

Are there cerebral abnormalities in eating disorders?

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A report from the University of Freiburg that is published in one of the last issue of *Psychotherapy and Psychosomatics* address the presence of cerebral abnormalities in eating disorders.

Most [eating disorders](#) (ED) patients – independent of diagnostic category – are characterized by an overevaluation of shape and weight, a strong dissatisfaction with their body and an engagement in some form of weight control behavior. They often switch diagnostic categories over time, and there is genetic cross-transmission. Furthermore, about half of ED cases do not fulfill all criteria of [anorexia nervosa](#) (AN) or bulimia nervosa (BN).

This study raises the question whether the increasing number of neurobiological studies, in particular imaging data, might contribute to this debate. One of the first functional [magnetic resonance imaging](#) (fMRI) studies was suggestive of functional cerebral substrates common to various EDs, demonstrating increased reactivity of the [medial prefrontal cortex](#). Investigations using visual body images yielded amygdalar activation in AN, but not in BN.

An increased response of the right amygdala and decreased signals of the midcingulate cortex in restrictive AN was also demonstrated, while BN patients had decreased frontocingular and temporal signals. A further recently published [functional magnetic resonance imaging](#) (fMRI) investigation demonstrated differences in [blood oxygen level](#)-dependent signals which comprised the cingulate cortex as well as the parietal,

temporal, insular, supplementary motor and subcortical (caudate nucleus) regions. Furthermore, using [magnetic resonance](#) spectroscopy of the anterior cingulate cortex, BN patients showed a positive correlation of glutamate with 'drive for thinness', whereas restrictive AN subjects did not.

With respect to ventromedial dysfunction (including the [anterior cingulate cortex](#)), it should however be kept in mind that this occurs in other mental diseases, too. Structural cerebral imaging also demonstrated strong differences between AN and BN with reduction of cingular and temporoparietal grey matter in AN, whereas BN was not affected in whole brain analyses.

In summary, from a neurobiological imaging point of view, there is evidence of functional and structural cerebral differences between BN and AN. Considering diagnostic issues, these results seem in favor of keeping a categorical approach. However, a complementary dimensional approach might be reasonable for specific pathopsychological features like drive for thinness, which are shared by most EDs, as shown by a positive correlation of grey matter volume of the right parietal cortex and drive for thinness in both restrictive AN and BN. These findings support the notion that biological features should be considered when discussing the classification of psychic syndromes in the future. Furthermore, considering these neurobiological facts might also form a bridge between neurobiological research and clinical thinking.

More information: Joos, A. et al. Distinct Functional and Structural Cerebral Abnormalities in Eating Disorders in the Light of Diagnostic Classification Systems. *Psychother Psychosom* 2012;81:394–395.

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