Medical Hypotheses xxx (2011) xxx–xxx

Contents lists available at SciVerse ScienceDirect

Medical Hypotheses

journal homepage: www.elsevier.com/locate/mehy

# Neuroscience and eating disorders: The allocentric lock hypothesis

Giuseppe Riva *

*Applied Technology for Neuro-Psychology Lab. – ATN-P Lab., Istituto Auxologico Italiano, Milan, Italy*

ARTICLE INFO

*Article history:*
Received 11 October 2011
Accepted 25 October 2011
Available online xxxx

ABSTRACT

Evidence from psychology and neuroscience indicates that our spatial experience, including the bodily one, involves the integration of different sensory inputs within two different reference frames *egocentric* (body as reference of first-person experience) and *allocentric* (body as object in the physical world). Even if functional relations between these two frames are usually limited, they influence each other during the interaction between long- and short-term memory processes in spatial cognition. If, for some reasons, this process is impaired, the egocentric sensory inputs are no more able to update the contents of the allocentric representation of the body: the subject is locked to it. In the presented perspective, subjects with eating disorders are locked to an allocentric representation of their body, stored in long-term memory (allocentric lock). A significant role in the locking may be played by the medial temporal lobe, and in particular by the connection between the hippocampal complex and amygdala. The differences between exogenous and endogenous causes of the lock may also explain the difference between bulimia nervosa and anorexia nervosa.



## Background

Evidence from psychology and neuroscience indicates that our spatial experience, including the bodily one, involves the integration of different sensory inputs within two different reference frames [1,2]: *egocentric* (body as reference of first-person experience) having its primary source in *somatoperceptions* – representations of the present state of the body and tactile stimuli from sensory inputs; *allocentric* (body as object in the physical world) having its primary source in *somatorepresentations* – abstract knowledge, beliefs, and attitudes related to body as an object of third-person experience.

These frames influence the way memories are stored and retrieved [3,4]: the rememberer may "see" the event from his or her perspective as in normal perception (*field mode*), or "see" the self engaged in the event as an observer would (*observer mode*).

More, they influence the ability of representing and recalling our body: an egocentric representation of how our body looks is matched by an allocentric one, used by our brain in different situations [3,5]: from spatial cognition to social perception.

The functional relations between these two frames are usually limited [2]. This is because the egocentric frame is based on an integrated, on-line percept of the current state of the body, while the allocentric one involves knowledge about one's body as a unique, continuous object.

However, Byrne and colleagues [6] recently proposed a model that uses the interaction between egocentric and allocentric representations to account for the interaction between long- and short-term memory processes in spatial cognition. Specifically, long-term spatial memory involves the generation of allocentric representations in the hippocampus and surrounding medial temporal lobe structures. Instead, short-term spatial memory and imagery are modeled as egocentric representations of locations in the precuneus, driven either by perception or by long-term memory. Within this framework, the process of spatial encoding and retrieval requires a translation between the allocentric long-term memory and the egocentric spatial updating, which occurs via a coordinate transformation in the posterior parietal and retrosplenial cortices.

On one side, an allocentric representation of the body – cued by knowledge of position and orientation and retrieved from long-term memory – is translated by the Papez's circuit in an egocentric representation that can influence the existing ones, including body dimensions and motor patterns [7].

On the other side, an egocentric representation of the body can influence an allocentric memory through the reactivation of a population of boundary vector cells located within parahippocampal cortex.

## Hypothesis

If, for some reasons, the above process is impaired, the egocentric sensory inputs are no more able to update the contents of the allocentric representation of the body: the subject is locked to it [8].

* Address: ATN-P Lab., Università Cattolica del Sacro Cuore, Largo Gemelli 1, 20123 Milan, Italy. Tel.: +39 02 72343734; fax: +39 02 72342280.
*E-mail address:* giuseppe.riva@unicatt.it

0306-9877/$ - see front matter © 2011 Elsevier Ltd. All rights reserved.
doi:10.1016/j.mehy.2011.10.039

This is what apparently happens in eating disorders (ED): a negative allocentric representation – that is not updated by contrasting egocentric parietal representations driven by perception [9] – primes the processing of any further body-related experience [10].

Apparently, a significant role in the locking may be played by the medial temporal lobe, and in particular by the connection between the hippocampal complex and amygdala. As we have seen, the hippocampus has a critical role in spatial memory by generating allocentric representations for long-term memory [6]. Further, a recent neurological study showed an important role for the left hippocampus and some parahippocampal regions, in particular the entorhinal cortex, in episodic memory consolidation and recall [11].

These data suggest that the hippocampus may provide the spatial context appropriate to recollection of episodic events, explaining its role in the context-dependent fear conditioning found in ED. In fact, there is a strict link between hippocampus and amygdala [12]: the hippocampal complex, by forming episodic representations of the emotional significance of a stimulus can lead to the activation of the amygdala, explaining the avoidance and negative feelings related to the body. At the same time, the amygdala may modulate both the storage and the consolidation of hippocampal-dependent memories, explaining the difficulty in updating the allocentric representation of the body. Specifically, amygdala may modulate within-object binding in the perirhinal cortex enhancing long-term consolidation of emotionally arousing objects [13]: whatever is considered to be an integral component of the emotionally arousing object will also show enhanced long-term consolidation. For example, experiencing a tight pair of trousers may consolidate the allocentric representation of the body.

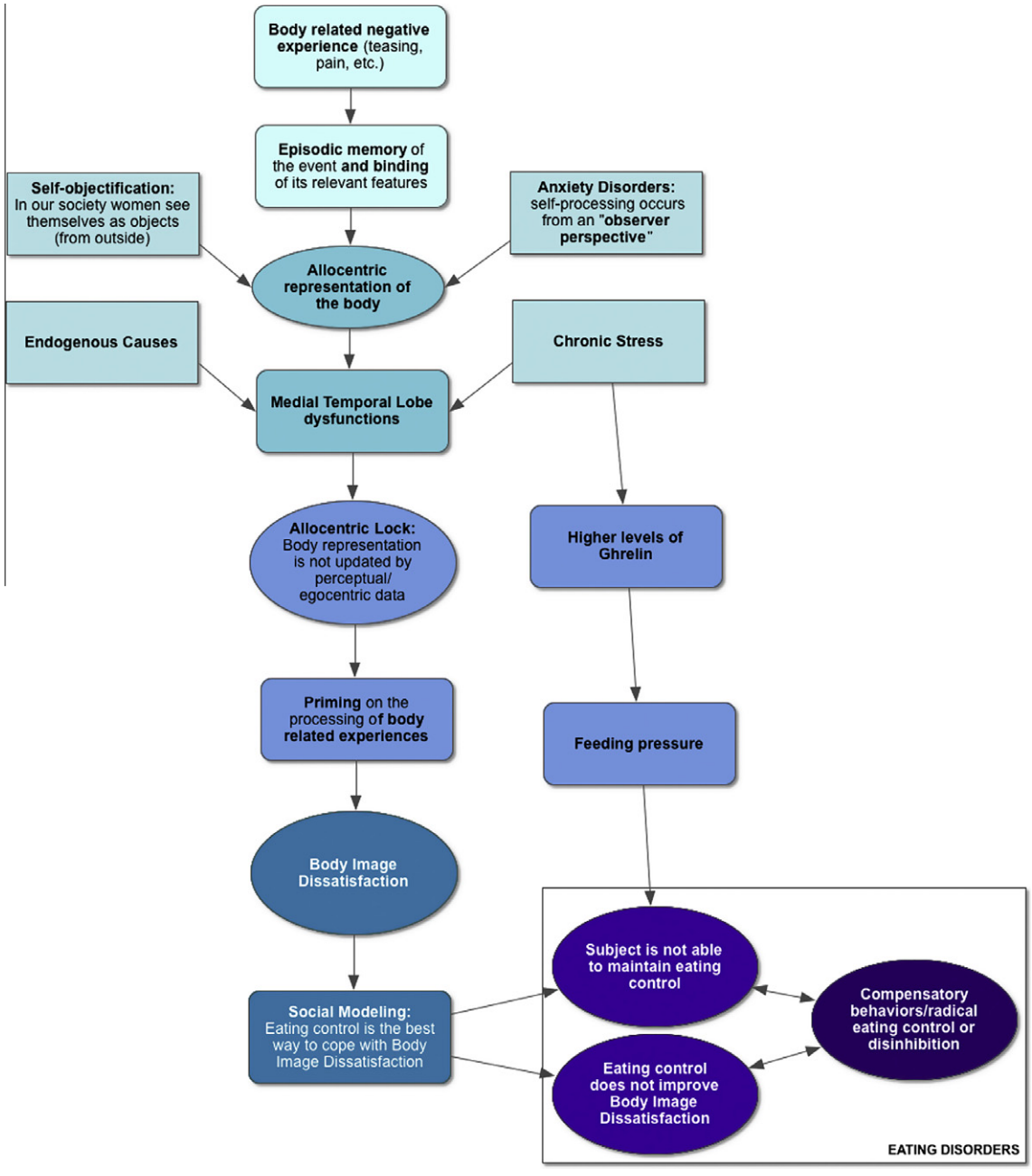


**Fig. 1.** The allocentric lock hypothesis.

Please cite this article in press as: Riva G. Neuroscience and eating disorders: The allocentric lock hypothesis. Med Hypotheses (2011), doi:10.1016/j.mehy.2011.10.039

## Evaluation of the hypothesis

A growing body of evidence has demonstrated that stress, and in particular chronic stress, can cause hippocampal damage [14]. Specifically, chronic stress produces consistent and reversible changes within the dendritic arbors of CA3 hippocampal neurons [14,15]. This process disrupts hypothalamic–pituitary–adrenal axis activity, leading to dysregulated glucocorticoid release that, combined with hippocampal CA3 dendritic retraction, contributes to impaired spatial memory [15]. Interestingly, some authors suggested that the onset of ED is often related to critical life events (personal and/or familiar) and that chronic stress is associated with the persistence of these disorders [16,17]. More, the ghrelin hormone, which increases during fasting, may defend individuals against depressive-like symptoms of chronic stress [18].

The passage from a locked allocentric representation to an ED may be explained by social influence (Fig. 1): media and culture promote diet as the best ways to improve body image satisfaction. However, the impossibility of updating the allocentric representation of the body, even after a demanding diet, locks the patient into an unsatisfying body.

Finally, the difference between exogenous (stress) and endogenous causes of the lock may explain the difference between bulimia nervosa and anorexia nervosa and between the anorexia subtypes: they are exogenous in bulimia nervosa and endogenous in anorexia nervosa. More, if in bulimia nervosa and in the binge-eating/purging type of anorexia nervosa the loss of appetite induced by stress (corticoliberin) is matched by a pressure towards an increased caloric intake (ghrelin), this may not happen in restrictive anorexia nervosa for an alteration in ghrelin secretion [19,20].

In fact, different neuroimaging data showed some impairment in the medial temporal lobe of anorectic patients. Connan and colleagues [21] found in anorectic patients a significant reduction in hippocampal volume (–8.2% right; –7.5% left) compared with healthy subjects. Wagner and colleagues [22] confronted female anorectic patients and healthy controls with their own digitally distorted body images: anorectics experienced a hyperresponsiveness in the parietal lobe, suggestive of a disturbance in the visuospatial processing of the own body shape. Yonezawa and colleagues [23] found bilateral decreased perfusion of the subcallosal gyrus, midbrain and posterior cingulate gyrus in both restricting and binge-eating/purging anorectic patients, as compared with the controls. The abnormalities of regional cerebral blood flow disappear after recovery in both anorexia and bulimia nervosa [24].

## Consequences of the hypothesis

Eating disorders are one of the most troubling behavioral disorders in our society. Eating disorders rob girls and young women of their future and, not uncommonly, their lives. However, as underlined by Fairburn and Harrison in their review for Lancet [25]: "virtually nothing is known about the individual causal processes involved, or about how they interact and vary across the development and maintenance of the disorders." (p. 409). For these reasons, advancing the understanding and treatment of eating disorders is an issue of immense public health importance and is recognized as an area of high priority by the National Institute of Mental Health (NIMH).

Actually, the most followed path in neuroscience research is to consider anorexia as the outcome of a primary disturbance of brain ventral and dorsal appetitive circuits [26]. In this paper we suggested an alternative view more focused on a different feature of eating disorders: the distorted experience of the body [27,28]. Specifically, we suggested that subjects with eating disorders are locked to an allocentric negative representation of their body, that they are not able to modify even after dramatic body changes.

As for any new perspective, much more research is needed before it can be retained or discarded. However, it provides a possible explanation of eating disorders that addresses the complex etiology of these disturbances by including socio-cultural and biological factors. Further, it suggests a clear link between eating disorders and the medial temporal lobe that may be helpful to enhance the therapeutic options for these disturbances.

In conclusion, we know the power of understanding etiology in the search for effective interventions: treatment is best accomplished when we know the causes of a disorder. For this reason any step, even if partial, towards a better understanding of eating disorders may be useful for advancing treatment of these complex and multifaceted diseases.

## Conflict of interest

None declared.

## Acknowledgment

This work was supported by the Italian MIUR-FIRB fund for investing in fundamental research: Project "Immersive Virtual Telepresence (IVT) for Experiential Assessment and Rehabilitation – IVT 2010 – RBIN04BC5C".

## References

[1] Galati G, Lobel E, Vallar G, Berthoz A, Pizzamiglio L, Le Bihan D. The neural basis of egocentric and allocentric coding of space in humans: a functional magnetic resonance study. Exp Brain Res 2000;133:156–64.
[2] Longo MR, Azañón L, Haggard P. More than skin deep: body representation beyond primary somatosensory cortex. Neuropsychologia, in press, doi:10.1016/j.neuropsychologia.2009.1008.1022.
[3] Amorim MA. What is my avatar seeing? The coordination of "out-of-body" and "embodied" perspectives for scene recognition across views. Visual Cogn 2005;10:157–99.
[4] Robinson JA, Swanson KL. Field and observer modes of remembering. Memory 1993;1:169–84.
[5] Juurmaa J, Lehtinen-Railo S. Visual experience and access to spatial knowledge. J Visual Impairment Blindness 1994;88:157–70.
[6] Byrne P, Becker S, Burgess N. Remembering the past and imagining the future: a neural model of spatial memory and imagery. Psychol Rev 2007;114:340–75.
[7] Vocks S, Legenbauer T, Ruddel H, Troje NF. Static and dynamic body image in bulimia nervosa: mental representation of body dimension and biological motion patterns. Int J Eating Disord 2007;40:59–66.
[8] Riva G. Virtual body, real pain: the allocentric lock hypothesis. In: Zampini M, Pavani F, editors. Body representation workshop, Università degli Studi di Trento, Rovereto, Italy; 2007. online: www.cimec.unitn.it/events/brw/Poster/107_abs_GIUSEPPE_RIVA.pdf.
[9] Smeets MA, Ingleby JD, Hoek HW, Panhuysen GE. Body size perception in anorexia nervosa: a signal detection approach. J Psychosom Res 1999;46:465–77.
[10] Sachdev P, Mondraty N, Wen W, Gulliford K. Brains of anorexia nervosa patients process self-images differently from non-self-images: an fMRI study. Neuropsychologia 2008;46:2161–8.
[11] Di Paola M, Macaluso E, Carlesimo GA, Tomaiuolo F, Worsley KJ, Fadda L, et al. Episodic memory impairment in patients with Alzheimer's disease is correlated with entorhinal cortex atrophy: a voxel-based morphometry study. J Neurosci 2007;254:774–81.
[12] Peissig J, Kollmeier B. Directivity of binaural noise reduction in spatial multiple noise-source arrangements for normal and impaired listeners. J Acoust Soc Am 1997;101:1660–70.
[13] Mather M. Emotional arousal and memory binding: an object-based framework. Perspect Psychol Sci 2007;2:33–52.
[14] McLaughlin KJ, Gomez JL, Baran SE, Conrad CD. The effects of chronic stress on hippocampal morphology and function: an evaluation of chronic restraint paradigms. Brain Res 2007;1161:57–64.
[15] Conrad CD. What is the functional significance of chronic stress-induced ca3 dendritic retraction within the hippocampus? Behav Cogn Neurosci Rev 2006;5:41–60.
[16] Lo Sauro C, Ravaldi C, Cabras PL, Faravelli C, Ricca V. Stress, hypothalamic-pituitary-adrenal axis and eating disorders. Neuropsychobiology 2008;57:95–115.
[17] Corstorphine E, Mountford V, Tomlinson S, Waller G, Meyer C. Distress tolerance in the eating disorders. Eat Behav 2007;8:91–7.

Please cite this article in press as: Riva G. Neuroscience and eating disorders: The allocentric lock hypothesis. Med Hypotheses (2011), doi:10.1016/j.mehy.2011.10.039

[18] Lutter M, Sakata I, Osborne-Lawrence S, Rovinsky SA, Anderson JG, Jung S. Et al.. The orexigenic hormone ghrelin defends against depressive symptoms of chronic stress Nat Neurosci 2008;11:752–3.
[19] Terashi M, Asakawa A, Harada T, Ushikai M, Coquerel Q, Sinno MH, et al. Ghrelin reactive autoantibodies in restrictive anorexia nervosa. Nutrition 2011;27:407–13.
[20] Germain N, Galusca B, Grouselle D, Frere D, Billard S, Epelbaum J, et al. Ghrelin and obestatin circadian levels differentiate bingeing-purging from restrictive anorexia nervosa. J Clin Endocrinol Metab 2010;95: 3057–62.
[21] Connan F, Murphy F, Connor SE, Rich P, Murphy T, Bara-Carill N, et al. Hippocampal volume and cognitive function in anorexia nervosa. Psychiatry Res Neuroimaging 2006;146:117–25.
[22] Wagner A, Ruf M, Braus D, Schmidt MH. Neural activity changes and body image distortion in anorexia nervosa. NeuroReport 2003;14:2193–7.
[23] Yonezawa H, Otagaki Y, Miyake Y, Okamoto Y, Yamawaki S. No differences are seen in the regional cerebral blood flow in the restricting type of anorexia nervosa compared with the binge eating/purging type. Psychiatry Clin Neurosci 2008;62:26–33.
[24] Frank GK, Bailer UF, Meltzer CC, Price JC, Mathis CA, Wagner A, et al. Regional cerebral blood flow after recovery from anorexia or bulimia nervosa. Int J Eat Disord 2007;40:488–92.
[25] Fairburn CG, Harrison PJ. Eating disorders. Lancet 2003;361:407–16.
[26] Kaye WH, Fudge JL, Paulus M. New insights into symptoms and neurocircuit function of anorexia nervosa. Nat Rev Neurosci 2009;10:573–84.
[27] Keizer A, Smeets MA, Dijkerman HC, van den Hout M, Klugkist I, van Elburg A, et al., Tactile body image disturbance in anorexia nervosa. Psychiatry Res 2011, doi:10.1016/j.psychres.2011.04.031.
[28] Espeset EM, Nordbo RH, Gulliksen KS, Skarderud F, Geller J, Holte A. The concept of body image disturbance in anorexia nervosa: an empirical inquiry utilizing patients' subjective experiences. Eat Disord 2011;19:175–93.

Please cite this article in press as: Riva G. Neuroscience and eating disorders: The allocentric lock hypothesis. Med Hypotheses (2011), doi:10.1016/j.mehy.2011.10.039