# Letters

## Altered pain threshold sensitivity and frontoparietal– cingulate network in anorexia nervosa: the role of disgust sensitivity

The July issue included an interesting study<sup>1</sup> investigating the contribution of the cingulate cortex in altered pain processing in anorexia nervosa. In particular, the study provides the first direct evidence of structural alterations (i.e., decreased grey matter volume and cortical thickness) in correspondence of the frontoparietal-cingulate network of this clinical population, which relates with their higher mean thermal pain threshold as well as with symptom severity and illness duration. According to these results, the authors conclude by arguing the existence of a common neural origin for thermal thresholds alterations and the clinical characteristics of anorexia nervosa.

The neural and behavioural patterns reported by the authors suggest another, not mutually exclusive, interpretation of this result, which fits with the hypothesis of an altered disgust sensitivity in anorexia nervosa.

First, we know that anorexia nervosa is affected by a consistently high disgust sensitivity.2 We also know that the experience of disgust and pain share common neural patterns of activity in the anterior, mid and posterior cingulate cortex and right parietal operculum.3 There is also evidence of a common autonomic nervous system activity in correspondence of the parasympathetic branch for both the experiences of pain<sup>4</sup> and disgust.5 Finally, the recent study by Oaten and colleagues<sup>6</sup> demonstrates that the experience of disgust is unique in generating a significant, increasing trend in pain sensitivity.

For all these reasons, the absence of measures for assessing the subjective

disgust sensitivity threshold in the anorexia nervosa participants represents an important limitation for interpreting the results reported by Bär and colleagues.<sup>1</sup>

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## **Author response**

We appreciate the point that elevated feelings of disgust have not been assessed in our study. However, we are at variance to his conclusion. In Vicario's former letter to the editor of the *American Journal of Psychiatry*, he has strengthened the point of altered insula activation being associated with feelings of disgust. However, we have not observed altered insula activation during pain perception in our patients. In addition, Vicario cites the work by Oaten and colleagues, which demonstrates that the experience of disgust is

generating an increase in pain sensitivity. However, the opposite is true in patients with anorexia. They are less sensitive to pain, as shown in our study. In addition, it is generally assumed that disgust is accompanied by increased activation of the parasympathetic nervous system (PNS). However, empirical support for the role of PNS in disgust is scarce. Probably, the story is far more complicated. He and colleagues3 reported an increase of heart rate associated with unpleasant odors and facial expressions of disgust, while de Jong and colleagues4 suggested some PNS involvement. However, physiologic reactions were independent from disgust sensitivity. To our knowledge, the specific relation between disgust and PNS activity has not been studied in patients with anorexia nervosa. Thus, we would like to express our sincere doubts on the relation between disgust and our findings as raised by Vicario.

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