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Neuroimaging cognitive reappraisal in clinical populations to define neural targets for enhancing emotion regulation. A systematic review.

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Highlights

- A systematic review of 32 neuroimaging studies on cognitive reappraisal in patients
- Lower vIPFC/dIPFC activation is a core deficit in downregulation across patients
- Amygdala hyperactivity is a specific deficit in downregulation in mood disorders
- dACC/parietal hypoactivity specific deficit in downregulation in anxiety disorders
- Implications: neural targets for therapeutic interventions need to be tailored

Abstract

Reduced capacity to cognitively regulate emotional responses is a common impairment across major neuropsychiatric disorders. Brain systems supporting one such strategy, cognitive reappraisal of emotion, have been investigated extensively in the healthy population, a research focus that has led to influential meta-analyses and literature reviews. However, the emerging literature on neural substrates underlying cognitive reappraisal in clinical populations is yet to be systematically reviewed. Therefore, the goal of the current review was to summarize the literature on cognitive reappraisal and highlight common and distinct neural correlates of impaired emotion regulation in clinical populations. We performed a two-stage systematic literature search, selecting 32 studies on cognitive reappraisal in individuals with mood disorders (n=12), anxiety disorders (n=14), addiction (n=2), schizophrenia (n=2), and personality disorders (n=5). Comparing findings across these disorders allowed us to determine underlying mechanisms that were either disorder-specific or common across disorders. Results showed that across clinical populations, individuals consistently demonstrated reduced recruitment of the ventrolateral prefrontal cortex (vIPFC) and dorsolateral prefrontal cortex (dIPFC) during downregulation of negative emotion, indicating that there may be a core deficit in selection, manipulation and inhibition during reappraisal. Further, in individuals with mood disorders, amygdala responses were enhanced during downregulation of emotion, suggesting hyperactive bottom-up responses or reduced modulatory

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