

# Disorder-specific dysfunction in dorsal anterior cingulate cortex and parietal cortex during cognitive reappraisal in anxiety disorder [Letter]

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## Dear editor

Following our recently published article<sup>1</sup> in *Neuropsychiatric Disease and Treatment*, many readers have commented that the effects of comorbid depression cannot be ruled out as a factor in our results, since anxiety disorders and major depression are heterogeneous disorders. We would like to respond to the points made.

Impaired cognitive emotion regulation is commonly seen in many psychiatric disorders.<sup>2</sup> A recent review concluded that individuals with depressive disorders consistently show decreased activation of the ventrolateral prefrontal cortex (vlPFC) and dorsolateral prefrontal cortex (dlPFC) during cognitive reappraisal.<sup>3</sup> Moreover, enhanced activity of the amygdala, which is associated with hyperactive bottom-up emotion responses during downregulation of negative emotion, is a disorder-specific deficit in the recruitment of brain regions during cognitive reappraisal in depression disorder.<sup>3</sup> Our meta-analysis was intended to provide a comprehensive description of the brain mechanisms underlying cognitive reappraisal deficits in anxiety disorder. We concluded that patients with anxiety disorder could not recruit the frontoparietal network, including dorsal anterior cingulate cortex (dACC), parietal cortex, dorsomedial prefrontal cortex and supplementary motor area, to down-regulate their emotion responses.

Notably, we did not find that abnormalities of vlPFC, dlPFC or amygdala activity were involved in anxiety disorder, which suggests that the cognitive-appraisal-related impairments in emotion regulation that occur in anxiety disorders and major depression have different neural correlates. There are several explanations for our negative findings. First, although we included some patients with anxiety disorder accompanied by depression, they were eligible for inclusion in the meta-analysis as anxiety disorder was the primary diagnosis. Second, although some studies reported that patients with anxiety disorder showed diminished vlPFC and dlPFC activity during downregulation of negative emotion,<sup>1</sup> the results are mixed. Our comprehensive meta-analysis was performed to provide a robust description of the neurobiological underpinnings of anxiety. Third, although patients with major depression consistently show enhanced amygdala activity during cognitive reappraisal,<sup>3</sup> none of the eight studies included in the meta-analysis reported enhanced amygdala during cognitive reappraisal in anxiety disorders.<sup>1</sup>

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Using a more stringent significance criterion ( $p < 0.001$ ), we found that during cognitive reappraisal patients with anxiety disorder showed decreased activity relative to healthy controls in the dACC (peak MNI: 4, 12, 42;  $p = 0$ ;  $Z = -2.838$ ; voxels = 2129) and parietal cortex (peak MNI: -48, -28, 46;  $p = 0.000002563$ ;  $Z = -2.329$ ; voxels = 544); thus, the main findings remained largely unchanged. The dACC is involved in allocating control and attentional resources during cognitive reappraisal,<sup>4</sup> whilst parietal cortex is mainly associated with selective attention and may be involved in holding reappraisals in mind.<sup>5</sup> Our results indicate that, in anxiety disorder, diminished dACC and parietal cortex activity may impair the allocation of attention and control during cognitive reappraisal. It should be noted that none of the nine major depressive disorder studies we reviewed reported reduced dACC and parietal cortex during cognitive reappraisal.<sup>3</sup> Importantly, patients with anxiety disorder, unlike those with major depression, did not show enhanced amygdala activity or reduced vIPFC and dlPFC activity during cognitive reappraisal. Conversely, patient with major depression did not show decreased activation of the dACC and parietal cortex, unlike those with anxiety disorder. We believe, therefore, that the effects of comorbid depression in anxiety disorder on cognitive reappraisal in our meta-analysis are negligible or non-existent.

These findings offer insight into the neurobiology of emotion dysregulation in anxiety disorder. They provide

robust evidence that frontoparietal network impairments play a key role in the pathogenesis of anxiety disorder. More specifically, it appears that dysfunctions of dACC and parietal cortex, which are involved in allocation of attention and control, are relatively specific to anxiety disorder, rather than depression.

## Disclosure

The authors report no conflicts of interest in this communication.

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