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Peripheral endocannabinoids in eating disorders and obesity



Individuals with eating disorders show a deficiency of the endocannabinoid system, a complex intercellular communication structure involved in the regulation of processes such as appetite, mood and addictive behaviors. A study co-directed by Professor Roser Granero compares the levels of different endocannabinoids between healthy patients and patients with eating disorder subtypes such as anorexia and bulimia, with results that may have therapeutic implications.

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The endocannabinoid system (ECS) is a complex intercellular communication structure with a role in regulating multiple neuro-physiological processes (including appetite, pain sensation, mood, learning and memory, emotion and motivation, and addictive behaviors), with the aim to balance metabolic processes and optimize the function of the bodies to the day-to-day experiences. ECS plays also an important role in the control of eating behavior by acting via central (brain) and peripheral (gut, liver, muscle and fat) mechanisms. The CB1 receptor (widely expressed in brain regions that control food intake, reward and energy balance) is considered the main responsible for most of the central and peripheral effects of cannabinoids on the eating behavior. CB1 receptor agonists possess or exigenic effects enhancing appetite and increasing the rewarding value of food. Conversely, CB1 antagonists have been shown to inhibit the intake of food.

On the other hand, eating disorders (ED) include a range of mental disorders characterized by persistent aberrant patterns of feeding behaviour and weight regulation, as well as a set of abnormal attitudes and perceptions toward the body shape image. These symptoms negatively affect the individuals' health state, including physical, psychological and social functioning. The most prevalent ED subtypes within population-based and clinical samples are anorexia nervosa (AN), bulimia nervosa (BN) and binge eating disorder (BED).

Given the strong link between ECS and the reward circuitry, it has been hypothesized a potential contribution of ECS in the processes underlying the motivation to eat and in the pathophysiology of ED. The results observed in a few clinical studies have showed that patients with ED evidence an abnormal functioning in the ECS, including the incentive processes stimulating behaviors towards food acquisition or the hedonic evaluation of ingested food. However it is unknown if the diverse ED-subtypes are related to different ECS profiles.

A study conducted by Doctors Fernando Fernández-Aranda and Susana Jiménez-Murcia (Psychiatric Service of the University Hospital of Bellvitge) and Dr. Roser Granero-Pérez (Department of Psychobiology and Methodology, UAB) compared fasting peripheral levels of endocannabinoids anandamide (AEA) and 2-Arachidonoylglycerol (2-AG) in eating disorder (ED) patients, obese subjects (OB) and healthy controls (HCs). This study also explored the association of AEA and 2-AG with clinical and anthropometric variables. The sample included a total of n=63 adult women (22 AN, 9 BED, 21 OB and 11 HC). Peripheral blood samples were collected to investigate fasting levels of AEA and 2-AG.

The global comparison between the groups showed significant differences in AEA levels. Specifically, individuals with AN exhibited lower AEA than OB and BED, while OB showed higher AEA than HCs. In addition, 2-AG was positively correlated with hostility dimension in EDs and negatively associated with impulsive traits in OB. AEA showed a direct association with body dissatisfaction in AN, contrary to OB. Finally, within the AN group, AEA negatively correlated with the body mass index, while 2-AG was positively associated with the fat mass.

The results of this research reinforce the evidence that AEA and 2-AG could play a role in food intake and pleasure-reward aspects of feeding. New studies are needed to assess whether the observed differences between the ED subtypes are caused by the disease or whether these are neurochemical alterations that serve as risk factors for developing an specific ED. This study also suggests an interaction between biological and clinical factors defining differentiate vulnerability pathways that could help fitting personalized therapeutic approaches.

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