Regulation of Nociceptin/Orphanin FQ system gene expression in Binge Eating episodes

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Episodes of binge eating (BE) in humans are characterized by compulsive, non-homeostatic consumption of an unusually large quantity of highly palatable food (HPF) in a short period of time. These episodes represent a central feature of bingeing related eating disorders, such as binge eating disorder, bulimia nervosa, and binge/purge subtype anorexia nervosa [1]. Binge eating is also an additional behavioral feature of obese individuals, significantly contributing to their high caloric intake and finally being overweight.
Many studies already suggested a substantial genetic influence in eating disorders and a dysregulation for different neurotransmitter, neuropeptide, and neuroendocrine systems, also at genetic level, have been reported [2]. Among them we recently underlined an important role for the Nociceptin/Orphanin FQ (N/OFQ) system in BE [3].
In order to provide further evidence on its involvement in BE development, we here studied the genetic and epigenetic regulation of the peptide N/OFQ. We used a well-validated animal model of BE which include four groups (rats fed normally and not stressed or stressed, rats exposed to cycles of restriction/refeeding and then stressed, or not stressed). Selectively in the group of rats restricted and stressed, which also exhibited BE for HPF, we observed an up-regulation of proN/OFQ ($p<0.01$ *vs* not restricted non stressed group) rat in the amygdala complex, brain region associated with stress responses. At the moment, DNA methylation studies at gene promoters did not provide a clear role for epigenetic mechanisms of this gene regulation in the amygdala complex. In agreement with our previous observations, we thus here further suggest that an altered regulation of N/OFQ system in response to food restrictions and stress might represent a very important event in the predisposition to binge-type eating disorders.

[1] American Psychiatric Association 2000 Diagnostic and statistic manual of mental disorders, IV-TR. APA
[2] Kelley A.E. et al. 2005 Physiology and Behavior 86:11–14
[3] Micioni Di Bonaventura MV et al. 2013 Psychopharmacology 2013 Mar 2 in press DOI 10.1007/s00213-013-3013-0