are in the same time obese and/or present binge-eating behaviours, so confounding factors could be detected very frequently [1]. „Eating addiction" is preferred by some authors to the term „food addiction", because eating, a behaviour, becomes an addiction, while the evidence for food provoking dependence, similar to a drug of abuse, are sparse [2].

A particular case of food-addiction is „sugar addiction", which is based on the observation that highly processed foods, especially those with high sugar content, are highly addictive [3]. Sugar exerts its powerful reinforcing effects through both gustatory and post-ingestive pathways [4].

Data from rodent studies support this observation, and this may translate to humans in certain conditions [5]. During sugar intake, suppressing hedonic value inhibited dopamine release in ventral striatum, while suppressing nutritional value inhibited dopamine release in dorsal striatum [4].

However, a review of the data from literature that included human studies didn't support the existence of this particular case of food addiction [3].

3.1. Psychometric evaluation

The Yale Food Addiction Scale (YFAS) was developed for quantifying the severity of food addiction, and updated to YFAS 2.0 after the publication of DSM-5 [6].

Elevated scores on YFAS 2.0 correlated with higher rates of obesity and more severe pathological eating, and both versions of YFAS were similarly associated with elevated body mass index, binge eating, and weight cycling [6].

3.2. Prevalence and risk factors

The prevalence of food addiction, according to a meta-analysis of 25 studies (N=196211 subjects), was 19.9% using YFAS scores [7]. Subjects included in meta-analysis with higher risk for food addiction were adults over 35 years, female, overweight/obese, and subjects from clinical samples [7].

All female patients with current bulimia nervosa received a food addiction diagnosis according to the YFAS score, while only 30% of the women with remitted bulimia nervosa had significant YFAS scores [8]. This suggests that bulimia nervosa is similar to an addiction-like behaviour, and symptoms of the food disorder are probably remitting when bulimia nervosa symptoms disappear [8]. This could be perceived as a proof that food can impair individual's self-control, similarly to any other drug [9].

A number of peripheral and central biological dysfunctions observed in bulimia nervosa lead to altered reward sensitivity in these patients, a phenomenon with dopaminergic mediation [9].

Positive correlation between food addiction and depression scores has been detected in a large group of university students [10].

3.3. Pathophysiology

Psychoactive drugs with abuse potential and palatable foods present similar ways of rewarding the consumer. Arguments for this hypothesis arise from neurobiology and clinical psychiatry: activation of dopaminergic rewarding system is common in both situations; tolerance is observed in drug-users but also in patient who abuse food; withdrawal is currently observed in patients with drug dependence, but withdrawal-like phenomena are detected in persons who abuse palatable food [11].

Some authors propose that highly processed foods have similar pharmacokinetic properties with drugs of abuse, d.e. concentrated dose, or rapid rate of absorption, which could explain the highly addictive properties of this kind of food [12]. This hypothesis was tested experimentally, and processed food, higher in fat and glycaemic load, were more frequently associated with problematic, addictive behaviours, probably due to their ability to induce a faster absorption of fat/sugar into the bloodstream [12].

An analysis of the pathways involved in food addiction, obesity and drug dependence realised by EM Schulte et al. [13] detected several common elements for these disorders: reward dysfunction, impulsivity and emotion dysregulation.

Regarding the reward system, which is essentially a dopamine based circuitry, it has been reported that in humans, consumption of palatable food induced activation of this circuitry, including dorsal- and ventral striatum and orbito-frontal cortex [14]. Consumption of drugs or palatable food in a repeated manner may result in sensitization of the dopaminergic system, with an increase of drugs/food salience [15].

The second common feature, impulsivity, is observed experimentally when obese patients and persons with addiction favor short-term reward of drug/food instead of long-term health health [13,16].

The third factor, emotion dysregulation, is reflected in the precipitation of drug/food intake when strong emotional states are felt, as a measure

to compensate for deficient emotional regulation function [17].

Many gene polymorphisms have been implicated in drug, food and other behavioural addictions [18].

A suggestion regarding the pathophysiology of food addiction is that a decrease of D2 receptors population (DRD2) could be induced by food, like any other drug; a meta-analysis of the studies focused on this aspect showed no difference in body mass index in patients with A1/A1 and A1/A2 alleles comparative to those with A2/A2 alleles [19]. Several authors reported an association between greater carbohydrate and fast food craving and DRD2 A1 (a dopamine-resistant polymorphism) versus A2 allele among Asian Americans [20].

3.4. Treatment

Food addiction is a multidimensional pathology which requires an integrated approach, with psychotherapy, pharmacotherapy, and social oriented support [21]. Due to the absence of well-defined diagnosis criteria for food addiction, no clinical trial focused on treatment efficacy for patients with this disorder was identified in the literature. Data regarding the treatment are derived from trial with other, related, eating disorders.

Serotonin has an important role in modulating food and drug reinforcement [22].

Lorcaserin is a 5HT2C receptor agonist recommended for the treatment of obesity, but it has also been administered in patients with substance abuse, obsessive-compulsive, and gambling disorder [22].

SSRIs lead to the highest rate of symptoms reduction in binge eating disorder, in placebo-controlled trials, and duloxetine induced also encouraging results [23].

Antiepileptic drugs, opioid antagonists, acamprosate, memantine, and sodium oxybate were also studied in clinical trials with binge eating disorder diagnosed subjects [23].

4 Conclusion

Food addiction is an elusive diagnosis, and no well-defined, either experimentally- or clinically-based criteria were detected in the literature. Still, a psychometric instrument for quantifying this addiction's severity is widely used for research purposes.

Neurobiological and behavioural features of food addiction are common with other addictions have been described in the literature.

Data from genetic studies are still sparse, but less functional dopamine 2 receptor allele has been associated with food addiction and other rewarding deficit syndromes.

No clinical trial focused on food addiction's treatment has been identified in the literature, and a high degree of overlap between eating disorders could be an obstacle for creating such trials.

Continuing the search of diagnosis criteria for food addiction could lead to the possibility of finding new treatments for this and other behavioural addictions. Understanding food addiction could be an important progress on the pathway to finding more effective therapies for obese patients [24].

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