Endocannabinoid and Gastroenteric Hormone Responses to Hedonic Eating in Healthy Humans: Implications for Eating Disorders

Monteleone AM¹, Nigro M¹, Caivano V², Perillo D¹, Di Marzo V³, Monteleone P¹,²

¹Department of Psychiatry, University of Naples SUN, Naples
²Neuroscience Section, Department of Medicine and Surgery, University of Salerno, Salerno
³Endocannabinoid Research Group, Institute of Biomolecular Chemistry, Consiglio Nazionale delle Ricerche, Pozzuoli (NA), Italy
(corresponding author: alessio.monteleone@fastwebnet.it)

In hedonic eating food is consumed uniquely because of its gustatory rewarding properties, so the subject eats also when not in a state of energy depletion. It is intuitive that hedonic eating may stimulate powerfully food intake; hence, understanding its physiological modulation could help to contrast eating disorders.

In order to explore the role of endogenous appetite and/or reward modulators in hedonic eating, we measured plasma levels of ghrelin, endocannabinoids, colecystokinin-33 (CCK) and peptide YY³-36 (PYY³-36), in 8 satiated healthy subjects after ad libitum consumption of highly palatable food as compared to the consumption of isoenergetic non-palatable food.

Hedonic eating was characterized by increased plasma levels of ghrelin and the endocannabinoid 2-arachidonoyl glycerol (2-AG) but a decreased secretion of the satiety hormone CCK. In both eating conditions, levels of the other endocannabinoids anandamide, oleoylethanolamide and palmitoylethanolamide progressively decreased while no significant changes in plasma PYY³-36 occurred.

Present findings suggest that when motivation to eat is generated by the availability of highly palatable food and not by food deprivation, a peripheral activation of the rewarding and hunger-promoting signals of 2-AG and ghrelin, but a decrease of the satiety signal of CCK occur. This could be responsible for the persistence of peripheral cues allowing a continued eating in spite of no energy need.