Published in final edited form as:

Int J Eat Disord. 2017 September ; 50(9): 1014–1017. doi:10.1002/eat.22742.

A Translational Neuroscience Approach to Body Image Disturbance and its Remediation in Anorexia Nervosa

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Abstract

Deviant perception of the body is a fundamental component of anorexia nervosa (AN). Here we offer a potential mechanistic explanation that involves perturbations within the visual system and the brain circuits that modulate perceptual organization. Based on the model proposed, we also suggest a mechanistic strategy for altering neuronal activity in the visual system to normalize perception of the body, and set out a strategy for empirically testing its clinical application.

1 INTRODUCTION

Persons with anorexia nervosa (AN) typically insist that their body's size, usually measured in terms of weight, is far greater than how it is perceived by others. As of yet, there is no satisfactory causal understanding, nor is there an effective therapy. Here we take a translational neuroscience perspective to (1) offer a neurally plausible, testable interpretation centered on circuit elements within the visual system, and (2) describe a novel behavioral manipulation to restore the normal operation of these elements.

2 VISUAL INFORMATION PROCESSING IN BRIEF

After the retinal tissue converts light into a neural signal, visual information travels the optic tract to the striate cortex where it is processed by two principal neuronal streams. One runs dorsally into the parietal cortex and receives inputs from phylogenetically old cells within the superior colliculus and pulvinar nuclei of the thalamus to rapidly process coarse—low spatial frequency (LSF)—information, in effect, an object's sensory “whole.” Because of its rapid action, the dorsal stream is thought to be a key substrate for engaging instinctive defensive behavior as its operation enhances detection of stimuli with high emotional valence, even when the stimulus is presented at unattended locations or below the threshold for conscious recognition. The other stream lies ventrally and involves the occipital fusiform gyrus and temporal cortex. An evolutionarily newer part of the visual system, it mediates a slower, piecemeal processing of an object's high fidelity attributes. For a coherent visual representation to be reliable the two inputs must be harmonized—the dorsal stream giving context to the object's unique details. Should the integration fail, for example, if there is

sufficient activation of only one pathway or if the system is unable to dynamically adjust to demands or features of a shifting visual landscape, perceptual capacity would suffer.

But human evolution conserved broad complexities in neural operations designed to maximize survival as our ancestors migrated to novel, more complex, potentially lethal ecosystems. Evidence of this complexity is the assortment of visually guided mechanisms that aid detection and appraisal of threat, as well as multiple, bilateral connections between the visual cortex and brain regions that modulate attention, instantiate negative emotion and appetitive motivation, and instigate motor actions. In short, evolutionary pressures tethered the visual cortex to information generating circuits that govern multiple physiological, cognitive, and affective states, some conscious, some not (Mobbs, Hagan, Dalgleish, Silston, & Prévost, 2015). This is to say, there are any number of brain pathways whose alteration can impact perceptual accuracy and, in general, perceptual experience.

3 THE MODEL IN CONTEXT

Commenting on abnormal visual object recognition reported in several neuropsychiatric conditions, Laycock and colleagues have argued that a dorsal stream locus of impaired early visual processing is at play, resulting not only in anomalous perceptions and defects in image categorization, but also bias in how complex social interactions are judged. It is notable that abnormal neural activity within the visual system has been described in obsessive-compulsive disorder, social anxiety, and schizophrenia (Laycock, Crewther, & Crewther, 2007).

We recently applied these concepts to the study of body dysmorphic disorder (BDD), a phenotype marked by preoccupation with small or non-existent flaws perceived on the face or head area. Using own-face, other-face, and house stimuli as probes, our fMRI studies (Li, Arienzo, & Feusner, 2013) revealed abnormally reduced activity in the dorsal visual stream in persons with BDD when the images were filtered to convey only LSF (again, holistic) information. This observation lead us to hypothesize that the anxiety-driven, hyper-scrutiny of miniscule facial details could be mechanistically understood as compensation for failing to “see” the body as a sensory whole. The hypothesis gained solid support from subsequent imaging and electro-cortical evidence and studies of saccadic eye movements. Specifically, we showed not only is BDD characterized by enhanced ventral stream processing of high detail images, they also rate faces as more unattractive as the magnitude of detailed processing increases. As to eye gaze, persons with BDD also exhibit a biased attention to facial areas deemed flawed, and a scanning pattern characterized by multiple fixations of brief duration—a high velocity of saccades.

We next reasoned that body image disturbance in AN might share these same neural phenotypes in common with BDD. In support of the hypothesis, we indeed found comparable evidence of cortical hypo-activity in the dorsal stream in AN using the same probes as described above. Additionally, in AN eye-gaze search patterns have been found similar to that described in BDD—thus our belief that interrogation of the visual system may allow for novel causal inferences and the development of a uniquely effective mechanistic therapy.

Important to consider here is a key point about the psychopathology of AN and the architecture of complex traits in general. Specifically, it is beyond question that anxious traits appear early in the development of persons who later manifest AN, and that the very same traits are common in their first-degree relatives. This suggests that a relevant substrate for the disorder's identifying features of weight related fears, nervous scrutiny of the body, and compensatory food avoidance is the cortico-limbic circuitry causally implicated in anxiety, fear, and behavioral inhibition. Equally well established is that human traits are polygenic, that genes are modulated by environmental events, and that psychopathologies once assumed to be causally independent share genomic loci in common. In fact, for AN recent genetic analyses show correlations with schizophrenia, neuroticism, and obsessive-compulsive disorder, which, as noted, are associated with perturbations in the visual system. Clearly, if we are to delve deeply into the challenge of body image in AN, a systems-based and transdiagnostic integration of mechanistic knowledge is crucial.

4 TOWARD AN EXPLANATORY MODEL

Vigilance is mediated by structures that guide attention and eye movements, including the frontal eye fields (FEF), superior colliculus, thalamic nuclei, and lateral parietal cortex. Depending on inputs from the visual cortex, striatum, and cortico-limbic systems regulating emotion and cognitive control, eye movements (saccades) are excited, or constrained in what Schall, Purcell, Heitz, Logan, & Palmeri (2011) described as a “perceptual decision.” As eye movement velocity accelerates, processing of low spatial frequency (LSF) visual information by the dorsal pathway is suppressed; by contrast, processing of high fidelity details by the ventral stream is enhanced. Here we draw on a point made by Mobbs et al. (2015): Since extended hyper-vigilance (inherent to anxiety) drains energy supplies and impairs homeostasis, surveilling objects in the environment will be maximal efficient when vigilance is “fleeting and restricted to times when high risk situations are (accurately) predicted.” What this implies to us is that by manipulating attention allocation—specifically, reducing the velocity of eye movements, in effect constraining vigilance—neuronal activity within the dorsal visual stream could be enhanced, and thereby improve perceptual accuracy.

Accordingly, our model of distorted perception rests on the following assumptions: (1) the existence of a bottom-up deficiency in perceiving LSF detail resulting from hypoactivity of the dorsal stream; (2) aberrant functioning of fronto-limbic and striatal systems, evoking a compulsively driven scanning of negatively valenced appearance features, resulting in (3): shorter visual fixations—a high velocity of eye movements. The outcome is an anomalous visual consciousness, wherein details are not effectively integrated into a sensory whole but rather stand alone as “flaws” (analogous to simultagnosia, in which individuals accurately see individual details of a scene but fail to perceive its larger identity).

5 A PROPOSED THERAPEUTIC

Beilharz, Alkins, Duncum, & Mundy (2016) recently showed that exposure to a Navon figure (a larger recognizable letter formed by copies of smaller letters) after a prompt to focus on the global letter reduced a local processing bias in persons with non-clinical body image concerns. Although not directly examined, the effect is presumed to result from

enhancing the visual processing of global (dorsal) inputs. Might it be the case then, for reasons outlined above, that by restraining eye-gaze behaviors we could enhance dorsal visual stream neuronal activity, resulting in altered holistic perceptual processing?

5.1 The hypothesis

Modifying eye gaze behavior will enhance the processing of holistic stimuli in the dorsal visual cortex stream while suppressing neuronal activity in the ventral visual stream.

5.2 The manipulation

While being scanned using fMRI, in a naturalistic viewing condition participants will view either an unaltered, valence-neutral stimulus, or the same image but scrambled. In the visual attention modulation condition, they will see both images with a centered white cross and will be instructed to keep their gaze centered on the white cross. The analytics would involve dynamic functional connectivity methods to examine changes in directional-specific connections within dorsal and ventral visual stream networks over the course of the experiment.

5.3 Regarding the model in context

Here the question broadens:

1. Given the feedback systems that are involved in perception, does modulation of eye gaze alter other relevant neurofunctional regions and their connectivities—extended amygdala, hippocampus, orbital frontal cortex, striatum?
2. Does resting state (task-free data collection that is more translationally adaptable to clinical settings) functional brain connectivity in visual systems reveal neuromarker predictors of treatment response?
3. Might psychological interventions designed to reduce anxious states (e.g. to enhance tolerance of uncertainty) enhance the effects of visual modulation? The effects of anxiety deserve focus in the present study because, as mentioned, anxious states have been linked to altered visual processing and may be a function of top-down enhancement of ventral visual stream activity.
4. Are adverse early life events associated with the degree of dorsal stream hypoactivity, with the degree of body image distortion, or with responsivity to the visual modulation?
5. Does change in dynamic connectivity predict post-weight recovery outcome? If so, how many manipulation exposures are required for optimal effects?
6. As genomic studies of AN expand, might gene variations be identified that predict how responsive the visual cortex is to modulation.

6 CONCLUDING REMARKS

With knowledge gleaned from visual neuroscience as background, we have offered a plausible framework for understanding the mechanisms driving abnormal perception of the

body in AN, and a logically derived experimental target for intervention; however, there are caveats to note. Cash and Pruzinsky (1990) put it succinctly when noting that how body image is defined depends on which of its many dimensions is the focus of investigation. As an embodiment of multiple influences—perceptual, cognitive, and affective—there is no single, sufficient explanation of its formation, nor is a single causal pathway to its deviance likely (Badoud & Tsakiris, 2017; Gaudio & Quattrocchi, 2012; Gaudio, Wiemerslag, & Schioth, 2016); therefore, our model carries the risk of missing important aspects of body image within the individual, or within certain clinically defined subgroups. For example, the insistence that one's body is manifestly large need not imply a perceptual defect; it might stem from an overvaluation of thinness, some form of judgment bias related to an implicitly generated threat or criticism associated with "heaviness," or the anticipation of uncomfortable visceral sensations that accompany changes in the body's physiology and form with puberty. Also, the symptom is not universal. Clinical experience shows that some patients, though appearing incapable of restoring their weight (or, perhaps, resistant to the idea) nevertheless describe their appearance as frightening (see Strober, Freeman, & Morrell, 1999). Whether inputs to the visual cortex differ between these subgroups is a question of importance that can be addressed in our model. Thus, we acknowledge that there are alternative possibilities underlying how the body is represented in AN. Still, in addition to research from our laboratory cited above, there is corroborating evidence of a perceptual factor, including decreased retinal fiber thickness, deficits in hemispheric integration of multisensory information, size overestimation, and visuospatial functioning characterized by weak central coherence—a bias toward the processing of detail over holistic information (see Badoud & Tsakiris, 2017; Farrell, Lee, & Sharan, 2005; Gardner & Brown, 2014; Gaudio & Quattrocchi, 2012; Gaudio, Wiemerslag, & Schioth, 2016; Lang, Lopez, Stahl, Tchanturia, & Treasure, 2014; Madsen, Bohon, & Feusner, 2013; Smeets, Smit, Panhuysen, & Ingleby, 1997; Weinbach, Perry, Sher, Lock, & Henik, 2017).

Clearly, the corrective intervention described does not yet translate readily into standard clinical practice. Even so, it might open the door for future creative, hypothesis-driven attention allocation approaches that do not require imaging of the brain to verify the effects. Finally, as the number of questions listed, certainly not exhaustive, is large, the importance of an iterative approach for any future examination of this idea worth researching is self-evident.

Acknowledgments

The authors declare they have no conflict of interest. Funding for this manuscript was provided by NIMH grant R01MH093676 (Feusner & Strober), and from the Resnick Family Endowed Chair in Eating Disorders (Strober).

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