With DSM-5 advancing there is a debate with respect to the classification of eating disorders (EDs). In principle, the categorical approach of DSM-IV will be kept, with anorexia nervosa (AN) and bulimia nervosa (BN) representing the main ED entities. However, there are also rationales to conceptualize ED as a continuum: Most 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 AN or BN. Here we raise 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 [1]. Recent fMRI studies have shown different results. One study that used words concerning body image demonstrated an increased response of the right amygdala in AN, while ventromedial abnormalities characterized BN [2]. Investigations using visual body images yielded amygdalar activation in AN, but not in BN [3, 4]. Our group used a food paradigm, which demonstrated an increased response of the right amygdala and decreased signals of the midsigmoid cortex in restrictive AN [5], while BN patients had decreased frontocingulate and temporal signals [6]. Contrasting these samples directly, BN was characterized by decreased frontocingulate activation, as shown in figure 1. A further recently published fMRI investigation used the active instruction to imagine eating the food shown in the images and compared AN with BN patients [7]. Again, differences in blood oxygen level-dependent signals were demonstrated, which comprised the cingulate cortex as well as the parietal, temporal, insular, supplementary motor and subcortical (caudate nucleus) regions [7]. A photon emission computed tomography study reported almost reverse perfusion patterns of AN and BN when subjects were compared in the resting condition and after food intake [8]. 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 [9]. With respect to ventromedial dysfunction (including the anterior cingulate cortex), it should however be kept

### Letter to the Editor

**Distinct Functional and Structural Cerebral Abnormalities in Eating Disorders in the Light of Diagnostic Classification Systems**

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1. Joos et al. [5] and Joos et al. [6]. At a less conservative threshold, i.e. when not correcting for multiple comparisons across the whole brain, two further clusters reached a p value <0.01 at the cluster level: the dorsal anterior cingulum [p_{corr. cluster level} = 0.003, z-value of peak voxel 3.99 at MNI 9 51 21] and the left superior frontal lobe [p_{corr. cluster level} = 0.008, z-value of peak voxel 3.23 at MNI –9 48 22].
in mind that this occurs in other mental diseases, too [10]. Structural cerebral imaging also demonstrated strong differences between AN and BN with reduction of cingular and temporoparietal grey matter in AN [11], whereas BN was not affected in whole brain analyses [11, 12].

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 [11]. We think that 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.

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