

Epigenetic Mechanisms on Food Addiction

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Editorial

In the last years, the way of the eating behavior has strongly changed. Western Countries are assisting to several changes in food culture, revealing a tendency to use always more frequently and increasingly amounts of those aliments once considered rare and valuable. The recent tendency to eat over the necessary, with significant imbalances of the nutrients of the diet, has induced a higher incidence of eating disorders (ED). Especially, the excessive consumption of sugar-based foods has contributed, together with other environmental risk factors (dieting, media exposure, body image dissatisfaction, and weight-related teasing), to an increase of cases of over-eating diseases, such as obesity, binge-eating disorder, and bulimia nervosa [1-4]. Neuroscience focused the research strategies on the possibility that a maladaptive eating behavior can characterize some eating disorders as well as maladaptive drug intake characterizes drug addiction. Thus, maladaptive binge-eating might be considered an “addiction” in its own right. Recently, DSM-V merged two main features of drug addiction into the diagnostic criteria of maladaptive eating-related symptoms: A strong craving and a pattern of compulsive use [5]. It has been recognized that the resemblance may reflect the involvement of the same neural systems, including those implicated in regulatory self-control and reward in both groups of disorders. It has been widely demonstrated that the DAergic “meso-limbic” reward system has a crucial role not only in the passage from an occasional use to an abuse of drugs, but also in the transition from a normal feeding to a maladaptive compulsive eating behavior [1,3,6-8]. Human studies showed that DA-ergic release correlates with the reward from both drug and food use [9,10]. Both addictive drugs and palatable foodstuffs exposure produce neuro-adaptations (e.g., a repeated stimulation of mesolimbic DA-ergic neurotransmission) that lead to compulsive drug- and food-seeking behavior [9,11,12]. Moreover, several studies found an implication of genes involved in weight regulation, eating behavior, mood, and stress responsivity [9,13]. Genetic variants in eating disorders have been investigated [14-16], and the Taq1A (minor) allele of DA receptors D2 (DRD2) gene is associated with many of substance-misuse disorders [16-18]. Furthermore, it has been defined that lower levels of striatal DRD2s can signal a “reward-deficiency” state leading to over-seeking/taking behaviors [7], thus suggesting that low DRD2 availability could be a formative genetic risk factor in compulsive food seeking/taking behavior [3]. Finally, is conceivable that gene-environment interactions are able to induce neuroadaptations in several brain areas related to substance use disorders, such as the DAergic “meso-limbic” reward system.

Recently, neuroscience moved its interest on epigenetic studies related to the biological mechanisms supporting the behavioral changes and, specifically, those behaviors learned during the establishment of the illness. Epigenetics is defined as the study of cellular and physiological phenotypic trait variations caused by environmental factors switching on or off different genes and affecting the genes-cells encoding through changes in DNA and associated histones and via the action of small non-coding RNA molecules [19]. The best known epigenetic modification is DNA methylation on the cytosine pyrimidine ring [20]. A third epigenetic system involves small interfering RNA (siRNA) that

suppress the activity of specific genes via RNA interference (RNAi), a process likely to be integral to developmental gene expression [21]. Epigenetic changes in DNA or associated histones coordinate gene expression during brain development. Epigenetic changes are dynamic during prenatal and perinatal periods [22]. These developmental moments are relevant factors associated with the risk of over-eating disorders and obesity. Furthermore, epigenetic modifications produce a range of phenotypes from genetically identical cells. Finally, several environmental-sensitive genetic changes, such as maternal stress and dieting behavior may alter the risk of developing an eating disorder.

Recently, because drug- and food-seeking behavior share similar brain areas and neurotransmission systems, it has been suggest the possibility to produce animal model of maladaptive over-eating disorders [1,6,8,23-25] using the same, adapted paradigms used to investigate drug addiction [8,24,26]. Furthermore, genetic animal models, such as transgenic mice and conditional knock-out mice have been used to investigate the possible role of epigenetic mechanisms underlying drug and food addiction [27-29].

Finally, beyond the necessity to better investigate the epigenetic mechanisms in eating-related behavior and Food Addiction, more is needed to know about the possible investigative tools and usable techniques in this field of research.

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