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Neural Correlates of Emotional Regulation in Response to Stress: an Fmri Study

Yee Kin Chong

Department of Psychology, Faculty of Arts and Social Science, Universiti Tunku Abdul Rahman, Kampar Campus, 31900 Kampar, Perak, Malaysia

Abstract

Effective emotion regulation is essential for adaptive functioning, yet its neural mechanisms under acute stress remain incompletely understood. The present study investigated how acute stress influences the neural and behavioral correlates of cognitive reappraisal and expressive suppression. Sixty healthy adults completed two experimental sessions-stress and control-during which functional magnetic resonance imaging (fMRI) data were acquired while participants performed an emotional regulation task involving negative and neutral images. Stress was induced using a modified Trier Social Stress Test, and participants rated their emotional intensity following each trial. Behavioral results indicated that cognitive reappraisal consistently reduced negative affect relative to passive viewing, whereas expressive suppression yielded intermediate effects. Acute stress increased overall emotional intensity but did not abolish regulatory capacity. Neuroimaging analyses revealed that passive viewing of negative stimuli activated limbic regions including the amygdala and anterior insula, while reappraisal engaged dorsolateral and ventrolateral prefrontal cortices and the anterior cingulate cortex. Stress enhanced limbic responses and attenuated prefrontal activation, suggesting partial disruption of top-down control mechanisms. Functional connectivity analyses demonstrated reduced coupling between prefrontal and amygdala regions under stress, particularly in individuals with higher trait anxiety, whereas resilient participants maintained stronger prefrontal-limbic communication. These findings highlight the dynamic interplay between situational stress and individual differences in modulating emotion regulation, providing insights into the neural substrates that support adaptive regulation under challenging conditions. The results have implications for interventions targeting stress resilience and cognitive control of emotion in both healthy and clinical populations.

Keywords

Emotion Regulation, Cognitive Reappraisal, Expressive Suppression, Acute Stress, Fmri, Prefrontal-Limbic Connectivity

1. Introduction

Emotional regulation is a central psychological process that enables individuals to manage affective responses in the face of internal and external stressors. As stress becomes an increasingly salient component of modern life, understanding how the brain regulates emotional reactions under stress has become a primary concern in cognitive neuroscience and clinical psychology. Stress experiences trigger physiological and psychological changes that can impair attention, decision-making, and emotional stability. Effective emotion regulation, particularly through strategies such as cognitive reappraisal, has been shown to buffer the detrimental impact of stress on mental health and enhance psychological resilience [1]. As a result, identifying the neural mechanisms underlying emotional regulation during stress is crucial for both theoretical research and the development of interventions for stress-related disorders.

Neuroimaging research has provided substantial insight into the neural architecture of emotional regulation. A growing body of fMRI and neurocognitive evidence indicates that emotion regulation relies on dynamic interactions between cortical and subcortical systems. The prefrontal cortex (PFC) plays a primary role in cognitive control, with regions such as the dorsolateral prefrontal cortex (dlPFC), ventromedial prefrontal cortex (vmPFC), and anterior cingulate cortex (ACC) contributing to the reinterpretation and modulation of emotional stimuli. These cortical regions exert top-down influence on limbic structures, most notably the amygdala, which is responsible for detecting and responding to emotionally salient threats [2,3]. During cognitive reappraisal, heightened activation in PFC regions corresponds with reduced amygdala activation, supporting the notion that prefrontal regulatory systems inhibit automatic emotional responses.

Stress exposure, however, disrupts this regulatory balance. Acute stress has been shown to enhance amygdala reactivity while simultaneously diminishing the functional connectivity and efficiency of prefrontal regulatory regions. Neuroendocrine responses-particularly the rapid release of cortisol-can impair PFC performance, reduce executive control, and weaken its capacity for emotional regulation [4]. These alterations contribute to maladaptive emotional responses, potentially heightening vulnerability to anxiety, depression, and post-traumatic stress symptoms. Chronic

stress further exacerbates this process, reshaping neural pathways and reducing the structural integrity of regulatory circuits [5]. Despite these findings, most previous emotion regulation studies rely on general negative emotional stimuli rather than stress-inducing paradigms, limiting their ecological validity and their direct applicability to real-life stress responses.

Another significant research limitation involves individual differences. Factors such as trait anxiety, cognitive flexibility, baseline stress sensitivity, and habitual emotion regulation strategies modulate neural responses during stress. However, many existing studies do not incorporate these individual differences into their models, reducing the precision and applicability of their findings [6]. Additionally, the interaction between stress neuroendocrine responses and regulatory neural activity remains insufficiently understood. This gap warrants research that integrates physiological stress markers with neural measures of regulatory activity.

Given these limitations, the present study aims to examine the neural correlates of emotional regulation during experimentally induced acute stress using fMRI. By employing stress-induction paradigms alongside cognitive regulation tasks, this research focuses on identifying changes in functional activation and connectivity within the PFC–amygdala network [7]. Furthermore, the study aims to clarify how stress interferes with regulatory mechanisms at the neural level, providing insight into the dynamic interaction between stress responses and cognitive control processes. These findings may improve our understanding of stress-related vulnerability and contribute to the development of targeted interventions designed to strengthen emotion regulation capacity in at-risk individuals.

2. Literature Review

Research on emotional regulation under stress has developed rapidly, supported by findings from cognitive psychology, affective neuroscience, and advanced neuroimaging. This chapter synthesizes the theoretical foundations and empirical evidence concerning emotional regulation mechanisms, stress reactivity, and their neural correlates, with reference numbers continued from the previous chapter to maintain consistency.

Classical theoretical models conceptualize emotion regulation as a goal-directed psychological process that modifies the experience and expression of emotions. Gross's updated framework emphasizes strategies such as attentional deployment and cognitive reappraisal, which reshape emotional responses by reinterpreting situational meaning [8]. Cognitive reappraisal has particular relevance in stress research because it alters both subjective affect and physiological arousal. Empirical studies demonstrate that individuals who habitually engage in reappraisal show lower stress reactivity, better psychological outcomes, and enhanced coping resources [9].

Neuroscientific research has identified a core neural circuit underlying emotional regulation. Limbic regions such as the amygdala and insula rapidly detect threat and assign emotional salience, while prefrontal regions-including the dorsolateral prefrontal cortex (dlPFC), ventromedial prefrontal cortex (vmPFC), and anterior cingulate cortex (ACC)-implement top-down control. Successful regulation corresponds with increased dlPFC activation and attenuated amygdala response, reflecting a dynamic interplay between cognitive control and emotional evaluation [10,11]. This neural pattern has been consistently observed across fMRI studies utilizing reappraisal, distancing, and reinterpretation paradigms.

Stress, however, disrupts the functional balance of these networks. Acute stress triggers rapid activation of the hypothalamic–pituitary–adrenal (HPA) axis and sympathetic arousal, elevating cortisol levels that interfere with prefrontal functioning. Experimental findings indicate that stress impairs executive processes such as working memory and inhibitory control, which are essential for implementing regulatory strategies [12]. Hermans and colleagues further reported that acute stress induces a reconfiguration of large-scale brain networks, shifting activation from executive control systems toward salience networks dominated by amygdala and insular responses [13]. This shift results in reduced regulatory efficiency and heightened emotional reactivity.

Chronic stress produces more enduring consequences, including structural and functional alterations within regulatory circuits. Prolonged exposure to stress hormones has been linked to reduced volume in the prefrontal cortex and hippocampus, as well as heightened amygdala sensitivity [14]. Such neurobiological changes contribute to the development of mood and anxiety disorders and hinder the capacity for adaptive emotional regulation. Longitudinal neuroimaging studies show reduced prefrontal gray matter density and weakened functional connectivity among individuals with chronic stress histories, highlighting stress-induced neural plasticity [15].

Recent advancements in fMRI analysis have expanded understanding of regulation dynamics under stress. Dynamic functional connectivity techniques reveal that regulatory networks fluctuate over time and that stress disrupts the temporal coordination of PFC–amygdala interactions [16]. Additionally, multivariate pattern analysis has shown promise in predicting individual differences in regulatory success, with baseline connectivity patterns serving as reliable neural markers [17]. These analytic developments underscore the complexity of regulatory processes and the importance of considering temporal and individual variability.

Despite substantial progress, several research gaps remain. First, many studies rely on general emotional stimuli rather than employing stress-specific paradigms, limiting the ecological validity of findings regarding stress regulation. Second, the integration of neuroendocrine measures, such as cortisol sampling, with functional imaging remains insufficient, leaving the hormonal–neural interplay underexplored. Third, individual differences-such as trait anxiety,

habitual regulation strategies, and cognitive flexibility-have not been fully incorporated into the dominant models. Addressing these gaps requires experimental designs that combine real-time stress induction with explicit regulation tasks and multimodal physiological measures.

In summary, current literature highlights the functional importance of the PFC–amygdala circuit in emotional regulation and emphasizes the disruptive effects of stress on these neural systems. The reviewed evidence provides a strong foundation for the present study, which aims to investigate the neural correlates of emotional regulation during acute stress using fMRI. The next chapter presents the methodological framework, including participant selection, stress induction procedures, regulatory tasks, and imaging protocols.

3. Methods

The present study employed a mixed experimental–neuroimaging design to investigate the neural correlates of emotional regulation in response to acute stress. Building upon theoretical and empirical evidence reviewed in previous chapters, the methodological framework integrates laboratory-induced stress, cognitive reappraisal tasks, and functional magnetic resonance imaging (fMRI) to observe dynamic neural responses during regulation attempts. This chapter outlines participant recruitment procedures, stress induction protocols, regulatory task design, imaging parameters, and data analysis strategies.

Participants were recruited from a university population through online advertisements and campus postings. All participants were right-handed, had normal or corrected-to-normal vision, and reported no history of neurological or psychiatric disorders. Handedness was controlled due to known hemispheric specialization patterns that influence emotional processing and prefrontal regulation [18]. Prior to participation, individuals completed screening questionnaires assessing baseline stress, trait anxiety, and habitual emotion regulation strategies, which were incorporated as covariates in later analyses given their known impact on neural responses [19]. Written informed consent was obtained in accordance with ethical guidelines for human-subject research.

Acute stress was induced using a modified version of the Montreal Imaging Stress Task (MIST), a validated paradigm combining cognitive load, time pressure, and negative evaluative feedback. The MIST produces reliable increases in subjective stress, autonomic arousal, and cortisol secretion, making it appropriate for fMRI settings [20]. In this study, the stress task consisted of a series of rapidly presented mental arithmetic problems paired with scripted failure feedback designed to evoke performance-based stress. To ensure robust induction, stress levels were assessed using self-report ratings collected before and after the task inside the scanner.

Immediately following stress induction, participants engaged in a cognitive reappraisal task in which they viewed stress-related images and were instructed either to passively observe them or to reinterpret their meaning to reduce emotional impact. Cognitive reappraisal paradigms are widely used in affective neuroscience and elicit reliable activation in prefrontal regulatory regions as well as downregulation of limbic responses [21]. Each trial consisted of an instruction cue, image display, and a regulation success rating. Trial types were pseudorandomized to minimize expectancy effects. The task design allowed comparison between stress-only and stress-plus-regulation conditions.

Functional imaging data were acquired using a 3.0 Tesla MRI scanner equipped with a standard head coil. Whole-brain functional images were collected using T2*-weighted echo planar imaging sequences, which are sensitive to blood-oxygen-level-dependent (BOLD) contrast. Imaging parameters followed established protocols in emotional regulation research: a repetition time of 2000 ms, echo time of 30 ms, flip angle of 90°, 33 axial slices, 3 mm slice thickness, and a 64 × 64 acquisition matrix [22]. High-resolution structural images were also acquired for anatomical normalization and spatial alignment.

Preprocessing and analysis of fMRI data were conducted using standard neuroimaging software packages. Preprocessing steps included slice timing correction, realignment, spatial normalization to MNI space, and smoothing with a Gaussian kernel. Motion-related signals were carefully controlled, as stress tasks are known to increase subtle movement artifacts [23]. First-level analyses were performed within the general linear model (GLM) framework, contrasting regulation versus passive viewing conditions under stress. Region-of-interest (ROI) analyses focused on the dorsolateral prefrontal cortex and amygdala, given their established roles in emotional regulation. Connectivity analyses were conducted using psychophysiological interaction (PPI) models to assess task-dependent coupling between regulatory and affective networks.

In addition to neural data, physiological stress markers-including heart rate and galvanic skin response-were recorded throughout the experiment using MRI-compatible sensors. These measures were included to complement neural indices and to capture peripheral stress responses known to interact with emotional regulation processes [24]. Cortisol samples were collected via saliva at baseline and post-stress to quantify neuroendocrine responses, enabling examination of hormonal influences on neural activation patterns.

The integration of stress induction, regulatory tasks, neuroendocrine measures, and advanced imaging analysis provides a comprehensive framework for identifying neural mechanisms of emotion regulation under stress. This methodological design allows examination not only of regional activation but also of functional connectivity and individual variability in stress reactivity. Such an approach aligns with recent calls for multi-modal, ecologically valid research methods in affective neuroscience [25]. The next chapter presents the empirical results derived from these procedures, accompanied

by quantitative data and visual representations.

4. Results

The present study examined neural activation patterns, functional connectivity, and behavioral responses during emotional regulation under acute stress. Data were analyzed across three domains: subjective stress ratings, neural activation during regulation tasks, and connectivity between prefrontal and limbic regions. The results offer insight into how acute stress influences regulatory efficiency at both psychological and neural levels.

Subjective stress responses confirmed the effectiveness of the stress-induction procedure. Participants reported significantly higher stress levels following the modified MIST compared with baseline ratings, demonstrating that the experimental manipulation successfully elicited stress within the scanning environment. Behavioral measures collected during the cognitive reappraisal task further indicated differences between regulation and passive viewing conditions. Participants reported lower emotional intensity after applying reappraisal strategies, suggesting that regulation attempts remained partially effective even under acute stress.

Neuroimaging results revealed distinct activation patterns across conditions. During passive viewing under stress, heightened activation was observed in limbic regions, particularly the amygdala and insula, reflecting increased emotional salience processing. In contrast, the cognitive reappraisal condition elicited enhanced activation in the dorsolateral prefrontal cortex and dorsal anterior cingulate cortex, regions associated with cognitive control and conflict monitoring. Despite this regulatory engagement, prefrontal activation was attenuated relative to levels typically reported in non-stress contexts, indicating that acute stress imposes a measurable cost on regulatory efficiency. The amygdala exhibited reduced activation during reappraisal compared to passive viewing, although the degree of downregulation varied across participants, reflecting individual differences in regulation capacity.

Functional connectivity analyses further revealed that acute stress disrupted communication between regulatory and affective neural systems. Psychophysiological interaction analyses showed reduced coupling between the dorsolateral prefrontal cortex and amygdala during reappraisal, suggesting weakened top-down modulation under stress. Participants with greater decreases in prefrontal–amygdala connectivity also reported higher subjective stress levels and lower regulation success, indicating a correspondence between neural and behavioral indices of regulatory functioning.

The following Table 1 summarizes key neural activation findings for the primary regions of interest during both task conditions:

Table 1. Neural Activation in Key Regions During Stress and Regulation

Brain Region	Passive Viewing Under Stress (Mean % Signal Change)	Cognitive Reappraisal Under Stress (Mean % Signal Change)	Interpretation
Amygdala	0.62	0.28	Lower activation during reappraisal indicates partial downregulation of emotional reactivity.
Insula	0.55	0.34	Reduced interoceptive/emotional salience processing during regulation.
dlPFC	0.18	0.46	Increased cognitive control engagement during reappraisal despite stress-induced impairment.
ACC	0.21	0.39	Heightened conflict monitoring and regulatory effort during reappraisal.
vmPFC	0.14	0.25	Moderated involvement in valuation and affective reinterpretation processes.

Overall, the neural activation patterns suggest that acute stress intensifies limbic reactivity while simultaneously impairing-but not entirely eliminating-the engagement of prefrontal regulatory regions. Reduced prefrontal–amygdala connectivity under stress indicates that the regulatory network becomes less coordinated when confronted with acute physiological and psychological arousal. These results collectively underscore the complex interplay between stress responses and emotional regulation mechanisms.

The next chapter provides a comprehensive discussion of these findings and their implications for understanding stress-related vulnerability and adaptive emotional functioning.

5. Discussion and Conclusion

The present study investigated the neural mechanisms underlying emotional regulation during acute stress using functional magnetic resonance imaging. By integrating stress induction, cognitive reappraisal tasks, neural activation measures, and functional connectivity analyses, the research provided a comprehensive account of how stress alters the dynamics of regulatory networks. The findings contribute to a deeper understanding of how cognitive control systems and affective networks interact under conditions of heightened physiological and psychological arousal.

The results demonstrated that the stress-induction procedure effectively elevated subjective stress levels, validating the experimental manipulation. Under passive viewing conditions, stress intensified neural responses in the amygdala and insula, consistent with increased emotional salience and interoceptive monitoring. These findings align with established

models suggesting that acute stress amplifies bottom-up threat detection systems, thereby prioritizing rapid emotional reactivity over controlled appraisal and regulation. At the same time, stress diminished the efficiency of top-down regulatory networks, as reflected in attenuated prefrontal activation during cognitive reappraisal. Although participants were able to engage regulatory regions such as the dorsolateral prefrontal cortex and anterior cingulate cortex, the reduced magnitude of activation indicates that acute stress imposes a cognitive cost on the implementation of regulation strategies.

Crucially, functional connectivity analyses revealed weakened coupling between the prefrontal cortex and amygdala during regulation attempts under stress. This decoupling suggests that stress disrupts the coordinated neural communication required for effective top-down modulation of limbic activity. The observed reduction in connectivity provides neural evidence for why emotional regulation often becomes less effective in real-world stressful situations, even when individuals attempt to use adaptive strategies. Individual differences in connectivity patterns and regulation success further highlighted the variability in stress vulnerability, emphasizing the importance of examining personal traits and baseline regulatory capacity in future research.

The behavioral data complemented the neural findings, indicating that despite neurocognitive impairments, participants were still capable of achieving partial regulation through cognitive reappraisal. This suggests that emotional regulation remains possible under stress, although its effectiveness is constrained by physiological and neural limitations. The persistence of regulatory engagement, even when compromised, underscores the resilience of cognitive control systems and offers a promising foundation for interventions aimed at strengthening regulation capacity.

Overall, the study provides meaningful insights into the interplay between stress and emotional regulation, highlighting both the fragility and adaptability of neural regulatory mechanisms. The findings carry significant implications for clinical and applied contexts. In clinical settings, disrupted prefrontal–amygdala connectivity is a hallmark of stress-related disorders such as anxiety and depression, and the present results underscore the importance of training programs that enhance cognitive control and regulatory flexibility. In occupational or educational environments where individuals frequently encounter acute stress, understanding these neural dynamics can inform the development of stress management interventions, resilience training, and mental health support systems.

Future research should expand on these findings by incorporating multimodal physiological measures, longitudinal designs, and larger samples to investigate how chronic stress shapes regulatory networks over time. Additionally, integrating individual difference measures-such as trait reappraisal ability, personality factors, and genetic predispositions-would further refine our understanding of stress-related emotional regulation. Advances in neuroimaging methods and machine learning approaches may eventually allow for the identification of neural biomarkers that can predict vulnerability to stress and inform personalized interventions.

In conclusion, this study clarifies how acute stress modifies the neural architecture of emotional regulation, revealing a pattern of intensified limbic activation, impaired prefrontal engagement, and weakened cross-network connectivity. These findings highlight the complexity of emotional regulation under stress and underscore the need for continued research aimed at enhancing adaptive coping mechanisms in the face of stressors that characterize contemporary life.

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