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The Neural Basis of Emotion Regulation: A Review of Prefrontal-Limbic Mechanisms and Their Implications for Psychotherapy

Kuan Jiang

Department of Psychology, University of Michigan, Ann Arbor, USA
kuanj@umich.edu

Abstract. Emotion regulation involves the strategies by which people adjust the strength, time course, or display of emotional responses. This review integrates psychological theory, functional neuroimaging, meta-analytic evidence, and intervention studies to characterize the neurocognitive basis for emotion regulation,which emerges from dynamic interactions among distributed brain networks. Regulation depends on context-sensitive activity in the amygdala, insula, and cingulate cortex during emotion generation, whereas intentional control engages lateral and medial prefrontal regions that maintain goals, interpret events, and shift attention or meaning. Effective regulation therefore relies on information integration rather than the inhibition of one brain region by another. Depression and anxiety involve maladaptive regulation patterns and ineffective prefrontal-limbic interaction, although these alterations are heterogeneous and non-specific to particular diagnoses. Cognitive behavioral therapy, exposure therapy, and mindfulness-based interventions may alter appraisals, salience, self-referential processing, and neural connectivity. However, small samples, heterogeneous tasks, and the correlational nature of functional imaging constrain causal and clinical inference. Future work should adopt integrated, longitudinal, multimodal, and ecologically valid designs to identify individual-level predictors of treatment outcomel.

Keywords: Emotion regulation, prefrontal-limbic circuitry, cognitive reappraisal, psychotherapy, network connectivity

1. Introduction

Emotions shape attention, memory, physiology, and behavior, but they can become maladaptive when they are excessively intensive, persistent, or incongruent with the situation. Emotion regulation therefore involves more than the suppression of unpleasant affect. Rather, it involves a range of processes that affect the occurrence, timing, experience, and expression of emotion. Antecedent-focused strategies such as reappraisal have been contrasted experimentally with response-focused strategies such as expressive suppression, and ostensibly similar strategies have shown to produce divergent experiential and physiological consequences [1]. This functional

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approach has placed emotional regulation at the center of research on adaptive functioning and psychopathology.

A neuroscientific approach offers an explanatory level of analysis by specifying how goals and appraisals operate in networks of brain systems. Current theories link the cognitive mechanisms of prefrontal and cingulate cortices with sensory, semantic, evaluative, and autonomic networks of emotional processing [2, 3]. They differentiate model-based and implicit learning approaches to regulation [4]. Nonetheless, the literature is sometimes reduced to a dichotomy between a "rational" prefrontal cortex and an "emotional" amygdala. This framing neglects indirect connectivity, strategy-specific processes, temporal dynamics, and network-level effects. This review therefore examines the theory of emotion regulation, the neural circuits implicated in its implementation, the contribution of dysregulation to depression and anxiety, and the potential of psychotherapy to modify these processes. Emphasis is placed on convergent findings, methodological limitations, and the gap between group-level imaging results and individual-level prediction of clinical outcome.

2. Conceptual foundations of emotion regulation

2.1. Defining emotion regulation and its adaptive function

The process model locates regulatory opportunities along the unfolding emotional response: situations may be selected or modified, attention may be redirected, interpretations may be changed, and responses may be adjusted after they emerge. In Gross' classic study, both suppression and reappraisal reduced expressive behavior, but only reappraisal decreased self-reported disgust, whereas suppression increased sympathetic activity [1]. These findings show that observable behavior alone does not indicate successful regulation. The adaptiveness of a strategy instead depends on timing, goals, context, and the response system involved. Regulation should therefore be evaluated as flexible coordination rather than a fixed division between "good" and "bad" strategies.

2.2. From psychological models to neurobiological models

Psychological constructs become tractable research questions in neuroscience when their underlying processes are composed into operations that can be manipulated and quantified. For instance, reappraisal involves representing a goal, attending to features that are important, retrieving knowledge, constructing a new interpretation, and updating the value of a stimulus. Such operations provide testable predictions regarding the activation and connections between control, language, memory, and affective networks. Neurobiological theories therefor postulate that lateral and medial prefrontal networks do not generate regulatory commands themselves, rather, they configure other networks according to the selected goal and strategy [2, 3].

2.3. Explicit and implicit regulation

Explicit regulation is goal-directed and operates through deliberate intent, as when a person consciously attempts to reappraise an aversive situation. Implicit regulation, by contrast, does not require deliberate intent and encompasses processes such as adaptation to emotional conflict, extinction learning, and habitual value updating. Explicit regulation is described by Etkin et al. as model-based and involving frontoparietal networks carrying out internal models of desired goals, while implicit regulation is based on ventral anterior cingulate and ventromedial prefrontal networks influenced by reinforcement learning [4]. The contrast is graded rather than absolute: explicit

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strategies can become automatized with practice, and implicit processes may be favoured in certain contexts because of their greater expected value. This gradient is clinically significant because treatment modifies not only explicit coping strategies but also automatic predictions that persist beyond the treatment period.

3. Neural circuits and mechanisms of emotional control

3.1. Emotion-generative and salience systems

Emotional reactions are generated through distributed networks that assess significance from both internal and external sources. The amygdala is critical for evaluating the relevance of biologically significant stimuli and for learning about them, the insula encodes bodily state and subjective significance, the dorsal cingulate cortex supports action preparation and conflict monitoring, and the ventral striatum and medial prefrontal cortex contribute to value evaluation. Memory and sensory mechanisms provide the context through which a stimulus will be seen as dangerous, rewarding, or irrelevant. None of these brain regions make up an emotion center per se, and their roles depend on the particular context or task [3, 4]. Amygdala activation, for instance, may reflect uncertainty or salience rather than fear alone. Regulation can therefore influence emotion through routes other than reducing amygdala activity.

3.2. Prefrontal and cingulate control systems

The dorsolateral and posterior prefrontal cortex supports the maintenance of regulatory goals and the manipulation of information in working memory. The ventrolateral prefrontal cortex is involved in selecting among interpretations and inhibiting competing representations, whereas the dorsomedial prefrontal and anterior cingulate cortices monitor conflict, mental states, and the necessity of control. The ventromedial prefrontal cortex integrates value and context and provides an indirect route through which cortical control influences amygdala and autonomic activity. This specialization is not absolute, because these regions also participate in numerous non-emotional tasks [2-4].

3.3. Prefrontal-limbic interaction in emotion regulation

Early fMRI studies provided evidence of correlated changes in the cortex and subcortex. Ochsner et al. found that, in 15 healthy young women, reappraising negative images reduced self-reported negative affect, increased lateral and medial prefrontal activation, and decreased amygdala and medial orbitofrontal activity [5]. Although the small, homogeneous sample limits generalization, these findings support the hypothesis that emotional appraisal is modulated in a top-down manner. A second study of 30 healthy participants used mediation analysis to identify two pathways associated with successful reappraisal. Right ventrolateral prefrontal cortex activity predicted changes via amygdala and ventral striatum, where the two pathways were linked differently with negative and positive appraisals [6]. Mediation of BOLD signal change cannot be taken as evidence of a causal relationship between brain regions.

3.4. Strategy-specific neural mechanisms

Convergence evidence is strongest for cognitive reappraisal, the most frequently studied strategy. A meta-analysis of 48 imaging studies found reliable recruitment of cognitive-control and lateral temporal regions together with amygdala modulation, but no reliable ventromedial prefrontal

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involvement [7]. This suggests an underlying mechanism whereby control systems manipulate semantic representation and affective responses as a consequence. Another meta-analysis revealed a network of superior temporal and angular gyri, supplementary motor regions, dorsolateral and ventrolateral prefrontal cortex, and anterior midcingulate cortex [8]. The discrepancy between these two meta-analyses reflects differences in inclusion criteria and analytic models, but it nonetheless challenges any single-region account. Hence, it is necessary to specify the neural mechanisms of reappraisal, suppression, distraction, acceptance, and extinction independently before making claims regarding "emotion regulation" generally.

3.5. Network-based integration of emotion regulation

Network models reconcile apparent inconsistencies by treating regulation as an interaction among control, salience, semantic, default-mode, and valuation systems. Lateral prefrontal regions can maintain goals without direct anatomical projections to the amygdala, influence may be relayed through medial prefrontal, cingulate, temporal, or striatal nodes [3, 4, 8]. What matters functionally is the alteration of coupling, timing, or representational content, even when activation levels within a given region remain stable. Salience systems provide information about the need for control, control systems sustain the task set, and semantic or valuation systems implement the altered appraisal. Finally, feedback of bodily and affective systems informs whether the effort should continue. A network approach explains both why identical instructions produce different activity patterns across individuals and why successful regulation can increase rather than decrease activity in emotion-processing systems.

4. Emotion dysregulation in psychopathology

4.1. Emotion dysregulation as a transdiagnostic mechanism

A meta-analysis of 114 studies and 241 effect sizes found that several habitual strategies were significantly associated with anxiety, depression, eating disorders, and addictive behaviors [9]. Rumination showed the strongest association, avoidance, suppression, and poor problem-solving were also significantly associated, whereas reappraisal and acceptance showed weak associations [9]. These findings illustrated that dysregulation is a transdiagnostic factor, although they do not establish causation. Most of the data relied on self-reported habits and associations very well across samples, including clinical and non-clinical populations.

4.2. Altered prefrontal-limbic coordination in depression and anxiety

Circuit pathology could arise from poor coordination rather than underactivation of control centers. In a cohort of 21 unmedicated adults with major depressive disorder and 18 control participants, intentional down-regulation activated the left ventrolateral prefrontal cortex in controls but was more bilateral in the depressed group. In controls, ventromedial prefrontal-amygdala coupling was inverse and mediated the relationship between lateral prefrontal activation and reduced amygdalar activation. In the depressed group, by contrast, this coupling was positive, and greater effort was correlated with increased amygdala, insular, and thalamic activation [10]. This pattern is consistent with compensatory recruitment, although a single cross-sectional study cannot establish that the altered coupling is causal. Similarly, theoretical models of anxiety emphasize heightened salience and threat processing relative to controls.

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4.3. From neural dysregulation to clinical symptoms

Neural dysregulation can provide links between symptoms that cut across diagnostic categories. Threat prediction can underlie hypervigilance and avoidance, lack of updating can maintain fear after danger subsides, self-referential semantic processing can maintain rumination, and deliberate but malcoordinated regulation can give rise to tiredness or exhaustion. Such correspondences are still probabilistic, as the same symptoms can arise via different pathways and the same neural signatures can be seen in several different disorders [9, 10]. Impaired regulation may reflect not only an inability to implement chosen strategies but also an inability to select them. A clinically useful model of regulation must therefore integrate circuitry, strategy selection, context, behavior, physiology, and symptom course rather than treating a brain image in isolation.

5. Implications for psychotherapy and future research

5.1. Cognitive-behavioral therapy and reappraisal-related plasticity

Cognitive-behavioral therapy (CBT) directly trains operations implicated in reappraisal: identifying automatic interpretations, testing beliefs, shifting attention, and constructing alternatives. In a randomized controlled trial of 75 individuals with generalized social anxiety disorder, Goldin et al. reported that 16 sessions of individual cognitive behavioral therapy reduced the intensity of negative emotion during both reactivity and reappraisal [11]. Therapy also increased medial and dorsolateral prefrontal activity, produced earlier dorsomedial prefrontal activation, and strengthened inverse connectivity between the dorsomedial prefrontal cortex and the amygdala [11]. Amygdala signal intensity was unchanged, suggesting that timing and coupling are more sensitive measures than regional activation.

5.2. Neural plasticity and mechanisms of psychotherapeutic change

Treatment-imaging syntheses show both convergence and important inconsistency. A meta-analytic study of 16 studies including 193 patients identified effects of psychotherapy on the dorsomedial prefrontal, posterior midline, and temporal regions, although exposure treatment for phobia was associated with reduced medial temporal reactivity, the expected effects on the dorsolateral prefrontal and amygdala activity were inconsistent [12]. A systematic review of 19 fMRI studies, including 509 patients with anxiety and related disorders, concluded that CBT might increase control over disorder-specific subcortical circuitry but noted substantial methodological heterogeneity [13]. A subsequent meta-analysis of 22 longitudinal studies found consistent post-treatment reductions in anterior cingulate/paracingulate, inferior frontal, and insular activation, however, pretreatment predictors derived from 11 studies did not yield a reliable individua-level marker [14]. Factors such as diagnosis, medication, scanner task, psychotherapy format, and analytic threshold may each alter the direction of effect. Psychotherapy may enhance efficiency, recalibrate salience, modulate self-referential processing, and normalize task-related responses, improvement cannot be attributed simply to "more prefrontal, less limbic" activity.

5.3. Mindfulness, acceptance, and bottom-up awareness

Mindfulness-based interventions go beyond deliberate reinterpretation. Mechanistically, Hölzel et al. propose four interacting components: attentional regulation, interoception, emotion regulation through reappraisal or exposure learning, and a changed perspective on the self. These components

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have been linked with the anterior cingulate cortex, insula, temporoparietal junction, frontolimbic network, and default mode networks [15]. Clinically, sustained monitoring of bodily and mental sensations may reduce avoidance and permit extinction or reconsolidation without requiring cognitive disputation of each emotion. Nevertheless, the evidence remains preliminary and largely cross-sectional, a neuroplasticity account requires a randomized, controlled longitudinal design.

5.4. Translational implications and research priorities

Current data favor mechanism-based intervention over intervention selection based on imaging markers alone. Future research should pre-register adequately powered longitudinal designs, harmonize tasks and outcome measures, include active treatment control conditions, and assess both symptoms and mediators. Repeated measures are needed to determine whether neural changes precede, accompany, or follow symptom remission. Dynamic connectivity, temporal metrics, autonomic measures, and ecological momentary assessment can test whether laboratory findings generalize to real life. Comparative effectiveness studies are needed, as are studies addressing development, culture, medication, and comorbidity. Individual prediction models require external validation, unbiased performance metrics, and comparison with low-cost clinical predictors. Perturbation experiments and mediation analyses can help distinguish treatment mechanisms from correlated non-specific factors. Unless these criteria are met, neural markers may serve only to generate hypotheses about intervention [11-15]. The most plausible clinical target is flexible coordination among networks, tailored to the patient's context and sustaining processes.

6. Conclusion

Emotion regulation is implemented through multiple brain systems rather than a single cortical structure inhibiting a limbic generator. Psychological theories specify when regulation can occur and distinguish processes with divergent outcomes. Neuroscience complements this account by identifying the operations involved in goal maintenance, meaning selection, conflict monitoring, salience representation, and value updating. Converging empirical and meta-analytical evidence indicates that cognitive reappraisal engages lateral and medial prefrontal, cingulate, and temporal regions while modulating amygdala and striatal responses through both direct and indirect pathways.

This network approach helps us understand psychopathology and psychotherapy. Depression and anxiety are characterized by rigid regulatory strategies and impaired interaction between control and emotion-generation networks, although such findings are transdiagnostic, heterogeneous, and mostly correlational. Cognitive behavioral therapy strengthens or accelerates appraisal-based control processes, exposure therapy revises threat associations, and mindfulness training enhances attention, interoception, and acceptance while reducing the rigidity of self-referential processing. Treatment-induced reductions in prefrontal or limbic activation may reflect increased efficiency, just as increased activation may reflect enhanced engagement.

The clinical promise of this research lies in specifying change mechanisms and improving prediction, not in assigning disorders or therapies to isolated regions. Current group-level research can generate plausible mechanistic hypotheses, however, small samples, inconsistent paradigms, and limited external validity currently prevent the clinical application of any marker. Future research must be longitudinal and multimodal, linking neural change to behavior, physiology, subject experience, and symptom reduction. Ultimately, emotion regulation must be viewed as a function of context-dependent flexibility.

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