusions

We have proposed a unified conceptual framework for understanding emotion regulation by drawing on advances in a related area, namely reinforcement learning, and considering emotion regulation in terms of predictions, prediction errors and valuation. Within this conceptual framework, it may now be possible to develop computational models for emotion regulation and to construct task conditions under which the interaction between model-free and model-based regulation can be studied, as has been productively undertaken for reinforcement learning[37]. More broadly, our framework suggests that motivated behaviour involves

a common set of model-free and model-based regulatory processes, whether it is considered from the perspective of reinforcement learning and value-based decision-making or from the perspective of emotion regulation.

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