

Brain Imaging and Bulimia Nervosa

Over the past two decades, neurobiological mechanisms underlying bulimia nervosa (BN) have been studied in human patients using brain imaging techniques. Similar to individuals with anorexia nervosa (AN), results of computerized tomography (CT) scans indicate that individuals with BN also display enlarged ventricles and/or sulcal widening (Krieg, Lauer, & Pirke, 1989). Studies utilizing magnetic resonance imaging (MRI) have demonstrated that individuals in recovery from BN show persistent decreased activation in the right anterior cingulate cortex and the left cuneus (Frank et al., 2006); whereas, those in long-term recovery show restoration to normal levels of cerebrospinal fluid (CSF) as well as gray and white matter volume (Wagner et al., 2006). Though results of single positron emission computerized tomography (SPECT) studies suggest that serotonin binding among BN patients does not differ significantly from individuals without BN (Goethals et al., 2004; Koskela et al., 2007), it is possible that serotonin binding differs among purging and non-purging individuals with BN (Koskela et al., 2007). Another SPECT study demonstrated significant differences in cerebral blood flow during bingeing and restricting phases (Hirano, Tomura, Okane, Watarai, & Tashiro, 1999).

In general, positron emission tomography (PET) studies have demonstrated differences between individuals with BN and health controls in cerebral blood flow within the frontal, temporal, and parietal cortical regions as well as the cingulate cortex (Kaye et al., 2005). However, it appears that these differences disappear with long-term recovery (Frank et al., 2007). When using PET imaging to examine serotonin binding among individuals in long-term recovery from BN, researchers demonstrated a persistent reduction in the medial orbital frontal cortex compared to healthy controls (Kaye et al., 2001). Finally, results of a functional magnetic resonance imaging (fMRI) study demonstrated that women with BN display lower activation than healthy controls in the lateral and apical prefrontal cortex, and women with any eating disorder showed greater activation than controls in the left medial orbitofrontal cortex and anterior cingulate cortex, as well as lower activation in the lateral prefrontal cortex, inferior parietal lobule, and cerebellum (Uher et al., 2004).

References

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