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Dopamine for "wanting" and opioids for "liking": a comparison of obese adults with and without binge eating.

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Abstract

Obesity research suffers from an overinclusion paradigm whereby all participants with a BMI beyond a certain cutoff value (e.g., 30) are typically combined in a single group and compared to those of normal weight. There has been little attempt to identify meaningful subgroups defined by their salient biobehavioral differences. In order to address this limitation, we examined genetic and psychological indicators of hedonic eating in obese adults with (n=66) and without (n=70) binge eating disorder (BED). Our analyses focused on dopamine (DA) and opioid genetic markers because of their conjoint association with the functioning of brain reward mechanisms. We targeted three functional polymorphisms related to the D2 receptor (DRD2) gene, as well as the functional A118G polymorphism of the mu-opioid receptor (OPRM1) gene. We found that significantly more obese controls had the "loss-of-function" A1 allele of Taq1A compared to their BED counterparts, whereas the "gain-of-function" G allele of A118G occurred with greater frequency in the BED group. A significant gene-gene combination chi2 analysis also indicated that of those participants with the gain-gain genotype (G+ and A1), 80% were in the BED group whereas only 35% with the loss-loss genotype (G- and A1+) were in this group. Finally, BED subjects had significantly higher scores on a self-report measure of hedonic eating. Our findings suggest that BED is a biologically based subtype of obesity and that the proneness to binge eating may be influenced by a hyper-reactivity to the hedonic properties of food--a predisposition that is easily exploited in our current environment with its highly visible and easily accessible surfeit of sweet and fatty foods.

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