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REVIEW

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A neurocognitive theory of flexible emotion control: The role of the lateral frontal pole in emotion regulation

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Abstract

Emotion regulation is essential to survive in a world full of challenges with rapidly changing contextual demands. The ability to flexibly shift between different emotional control strategies is critical to successfully deal with these demands. Recently, decision neuroscience has shown the importance of monitoring alternative control strategies. However, this insight has not been incorporated into current neurocognitive models of emotional control. Here, we integrate insights from decision and affective sciences into a novel viewpoint on emotion control, the Flexible Emotion Control Theory (FECT). This theory explains how an individual can flexibly change emotion-regulatory behavior to adapt to varying goals and contextual demands. Crucially, FECT proposes that rapid switching between alternative emotional control strategies requires concurrent evaluation of current as well as alternative (unchosen) options. The neural implementation of FECT relies on the involvement of distinct prefrontal structures, including the lateral frontal pole (FPl) and its connections with other cortical (prefrontal, parietal, motor) and subcortical systems. This novel account of emotion control integrates insights from decision sciences, clinical research, as well as meta-analytic evidence for the consistent FPl involvement during emotional control when monitoring of alternative emotional control strategies is required. Moreover, it provides novel, neurocognitively grounded starting points for interventions to improve emotion control in affective disorders, such as anxiety and aggression.

KEYWORDS
alternative action strategies, emotion control, emotion regulation, lateral frontal pole

INTRODUCTION

Our ability to dynamically control emotions is essential to successfully operate in a world that challenges us constantly with complex and rapidly changing situations. Emotion control refers to nonconscious and conscious strategies by which the behavioral, psychophysiological, and/or subjective aspects of an emotional response are altered or regulated.[1] Emotion regulation strategies include changing the perception of an emotional stimulus (e.g., through distraction), its affective re-evaluation (e.g., through cognitive reappraisal), or its concomitant behavioral response (e.g., emotional action control).[1,2] Because the efficacy of these different emotional control strategies is largely dependent on individual traits and contextual demands,[3–6] effective emotional control requires continuous monitoring and adjustments. The importance of monitoring current affective states is acknowledged in current neurocognitive models of emotion control,[7,8] but it

remains unclear how adjustments are cognitively selected and neurally implemented. This review addresses this largely neglected issue. We elaborate on the suggestion, drawn from the decision-making literature, that effective control requires alternative strategies to be concurrently monitored.

We argue that counterfactual reasoning enables an individual to arbitrate when to switch to which alternative strategy. In terms of neural implementation, decision neuroscience has clearly linked the lateral frontal pole (FPl) to the optimization of control strategies to changing contextual demands.[9–11] The FPl is also consistently recruited during complex emotion control tasks[2,12] and it is involved in higher-order integrative processes and control functions, including emotional awareness.[13,14] Yet, the frontal pole has been largely overlooked in the empirical emotion control literature as well as in neurocomputational models of emotion regulation. The current contribution introduces a novel neurocognitive viewpoint on emotion and action control, the Flexible Emotion Control Theory (FECT). This theory includes an integrated view on the neural implementation of emotion control and provides neurocognitively grounded starting points for intervention into affective disorders characterized by maladaptive emotion control.

NEUROCOGNITIVE THEORIES OF EMOTION REGULATION

Emotion theories have traditionally been divided into two broad clusters. On the one hand, there are evolutionary-based theories, including theories of basic emotions[15,16] and Jamesian theories stressing the relevance of bodily percepts.[17–19] On the other hand, there are theories emphasizing the importance of cognitive appraisals,[20–22] influenced by social constructivist views.[23,24] Despite the differences in focus, there is more convergence between these two clusters than traditionally assumed.[25] Darwin and James already acknowledged the role of cultural influences in emotional responses. Furthermore, most evolutionarily grounded emotion researchers would agree that an emotional experience evolves not purely from the perception of an external cue[15] or bodily percept[19] but requires a form of appraisal. For instance, several accounts consider a Bayesian neural prediction system weighing the perceptual evidence provided by an external cue against prior knowledge to appraise the emotional value of the cue. Also, most theories agree that such appraisal can occur automatically. Finally, it has been suggested that a behaviorally adaptive neural predictive system should operate allostatically,[26] integrating expected changes in interoceptive and exteroceptive signals across different homeostatic systems.[2,27,28] The insight of allostatic control can be also applied to emotion control, considering accounts focused on a single emotion regulation strategy as special cases of a more general allostatic system. Below, we will review the main principles and empirical evidence provided by reinforcement learning theories that mainly focus on a single emotion regulation strategy.[7,14,27–30] Then, we will integrate recent insights, showing how varying contextual demands of real life require an allostatic control system that can proactively arbitrate between different emotional control strategies.[2] The overall goal is to generate an integrated view that explicitly accounts for the neurocognitive implementation of multiple-strategy emotion control.

Single-strategy neurocognitive models of emotion regulation

One of the currently predominant and most explicit neurocomputational models of emotional regulation (by Etkin and colleagues)[7] conceptualizes emotional control in the context of reinforcement learning (RL).[31] In RL models, behavioral choices are based on predicted values of actions and stimuli. Alternative actions are chosen in case discrepancies between actual and predicted rewards or punishments arise, thereby reinforcing behavior that optimizes rewards and minimizes punishments.[31] Etkin and colleagues[7] applied this model to the case of emotion regulation. According to their view, during emotion regulation, behavior is aimed at achieving a desired emotional state (the predicted outcome) by engaging an emotional control strategy, such as reappraisal. The effectiveness of the emotional control strategy is evaluated based on discrepancies between the actual and the expected emotional state. The discrepancy is known as prediction error. Prediction errors thus signal that the emotion regulation strategy does not deliver the desired emotional outcome. Prediction errors can arise either because the desired emotional state was not obtained (negative prediction error), or because it was surpassed (positive prediction error).[7] Emotional prediction errors can be generated in several subcortical and cortical structures, including the amygdala, striatum, insula, thalamus, periaqueductal grey (PAG), and anterior cingulate cortex (ACC).[32–35] In case of a negative prediction error, the emotion regulation strategy would need to be adapted. Etkin and colleagues[7] link different forms of emotion regulation to different forms of RL models. Model-free RL processes are based on experienced prediction errors only, not requiring a priori knowledge of the context and resulting in reinforcement of the recent choices with the greatest reward value. Model-free processes are computationally simple but rather inflexible.[36,37] They have been linked to processes, such as fear inhibition (e.g., during extinction learning) and emotional conflict effects (e.g., in emotional Stroop tasks).[7] In contrast, model-based emotional control is more cognitively demanding, as it depends on internal models of the individual's emotional state and contextual information, for instance, experiences of how the emotional control strategy was used in a similar context in the past.[36,37] Model-based emotion regulation processes have been linked to cognitive reappraisal and distraction.[7] RL models of emotion control, like the model by Etkin and colleagues,[7] additionally postulate continuous monitoring of the effectiveness of the current emotional control strategy, with adjustments as required to achieve a desired emotional state.[7] However, those and other neurocognitive theories of emotion control[1,7,8] do not explain how options for adjustments are generated. Thus, although the model by Etkin and colleagues[7] provides an elegant RL-based view of a so-called "emotional state comparator," there are a few issues when trying to explain how our brain arbitrates between different types of model-based emotion regulatory strategies.

Toward multiple-strategy neurocognitive models of emotion regulation

According to Etkin and colleagues,[7] model-based emotion regulation is a costly mechanism that requires working memory and relies on ventrolateral, dorsolateral PFC, and parietal and (supplementary) motor cortices, because it requires the generation of an internal model (with a priori knowledge about the costs and consequences of the emotion regulation strategy). The model proposes RL principles to come to the decision to "regulate or not" or to implement a "model-free versus model-based strategy." However, it remains unclear how this mechanism arbitrates between a currently employed regulation strategy and an alternative strategy. It also remains unarticulated how our brain can implement choices between different model-based emotion regulation strategies in a flexible fashion. We complement this RL theory with a contrasting hypothesis on how this arbitration is neurally implemented. More specifically, in contrast to an "emotional state comparator" proposed by Etkin and colleagues,[7] we propose an "alternative emotion regulation strategy comparator" to account for flexible emotion control. We ground this new theory in three recent lines of evidence. First, we build on recent insights from the decision-making literature indicating that not only evidence for the chosen option (current regulation strategy) but also for nonchosen options is actively monitored online. This parallel monitoring does not only lead to yes/no decisions on whether or not to regulate, but provides an explanatory framework on how one can flexibly shift between distinct emotion regulation strategies. Second, we incorporate neuroscientific evidence showing that distinct parts of the frontal lobe are implicated in these types of monitoring, including the FPl for monitoring evidence for the nonchosen option.[38] We argue that we have to keep online evidence for the nonchosen regulation option to be able to switch flexibly between regulatory strategies. Third, we integrate evidence for the involvement of the FPl in the emotion control literature, a literature that has been largely ignored in neurocognitive models of emotion regulation, despite the consistent evidence for the involvement of this brain region when more than one emotion regulation type can be implemented.[2,12]

In short, we argue here that alternative emotional control strategies should also be concurrently monitored. This process would enable an individual to decide when to switch to which alternative strategy when needed. Below, we argue that this is a critical feature of emotion control. We aim to extend current neurocomputational models of emotion control by integrating evidence from cognitive flexibility, decision neuroscience, and meta-analytical insights on emotion regulation, and we provide an alternative hypothesis for the neural implementation of flexible emotion regulation.

Insights from decision neurosciences—The relevance of monitoring the nonchosen option

It has been well established that the outcome of an emotion regulatory strategy can vary in different contexts, and as a consequence, this requires the ability to flexibly switch between different strategies to meet contextual demands.[4,5] These authors developed an emotion regulation choice task in which participants are presented with high- or low-intensity emotional pictures. Based on the observation that healthy individuals tend to flexibly shift to choose reappraisal for low-intensity and distraction for high-intensity pictures, adaptive regulatory choice flexibility is defined as the maximal switch in regulatory preference from choosing distraction under high-intensity pictures to selecting reappraisal under low intensity.[39] Interestingly, such regulatory flexibility has been associated with stress resilience in primary responders: repeated trauma exposure was associated with increased post-traumatic stress symptoms in firefighters with low regulatory ability, but not in those with high regulatory flexibility.[39] Thus, this contextual switching ability may be crucial for effective emotional control (see also Refs. 40–42).

However, these models do not consider where different emotional control strategies come from. Contextual switching ability implies that an individual generates *alternative* emotional control strategies. Drawing on the decision-making literature, we recently proposed that contextual switching between different emotional control strategies could be explained by hierarchical models.[2] In these models, a repertoire of strategies is considered.[43,44] These models imply that alternative (counterfactual) emotional control strategies, including those not directly guiding ongoing behavior, are concurrently evaluated to achieve emotional control.[10,44] Besides monitoring the effectiveness of the *ongoing* emotional control strategy, evidence in favor of several *counterfactual* emotional control strategies should be concurrently evaluated to enable an individual to adaptively change emotional control behavior to meet personal and contextual demands.[43,44] The decision-making literature indicates that, in order to infer when to switch to which alternative strategy, evidence in favor of multiple emotional control strategies should be concurrently evaluated. This enables retrieval of an alternative course of action previously used to guide behavior, in case the ongoing behavior does not result in the expected outcome. Additionally, new control strategies may be created based on internal models of previously learned behavior, given current action outcomes and external cues.[43,44] By focusing on the evaluation and generation of alternative emotional control strategies, this decision-making framework may account for the human ability to flexibly adapt emotional behavior to unknown and/or changing situations.[2]

THE ROLE OF THE FPl IN EMOTION AND ITS CONTROL

The FPl and its role in cognitive control—Monitoring alternative strategies

One of the key regions that might be involved in monitoring alternative emotion regulation strategies is the rostrolateral prefrontal cortex or frontal pole. This region is located on the most anterior part of the human prefrontal cortex. It is larger, both in absolute size and relative to total brain volume, than the corresponding frontal regions

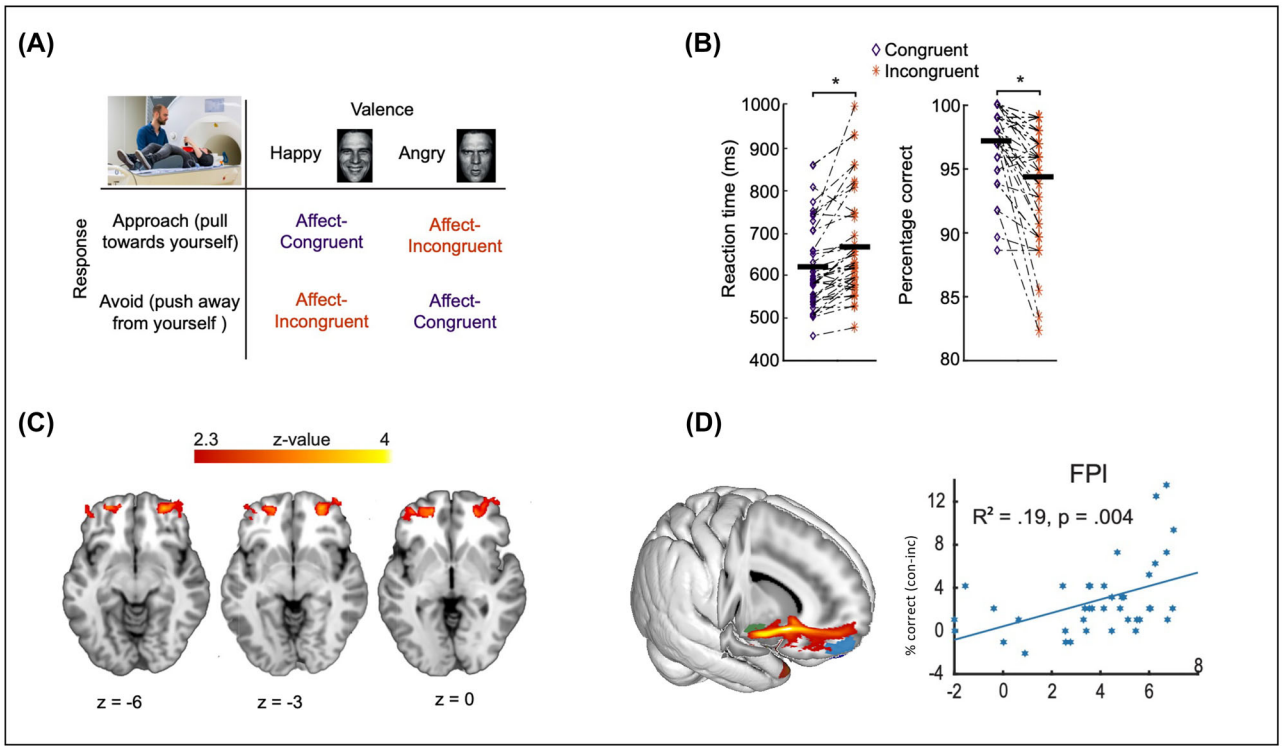

FIGURE 1 Functional and structural evidence for the involvement of the FPl in emotional action control in a social approach-avoidance (AA) task. (A) Schematic representation of the AA task typically used to measure control over emotional action tendencies. The affect-incongruent condition requires control to override the automatic tendencies to approach happy faces and to avoid angry faces in a speeded MR-compatible joystick task. (B) Behavioral effects of the AA task: Participants show longer reaction times and more errors during the affect-incongruent condition, illustrating the cost of exerting control (asterisk indicates $p < 0.01$). (C) Neural effects of the AA task: Incongruent trials recruit stronger activation in the anterior PFC than congruent trials. (D) Illustration of the reconstructed amygdalofugal tract that leads from the amygdala to the anterior PFC. The number of times the tract ends in the FPl correlates with behavioral congruency in error rates on the AA task ($r = 0.44$, $p = 0.0044$), suggesting that the amygdalofugal pathway is involved in mediating FPl–amygdala functional interactions during emotional action control. Probabilistic tractography indicated that 10–20% of interindividual variation in emotional regulation abilities is accounted for by the strength of structural connectivity between the FPl and amygdala.[74] Figure adapted from Bramson et al.[74]

of other hominoids.[45] The frontal pole consists of a lateral and a medial subdivision, each with distinct cytoarchitecture, function, and connectivity patterns.[46,47] It has been suggested that the medial frontal pole monitors the relevance of the current goal or behavioral strategy (allowing for undirected exploration of alternative strategies when internal or external contingencies change).[11] In contrast, the FPl supports the monitoring of multiple alternative task sets and goals and implements switching to the best alternative (i.e., directed exploration).[11] This FPl function is supported by the peculiar cytoarchitectonic and connectional fingerprint of this anatomical region. Namely, relative to the rest of the brain, the FPl has connections to other high-order integration areas rather than primary sensory regions, fittingly for controlling domain-general processes.[47,48] There are also microstructural properties, suggesting that FPl has among the highest level of information integration and control possibilities within the PFC. For instance, compared with caudal and midlateral PFC structures, the FPl has a low cell body density, reduced laminar differentiation, and increased spine number along longer dendritic trees[49–51] (see also Refs. 13 and 52 for reviews).

Consistent with these anatomical observations, the FPl has been associated with higher-order cognitive functions that require maintaining representations of alternative courses of action (i.e., "cognitive branching"),[53] such as multitasking behavior,[54,55] prospective memory,[56–58] relational reasoning,[59–61] and arbitrating between model-based and model-free RL.[62]

Evidence for a role of the FPl in monitoring counterfactual choices comes from several neurocognitive studies, including functional magnetic resonance imaging (MRI),[9,63,64] transcranial magnetic stimulation (TMS),[65] and electroencephalogram (EEG),[66] consistently showing FPl involvement during decisions to explore alternative options. During voluntary decision-making, the FPl seems to accumulate evidence in favor of switching to alternative actions and communicates with the mid-intraparietal sulcus for the actual switching.[9] For instance, when presented with two alternative options, the FPl collects evidence in support of the best alternative option, linearly increasing activity with increasing reward probabilities of the best alternative.[9] Encoding the value of the best counterfactual option is essential to enable efficient switching to that option if needed.[9–11,67]

The FPl and its potential role in emotion control

Anatomic and functional evidence suggests that the FPl may play an important role in emotional awareness and emotion regulation.

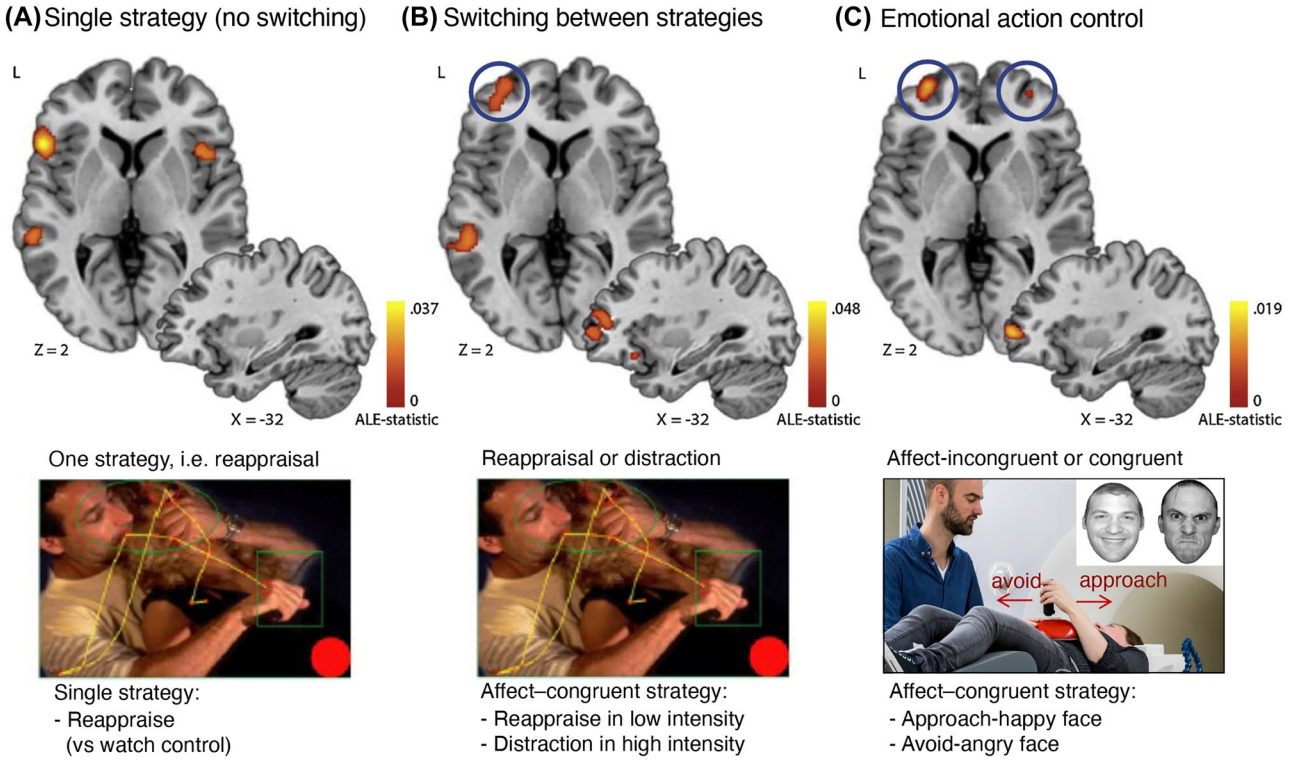

FIGURE 2 The FPl is involved in multiple-strategy emotion control and not during single-strategy emotion regulation. The figure presents the results from a meta-analysis of emotion regulation studies based on single strategy and multiple strategies.[2] The finding that the FPl is involved in emotion control when participants have to switch between different strategies supports the notion that the FPl encodes alternative options during cognitive control. The studies considered in the meta-analysis of Morawetz et al.[8] were separated into two groups: Studies instructing a single strategy ($n = 35$), for which there was absence of evidence for FPl involvement during reappraisal (A); and studies instructing or allowing for alternative strategies ($n = 42$), for which there was evidence for FPl activation during reappraisal (vs. control conditions) when alternative strategies were available, that is, reappraisal in low-intensity emotional conditions and distraction in high-intensity emotional conditions (B).[6] (C) FPl activity was also found when automatic emotional actions needed to be over-ridden with an alternative action ($n = 8$; see Figure 1). Coordinates in MNI stereotaxic space. Figure adapted from Koch et al.[2]

Concerning the former, strong anatomical connections with medial and lateral prefrontal cortices place the FPl in an ideal position to integrate and transmit information about internal states to the rest of the lateral prefrontal cortex (LPFC). This connectivity pattern is well suited to mediate the influence of emotion on goal-directed behavior[14,68,69] and to integrate object, emotional, and self-related information, an integration seen by some theorists as essential to achieve emotional awareness.[14,70] Concerning the latter, the FPl is in the unique position, among LPFC regions, to receive projections from the amygdala via the ventral amygdalofugal fiber bundle.[71–73] We recently showed that stronger structural connectivity of this pathway is associated with a stronger influence of emotion on approach–avoidance actions during an emotional action control task[74] (Figure 1). FPl not only receives emotion-relevant information; there is increasing evidence that it also plays a role in integrating emotional and action goals and controlling emotional responses. A first piece of evidence comes from a study in which inhibition of FPl activity using TMS (continuous theta burst stimulation) amplified the influence of emotions on approach and avoidance behavior, while at the same time resulting in increased activation in the amygdala and decreased activation in posterior parietal cortex (PPC).[75]

Second, and in line with the notion that the FPl can play a role in emotional regulation, meta-analyses found FPl activity during cognitive emotion regulation tasks.[8,12] More precisely, spatially consistent activation was found in the left FPl (BA10) during cognitive reappraisal across 80 studies.[8] A reasonable proportion of the studies (22.5%, $n = 18$ studies) appeared to contribute to this observation, raising the question whether there was something specific with those studies. In light of the role of the FPl in monitoring current and alternative strategies, Koch and colleagues[2] followed up on this observation and conducted a meta-analysis, testing whether the presence of alternative strategies influences FPl contribution to emotional control. When participants performed a single emotional control strategy and had little room to consider alternative options, no significant FPl activity was found (Figure 2). In contrast, when emotional control involved situations that left room for evaluating alternative options, consistent FPl activity emerged across studies.[2] The results confirmed the role of the FPl in this more complex form of emotion regulation, with a striking overlap with BA10 involvement in emotional action control studies. Furthermore, the observation casts fresh light on a finding of an earlier meta-analysis that showed FPl involvement in "late" reappraisal processes.[12] Besides maintaining an emotional control strategy

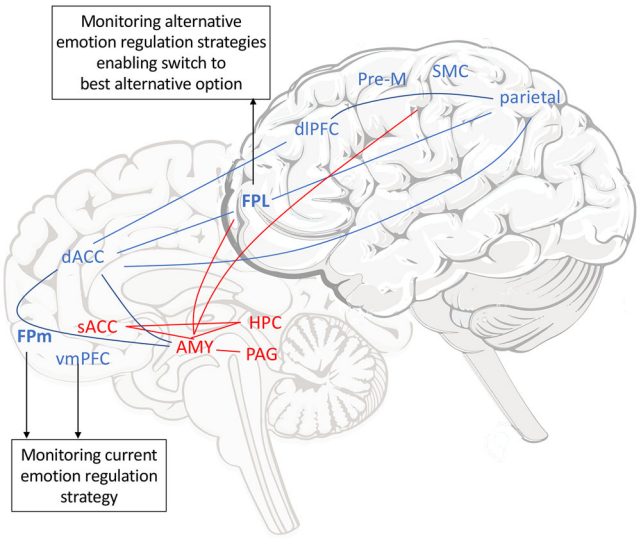

FIGURE 3 Schematic model of Flexible Emotion Control Theory (FECT). The model indicates how the frontal pole (lateral FPl and medial FPm) interacts with emotion regulation circuits. Red areas/lines indicate areas/circuits involved in Pavlovian emotional reactions. Blue areas/lines indicate areas/circuits involved in emotion control by integrating emotion information with action goals. The FPm and ventromedial PFC (vmPFC) are involved in monitoring evidence for the current emotion control strategy. The FPl is implicated in generating predicted outcomes of alternative control strategies. The FPl enables switching to the best alternative option via connections with other frontal control regions (including the dorsolateral PFC [dlPFC], dorsal anterior cingulate cortex [dACC], and the posterior parietal cortex [PPC]). Abbreviations: AMY, amygdala; HPC, hippocampus; PAG, periaqueductal grey; Pre-M, premotor cortex; sACC, subgenual ACC; SMC, sensorimotor cortex.

in working memory, late reappraisal arguably involves monitoring regulation success in the light of alternative strategies. This interpretational framework aligns with recent work examining the representational content of FPl activity during emotion control.[69]

This brings us to the third line of evidence for the role of the FPl in emotion control. In an elegant study, Lapate and colleagues[69] used an affective Go/No-Go task to probe goal-oriented action selection during emotional processing. FPl contained conjunctive emotion–action goal representations that were related to successful cognitive control during emotional processing. These representations differed from conjunctive emotion–action goal representations found in the basolateral amygdala. Action goal representations were also present in mid-lateral PFC, but those were not modulated by emotional valence. FPl emotional valence signals likely originated from the interconnected subgenual anterior cingulate cortex (sACC), which was in turn functionally coupled with the amygdala. Thus, FPl represented integrated emotion–goal information, and received emotional information via amygdala-ACC pathways (see also model presented in Figure 3). These different pieces of empirical evidence call for a fresh evaluation of the neural bases of emotion regulation, a function that, to date, has been predominantly linked to frontoparietal and dorsal midline cortices.[8,76–81] The evidence reviewed here indicates that, when the experimental conditions do not prescribe a single control strategy and instead consider ecologically relevant forms of emotion control, then FPl is causally involved in implementing control. Presumably, the FPl integrates emotions and action goals to continually assess the potential benefit of counterfactual emotion control strategies, communicating switches in strategy to connected circuits that can further implement changes in behavior, such as PPC, sensorimotor cortex (SMC), and sACC.[10,69,75,82,83]

CLINICAL IMPLICATIONS OF THE ROLE OF THE FPl IN EMOTION CONTROL

Impaired emotional control abilities are the hallmark of various psychiatric disorders, such as major depressive disorder (MDD), anxiety disorders, post-traumatic stress disorder (PTSD), and psychopathy.[84–88] Currently available psychotherapies for these disorders, such as cognitive behavioral therapy, largely focus on emotion regulation aspects. However, in the FECT framework, diminished emotional control abilities in psychiatric disorders may reflect the (in)ability to select or switch to contextually more suited emotional control strategies.[42,89] Given the efficacy of currently available psychotherapies,[90–92] it may be a fruitful venue to investigate the (in)ability to switch between emotional control strategies in psychopathology and how to improve this ability within psychotherapy. For instance, in a recent prospective longitudinal study, we showed that FPl activity during emotional action control predicted resilience against the adverse effects of repeated traumatization, buffering the development of PTSD symptoms in police recruits[93] (Figure 4).

Furthermore, FECT has important implications for research on the neural correlates of (impaired) emotional control, both in healthy individuals and in psychopathology. It provides an explanation for apparent inconsistencies in task-based fMRI findings on (deficient) involvement of different prefrontal areas in emotional control. Findings may largely depend on whether alternative emotional control strategies are available or whether only a single strategy is instructed. For example, during the downregulation of negative affect using detachment as a single strategy, impaired amygdala downregulation was found in MDD patients compared to healthy controls,[94] whereas prefrontal recruitment was similar in both groups.[94,95] In contrast, when multiple emotional control strategies were provided, relative over-recruitment of ventrolateral PFC activity[96] as well as reduced downregulation of prefrontal default mode network nodes[97] was observed in MDD patients during downregulation of negative affect. Thus, conclusions on whether emotion regulation deficits in MDD are associated with impaired amygdala downregulation[94] or increased prefrontal recruitment[96,97] may depend on the availability of counterfactual emotional control strategies during task performance as well as the ability to switch between them. Therefore, the FECT framework may be helpful when reviewing the neurocognitive literature on emotion regulation and emotion control deficits in clinical samples.

In line with FECT, several studies have shown that individuals with various affective disorders have reduced FPl activity when engaged in

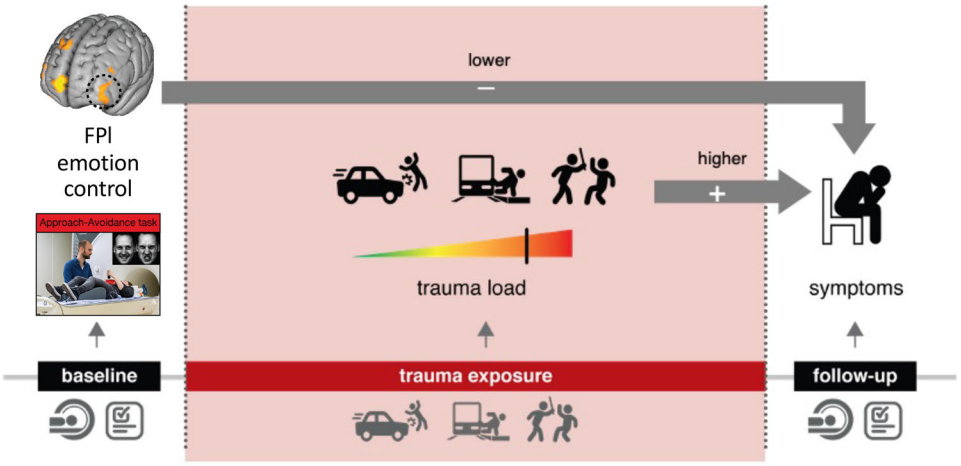

FIGURE 4 Schematic overview of the prospectve longitudinal emotion regulation–resilience study by Kaldewaij et al.[93] A total of 185 police recruits who experienced their core trauma in the line of duty participated in a prospective longitudinal study. They performed the MR-adapted approach–avoidance task, and the neural circuits supporting impulsive and controlled emotional actions (see also Figure 1) were mapped at baseline (before start of police training) and after 1.5 years follow-up. Trauma load was related to significant increase in symptoms of post-traumatic stress disorder (PTSD). This effect was buffered by prefrontal emotion regulation capacity: Higher baseline activity in FPl, as well as in dorsal and medial frontal pole, was related to lower PTSD symptoms after trauma exposure. FPl activity predicted symptoms development over and above self-reported and behavioral measures. These findings suggest that FPl emotion regulation activity predicts increased resilience and may buffer against developing post-traumatic stress symptoms.

emotion control tasks that allow for multiple strategies. For instance, we observed reduced FPl activation in patients with borderline personality disorder and in aggressive delinquents, as well as reduced FPl-amygdala functional connectivity in aggressive delinquents during approach-avoidance tasks where the unchosen option has a higher value.[98–100] Other affective disorders, such as depression or PTSD, have been neurally characterized by hypoactivity of the prefrontal cortex, including the inferior frontal gyrus and ventromedial (vmPFC) parts, and hyperactivity of salience processing areas, including the amygdala and dorsal anterior cingulate cortex.[101–105] This imbalance in activation, in combination with reduced connectivity between the vmPFC, amygdala, and hippocampus, is thought to underlie difficulties in downregulating emotional responses toward threat-provoking or trauma-related stimuli.[106,107] However, there are ample examples of abnormalities of the FPl in PTSD patients, in terms of decreased activation and connectivity[108,109] and decreased cortical thickness.[110,111] Moreover, meta-analytic evidence in patients with PTSD indicated that reduced FPl activation is specific for the affective symptoms during PTSD and not a consequence of trauma exposure in general.[104] Interestingly, a recent study indicates that this reduction in activation can be reversed: successful psychotherapy of PTSD patients was associated with increased activation of the left frontal pole during emotion regulation at follow-up.[112] This frontal region shows remarkable overlap with the FPl region previously identified as predicting resilience toward the negative effects of trauma-exposure in a large prospective longitudinal study among police recruits.[93] Moreover, successful psychotherapy was associated with increased FPl–vmPFC connectivity, and follow-up TMS revealed that this connectivity reflected downstream modulation of the vmPFC by the FPl.[112]

These results, together with previous findings of FPl activity as a resilience factor for both acute and long-term stress-symptoms,[93,113] suggest that standard pathophysiological models of emotion regulation should be adjusted to include the FPl. By the same token, the findings highlight the relevance of devising interventions to optimize FPl functioning. For example, versions of the Approach Avoidance Task (see also Figure 1) have been used to alter emotional action tendencies in socially anxious individuals and alcohol-dependent patients.[114,115] It might prove feasible to boost FPl control functionality using nonemotional tasks, for example, decision-making tasks that require the tracking of the value of the alternative action.[9] The crucial test would involve assessing the generalization of FPl training to situations requiring emotional control in a clinical group, for example, PTSD. Noninvasive brain stimulation may offer another therapeutic avenue. Recently, stimulation by means of transcranial alternating current stimulation (tACS) over the FPl and sensorimotor cortex has been shown to successfully improve performance during an emotional control task[83] (Figure 5). It would be valuable to investigate whether noninvasive brain stimulation could also induce long-term improvement of emotional action control. High-frequency TMS has been applied successfully in therapeutic settings, for example, in depression.[116,117] There is also some evidence for applying repetitive TMS in PTSD, although the number of studies testing its therapeutic efficacy is relatively low.[118] In sum, optimization of FPl functioning is a promising new research avenue, which may eventually lead to new treatment options or even preventive approaches for individuals who are at high-risk for psychopathology and emotion control problems.

DISCUSSION AND OUTSTANDING QUESTIONS

We have presented a neurocognitive theory of flexible emotion regulation (FECT). This theory integrates well-established RL accounts

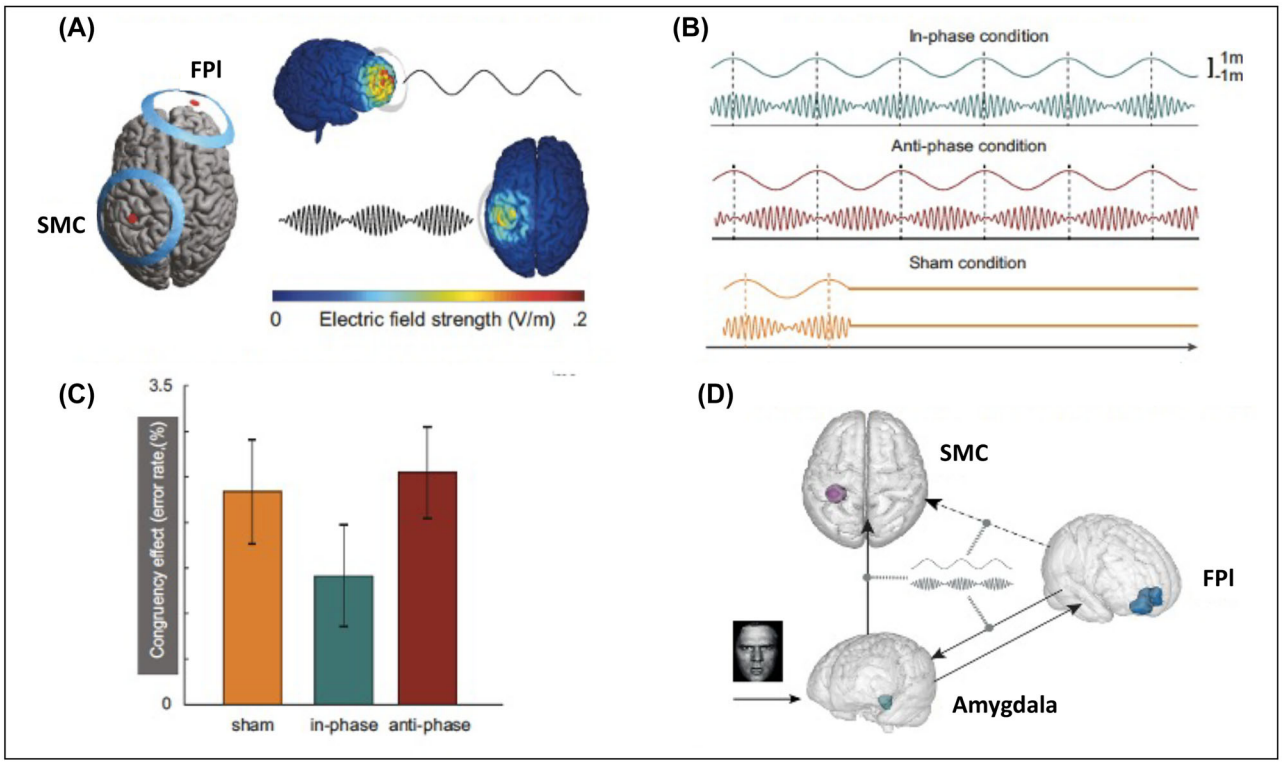

FIGURE 5 Effects of brain stimulation targeting FPl and its interaction with the sensorimotor cortex (SMC) to improve emotional action control. In this study,[83] dual-site phase-coupled electrical brain stimulation was applied to facilitate theta-gamma phase-amplitude coupling between the FPl and the SMC, while participants controlled their automatic action tendencies using the emotional approach-avoidance task (see Figure 1). (A) Two sets of ring electrodes were placed over the right FPl and left SMC. Modeling of the current density indicated that stimulation reached both regions of interest with intensities known to support phase entrainment when matched to the endogenous rhythms.[82] (B) During the experiment, stimulation conditions were alternated between in-phase, anti-phase, and sham conditions in pseudo-random fashion. The 75-Hz stimulation over the SMC was amplitude modulated according to the 6-Hz stimulation over the FPl, either in-phase or anti-phase with the peaks of the 6-Hz aPFC stimulation. Sham consisted of an initial stimulation of 10 s, after which stimulation was terminated. (C) Behavioral effects: Participants with stronger inhibitory responses to theta-band stimulation over the FPl improved their control over emotional actions (decreased congruency effect) during FPl–SMC in-phase tACS (in green) but not during FPl–SMC anti-phase tACS (in red). (D) Modulatory effects of dual-site phase-coupled tACS on emotional action control depend on effective connectivity between the FPl and the SMC. Model selection compared models with and without a direct connection between FPl→SMC (dashed black lines with arrow tips) and tACS modulations on different connections (dashed gray lines with oval tips). tACS affected all nodes in the model, including the amygdala, but only tACS-related changes in connectivity between FPl→SMC predicted behavioral effects of the stimulation. Those participants with stronger inhibitory influence of FPl over SMC in the in-phase condition showed decreases in congruency effects in the in-phase condition but not in the anti-phase and sham condition (see Ref. 83). Thus, participants increased control over their emotional action tendencies, depending on the relative phase and dose of the intervention. Concurrently measured fMRI effects of task and stimulation indicated that the intervention improved control by increasing the efficacy of anterior prefrontal inhibition over the SMC. Figure adapted from Bramson et al.[83]

of emotion control with neurocognitive insights into counterfactual control from decision neurosciences, meta-analytic evidence from affective neurosciences on the role of FPl in emotion control, and insights on the role of the FPl in emotional disorders and their treatment. FECT broadens the space of possibilities considered when exerting control over emotional action tendencies, emphasizing the importance of concurrently handling both active and possible control options. Grounding emotional control into counterfactual reasoning introduces a step-change into current models of emotion control. Counterfactual questions raised by an agent engaged in emotional control ("what if I make that happen?") cannot be articulated, let alone answered by RL models, that is, models that operate purely on the basis of recent statistics of stimuli and responses.[118] Inevitably, FECT also raises a number of interpretational issues and open questions.

The first question relates to the possibility to train FPl function in patients with emotional disorders or individuals at risk. Although tACS over the FPl and sensorimotor cortex has proven to be effective in increasing emotion control by acting on fronto-amygdala-motor circuits in healthy participants,[83] it remains to be tested if such intervention is also effective in clinical samples and whether its effects can outlast the online stimulation period.

Second, our hypothesis on the role of the FPl in monitoring alternative emotional control strategies should be empirically investigated during emotion control tasks. For instance, it remains to be tested

whether the involvement of the FPl in emotional control indeed depends on the number of available emotional control options given contextual demands,[4,5] and whether it reflects monitoring of and/or switching between alternative emotional control options. One could argue that FPl activity may rather reflect domain-general motivation to obtain a specific (emotional control) goal.[119] However, the motivation to change affect is presumably comparable in emotion regulation studies instructing multiple strategies versus a single strategy, rendering this alternative explanation unlikely.

Third, and relatedly, empirical evidence on the role of flexibility in switching between emotion regulation strategies, such as appraisal and distraction, has so far largely been operationalized by strategy-switching to obtain predefined contingencies, for instance, a switch in regulatory preference from choosing distraction under high-intensity pictures to selecting reappraisal under low intensity.[39] It would be relevant for future studies to leave room for more personalized choice patterns, because high and low intensity may not mean the same for each individual, other emotion regulation strategies may be preferred by some individuals, and preferences may change over time. Likewise, congruency effects during emotional action may not be the same for every individual, although this type of control appears to generalize to more symbolic responses mediating appetitive/aversive outcomes, such as those evoked through mannikins and button presses associated with various levels of risks and rewards.[120,121] It remains to be seen whether FECT can explain emotional control across those various behavioral and more symbolic and even linguistically mediated actions.

Fourth, it is important to evaluate the generalizability of FECT to emotional cues other than visually presented faces and arm movements. A first investigation into the crossmodal involvement of FPl in emotion regulation, using visual cues (happy/angry faces) as well as auditory cues (happy/angry vocalizations), has shown that the FPl supports emotional action control in both conditions, coordinating different downstream circuits as a function of the sensory modality of the emotional cue—amygdala for visual cues and insula for auditory cues.[68] However, it remains to be seen whether FPl involvement generalizes across other sensory modalities (e.g., olfactory) and emotional categories (e.g., linguistic prosody, food items). Representational similarity analyses and cross-classification of fMRI or M/EEG signals, as implemented by Lapate and colleagues,[69] might offer the functional resolution necessary to further clarify the conditions evoking FPl involvement in emotional control.

Fifth, it is well known that the FPl continues to develop into early adulthood[122–125] raising questions related to the changing contributions of the FPl to emotional control during human development. It has previously been found that, within same-age adolescents, less developmentally mature participants showed less FPl involvement during emotional action control, compared to more mature peers.[126] The finding raises the possibility that FECT-like control, that is, based on monitoring alternative strategies through FPl, is a late developmental acquisition, presumably constrained by both neurobiological factors (late FPl maturation) and cultural factors (unusually long period of protected cognitive exploration afforded by human social structure). More generally, FPl is also a novel anatomical structure, acquired recently in the human lineage.[45,127] It seems hardly coincidental that this novel anatomical acquisition, supporting a computationally sophisticated control mechanism, is used by a species with a virtually cost-free signaling system, that is, a system that needs tight control over the hypercooperative norms shared across human groups[128,129] and requiring evaluation of long-term consequences of our actions.[130]

Addressing these outstanding questions will be important for understanding the ability of humans to flexibly adapt their emotional control strategies to transitory social environments and to integrate the frequently contrasted domains of emotion and rationality. Most critically, it may help guide the future development of better-tuned interventions to specific problems in emotion regulation in mental disorders, where emotion control goes awry.

AUTHOR CONTRIBUTIONS

K.R. drafted the manuscript; K.R., B.B., and I.T. were involved in the conceptualization, writing, and revision of the manuscript.

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COMPETING INTERESTS

The authors declare no competing interests.

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