Thus, how can reward sensitivity (a general measure of sensitivity to reward) linked to the putamen in all eating disorder groups in the Frank et al. study (1) be reconciled in relation to taste reward or reward and punishment sensitivity that was found in their previous study (2)?

Finally, given that bulimia nervosa is characterized by uncontrollable bouts of eating, whereas sustained food avoidance is characteristic in anorexia nervosa, how does the model proposed ("greater gyrus rectus volume in eating disorders is associated with stronger sensory experience of food stimuli… which could trigger cognitively driven food avoidance") account for differences between anorexia nervosa and bulimia nervosa? Perhaps most important, however, is that the described pattern of greater gyrus rectus volume in eating disorders being associated with stronger sensory experiences of food stimuli is the pattern for healthy comparison subjects; hence, the authors should be cautious as to pathologizing the healthy control relationship.

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Response to Keating and Rossell

TO THE EDITOR: The correlation between gyrus rectus volume and sweet taste pleasantness in the comparison group is striking as it supports the previous literature and is consistent with an important concept established by the works of Rolls, Kringelbach, and others (1). All eating disorder groups in our sample showed increased gyrus rectus volume relative to individuals in the comparison group, suggesting comparable pathophysiology. Groups were combined for that reason, and we did find a positive correlation between sweet taste pleasantness and volume in the eating disorder case subjects. In addition, within eating disorder subgroups, each individual group showed a positive correlation (anorexia nervosa, r=0.120; bulimia nervosa, r=0.287; recovered anorexia nervosa, r=0.345) between sweet taste pleasantness and gyrus rectus volume, supporting the idea that analyzing the groups as a whole was valid. The correlation coefficients were modest. Despite a significant amount of research, we still know little about the connection between brain structure and function and these results are a step toward identifying target areas that could contribute to altered food intake. Taste pleasantness in individuals with eating disorder could be less well mapped (yet still positively) onto gyrus rectus neurons due to the altered volume, and this could interfere with correct value computation.

It is important to view eating disorders as complex problems with a variety of factors that most likely contribute to development and then perpetuation of the illnesses. Expecting direct correlations between all those measures would be overly simplistic, and we believe that dissecting eating disorders by brain circuits that may contribute to the psychopathology will be more fruitful. Trait alterations seem to exist that cause heightened sensitivity to various (food- and nonfood-related) rewarding or punishing stimuli, contributing to a sense of instability across individuals with anorexia and bulimia nervosa (2, 3). This instability may contribute to heightened anxiety and intolerance of uncertainty (4). Additionally, as suggested in this study, there may be developmental factors such as altered brain volume in the orbitofrontal cortex that could interfere with the processing of value of food stimuli. We recently published a similarly designed study in adolescents with anorexia nervosa and found similarly larger gyrus rectus volumes, further supporting that this structure could be involved in the pathophysiology of eating disorders (5).

All eating disorder groups share an enlarged gyrus rectus, which could impair a healthy sensory satiety processing. In fact individuals with eating disorders share many similar behaviors but on different scales, and there is overlap in symptoms not only across subgroups but even depending on stage of recovery. For instance, individuals with bulimia nervosa are often able to restrict food intake in-between binge episodes. Furthermore, many individuals who meet criteria for one category of eating disorder will often shift into another eating disorder during the course of illness, which is consistent with a common vulnerability.

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