LINK TO ORIGINAL ARTICLE
LINK TO AUTHORS' REPLY

Body-image distortion in anorexia nervosa

Antonios Dakanalis, Santino Gaudio, Silvia Serino, Massimo Clerici, Giuseppe Carrà and Giuseppe Riva

In their recent Primer (Anorexia nervosa. *Nat. Rev. Dis. Primers* **1**, 15074 (2015))[1], Janet Treasure and colleagues give an important update on the advances made in the aetiology, assessment, prevention and treatment of anorexia nervosa (AN). The authors also highlight the need for better, faster and lasting improvements in the management of this 'enigmatic' disorder, which crucially depends on improved understanding of specific disease mechanisms. However, in their description, they do not include the consistent evidence that disturbed body image does not only motivate severe dietary restriction and other weight loss behaviours but also plays a central part in the initiation, persistence and relapse of AN[2–8]. The Primer[1] states that the underlying mechanism of the so-called body-image distortion (BID), in which emaciated individuals perceive themselves as fat[9], remains obscure. Yet, there is widespread agreement that this evidence — at both the conceptual and the empirical levels — is imperative for elucidating what is behind severe and intense BID[2–10].

Findings from an increasing number of functional MRI (fMRI) studies, conducted (based on the symptom provocation paradigm) over the past 15 years and recently reviewed by one of us[7], provide valuable insights into the neural basis of BID in AN. Unfortunately, we felt that these issues were not entirely addressed by the Primer[1], making it difficult to understand the 'reasonably consistent' (REF. 7) evidence produced by this research[3]. The review[7] summarized that the 'affective' component of BID in AN is related to alterations of the prefrontal cortex, the insula and the amygdala and that the 'perceptive' component of BID is related to alterations of the parietal lobes (which have roles in spatial and body representations[4–7,10], body ownership[9–12] and other features requiring multisensory integration[4–7,11–14]) or, more accurately, the posterior parietal regions (which are involved in visuospatial processing[4–7,10,15]). A deficit in parietal cortex-mediated functions in AN is also underscored by findings from neurocognitive studies[4–6]. Although both extant neuroimaging and behavioural data[2,4,7,8] suggest that two components of body image (the estimation of one's own body size and the attitude towards one's own body in terms of an emotional evaluation) are disturbed in individuals with AN, these aspects might have been described in more detail in the Primer[1]. In fact, although two (widely accepted) body-image components can be distinguished, this does not imply that they are independent[4,5,15]. Indeed, experimental evidence supports a direct (unidirectional) link between how we perceive and how we feel about our body[12]. The aforementioned specific neural bases of the affective component of BID in AN also support an altered emotional response to unpleasant (for example, self-distorted fat image) stimuli[7]. Furthermore, in the few available fMRI studies based on a word paradigm (that is, tasks using 'fat', 'thin' and 'neutral' words), a variation in amygdala response was absent — making the involvement of this brain region less clear but suggesting the greater relevance of self-perception and the mechanism of body-image construction[5,6,9] (see below). There is the need to take into account these (and other convergent[4,5,12,13]) clues and the considerable room for improvement that remains from the first-line prevention and psychotherapeutic interventions[2,3,5,8,10,12,13], currently described in the Primer[1] (for example, the Body Project and enhanced cognitive–behavioural therapy), and targeting the 'affective' body-image component[2,5,10,12,13]. Thus, we would suggest that it is now time to consider the development of intervention strategies that target the perceptive component.

Cognitive neuroscientific strides in the field of body self-consciousness (that is, the experience of being in a body process[5], with body ownership being its fundamental constituent[5,11]) offer a novel perspective for, if not a paradigm shift in, understanding the mechanism of body-image construction[5,6,10,11,13,15] — only inferred by the already mentioned experimental paradigms used to explore the neural basis of BID in AN[4,7]. Fundamentally, our spatial experience, including the bodily one, is organized around two different reference frames: egocentric, which has its primary source in 'online' representations (referring to the temporal flow of information that constructs how our body is right now), and allocentric, which has its primary source in 'offline' representations (referring to what our body is normally like)[5,6,10,13,15]. That is, people use both the memory of how the body, including its shape and size, is believed (or remembered) to be (offline) and the perception of the body 'here and now' (online) to construct their body image[5,6]. The conceptual distinction between online and offline representations should not imply that these representations of the body are unconnected, as they can and do interact, with the online information about the body being continuously integrating with and compared to the offline model of the body in the brain[5,6,10,13,15]. Neuroscientifically informed models highlight how a similar process of amending and updating offline representations based on new online representations might underline the complex relationship between body image, dietary restriction and weight loss[5]. From this perspective, individuals who lose large amounts of weight might adapt their offline model of the body accordingly, as the new online information is received and updates the oldest stored model of the body[5]. Accordingly, an impediment in the transaction between online and offline information might be at play in patients with AN who have a persistent experience of being fat, even when they are objectively emaciated[5]. In support, sophisticated contemporary research[10] has shown that individuals with AN in the earliest stages are 'locked' in a virtual 'wrong' body that they detest, which differs from the real one.

Despite the importance of these[10] (and additional[5,13,14]) findings and the known role of brain abnormalities in the (posterior parietal) areas involved in the block of the online–offline transformation process[5,6,11,15], further research is needed to investigate additional factors (for example, stress) involved in the impaired ability of updating[5,10], which were previously linked to food intake reduction in AN[10]. Some additional hypotheses have been proposed based on research on neuropsychological functioning[1,4,6,10] in patients with AN but they require specific testing. For example, some evidence suggests that people with AN not only focus on details (weak central coherence) but they also seem to have an attentional bias towards negatively charged details[6]. This feature has been suggested to affect both the perception of the body and how it is remembered[6]. Another hypothesis states that weak set shifting (that is, reduced mental flexibility as indicated by the ability to move back and forth between tasks) and poor visuospatial memory could affect the ability to adapt offline body representation based on new information and form a correct visual representation of the body, respectively[6,10,14].

Collectively, we now have a rich conceptual framework and empirical data for understanding BID in AN, but also a set of open questions that could provide further contributions to the knowledge of its complexity[5,10] and valuable insights to further improve the effectiveness of existing prevention and psychotherapeutic interventions[2,3,10]. Substantial advances in this area crucially depend on improved understanding of disease mechanisms[1], and extant prevention and treatment strategies are hindered by the lack of specified conceptual models underlying the mechanism driving the extremes of BID in AN[2].

Antonios Dakanalis is at the Department of Brain and Behavioural Sciences, University of Pavia, P.za Botta 11, 27100 Pavia, Italy; and at the Department of Interdisciplinary Medicine, University of Milan-Bicocca, Milan, Italy.

Santino Gaudio is at the Centre for Integrated Research, Area of Diagnostic Imaging, University Campus Bio-Medico, Rome, Italy; and at the Department of Neuroscience, Functional Pharmacology, Uppsala University, Uppsala, Sweden.

Silvia Serino is at the Department of Psychology, Catholic University, Milan, Italy.

Massimo Clerici is at the Department of Interdisciplinary Medicine, University of Milan-Bicocca, Milan, Italy.

Giuseppe Carrà is at the Department of Interdisciplinary Medicine, University of Milan-Bicocca, Milan, Italy; and at the Division of Psychiatry, Faculty of Brain Sciences, University College London, London, UK.

Giuseppe Riva is at the Department of Psychology, Catholic University, Milan, Italy; and at the Applied Technology for Neuro-Psychology Laboratory, Istituto Auxologico Italiano, IRCCS, Milan, Italy.

Correspondence to A.D.
antonios.dakanalis@unipv.it

Article number: 16026
doi:10.1038/nrdp.2016.26
Published online 21 April 2016

1. Treasure, J. *et al.* Anorexia nervosa. *Nat. Rev. Dis. Primers* **1**, 15074 (2015).
2. Delinsky, S. S. in *Body Image: A Handbook of Science, Practice, and Prevention* (eds Cash, T. F. & Smolak, L.) 279–287 (Guilford Press, 2011).
3. Zipfel, S., Giel, K. E., Bulik, C. M., Hay, P. & Schmidt, U. Anorexia nervosa: aetiology, assessment, and treatment. *Lancet Psychiatry* **2**, 1099–1111 (2015).
4. Suchan, B., Vocks, S. & Waldorf, M. Alterations in activity, volume, and connectivity of body-processing brain areas in anorexia nervosa. *Eur. Psychol.* **20**, 27–33 (2015).
5. Riva, G. Out of my real body: cognitive neuroscience meets eating disorders. *Front. Hum. Neurosci.* **8**, 236 (2014).
6. Øverås, M. in *Eating Disorders and the Brain* (eds Lask, B. & Frampton, I.) 129–141 (Wiley Blackwell, 2011).
7. Gaudio, S. & Quattrocchi, C. C. Neural basis of a multidimensional model of body image distortion in anorexia nervosa. *Neurosci. Biobehav. Rev.* **36**, 1839–1847 (2012).
8. Smolak, L. & Levine, M. P. in *The Wiley Handbook of Eating Disorders* (eds Smolak, L. & Levine, M. P.) 2–12 (Wiley Blackwell, 2015).
9. Frank, G. K. & Kaye, W. H. Current status of functional imaging in eating disorders. *Int. J. Eat. Disord.* **45**, 723–736 (2012).
10. Serino, S. *et al.* Out of body, out of space: impaired reference frame processing in eating disorders. *Psychiatry Res.* **230**, 732–734 (2015).
11. Giummarra, M. J., Gibson, S. J., Georgiou-Karistianis, N. & Bradshaw, J. L. Mechanisms underlying embodiment, disembodiment and loss of embodiment. *Neurosci. Biobehav. Rev.* **32**, 143–160 (2008).
12. Preston, C. & Ehrsson, H. H. Illusory changes in body size modulate body satisfaction in a way that is related to non-clinical eating disorder psychopathology. *PLoS ONE* **9**, e8577 (2014).
13. Riva, G., Gaudio, S. & Dakanalis, A. The neuropsychology of self-objectification. *Eur. Psychol.* **20**, 34–43 (2015).
14. Guardia, D. *et al.* Imagining one's own and someone else's body actions: dissociation in anorexia nervosa. *PLoS ONE* **7**, e43241 (2012).
15. Berlucchi, G. & Aglioti, S. M. The body in the brain revisited. *Exp. Brain Res.* **200**, 25–35 (2010).

Competing interests
The authors declare no competing interests.