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# Unravelling the genetic and neuroendocrine basis of adolescent eating disorders

Johannes Hebebrand[1]

[1]Department of Child and Adolescent Psychiatry, Psychosomatics and Psychotherapy, University of Duisburg-Essen, Essen, Germany

The three major eating disorders anorexia nervosa (AN), bulimia nervosa (BN) and binge eating disorder (BED) can be diagnosed according to DSM5; in total these disorders effect up to 6% of adolescents. The overview will focus on the relationship between eating and weight disorders and attempt to associate psychopathology with specific body weight categories. Recent genome wide association studies (GWAS) and meta-analyses thereof point to an overlap between genetic factors predisposing to a low body weight and those involved in the genetic predisposition to AN. Genetic correlations have also been found between body mass index (BMI; kg/m$^2$) and other psychiatric disorders including schizophrenia. The premorbid body weight in AN patients has been shown to be in the normal or lower range; in contrast, overweight has been identified as a risk factor for the development of BN and BED. Because primary symptoms of AN are tightly intertwined with symptoms secondary to starvation, specific symptoms of the disorder are potentially amenable to treatment via targeting of the neuroendocrine basis of starvation. In particular, the hormone leptin appears promising to reduce starvation related hyperactivity. In addition, exogenous application of leptin may reduce an addictive like restrained eating behavior and reduce some eating disorder specific cognitive symptoms.