ISSN: 0975-833X

Available online at http://www.journalcra.com

INTERNATIONAL JOURNAL OF CURRENT RESEARCH

International Journal of Current Research
Vol. 14, Issue, 03, pp.21086-21093, March, 2022

DOI: https://doi.org/10.24941/ijcr.43291.03.2022

RESEARCH ARTICLE

PSYCHIATRIC AND PSYCHOLOGICAL INTERLINK AGES WITH FOOD ADDICTION ON HEALTH QUALITY AMONG YOUNG ADULTS – A SYSTEMATIC REVIEW

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ARTICLE INFO

Article History:
Received 24th December, 2021
Received in revised form
19th January, 2022
Accepted 24th February, 2022
Published online 30th March, 2022

Keywords:

Food Addiction,
Hyperpalatable Foods,
Obesity, Eating Disorders.

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ABSTRACT

The concept of "food addiction" has received increasing attention in recent clinical and experimental investigations (1,3). While cogent arguments have been made against the establishment of food addiction as a psychiatric diagnosis in its own right, there is a substantial evidence to suggest a significant role in the process of substance abuse disorders(4). Proposed review research has inherent limitations based on the scarcity of data on food addiction derived from clinical trials, which seriously limits the possibility of treatment recommendations (5). Diagnostic criteria for "food addiction" are controversial, and the heterogeneity of the studied population also limits the possibility of formulating screening strategies that are already implemented for other addictive disorders (11). Food addiction could possess strong implications on various aspects of life (12). Food addiction, if left ignored or untreated it can rapidly alter the biomechanics and health quality of an individual (5). Furthermore, validating data base relative to the concepts of toxic and lethal doses of hyper palatable foods on humans are crucial, as doses below the toxic and/or lethal range perhaps play a causal role in inducing intoxication or death. Evidently formulating a cohesive approach to diagnosis and treatment uniquely enable a vital impact on the proliferation of food addiction in the coming years.

Citation: Shreeya, R., Rajesh Kumar, Shweatha, H.E. and Sushma B. V. "Psychiatric and psychological interlink ages with Food addiction on health quality among young adults – A Systematic review", *2022. International Journal of Current Research, 14, (03), 21086-21093.*

INTRODUCTION

The term food addiction was first introduced in the scientific literature in 1956 by Theron Randolph. Although comparisons between addiction and eating behavior were sporadically drawn in the following decades, approaches to systematically examine and define food addiction were not pursued until the early 2000s (20). Food addiction is a controversial diagnosis which is not included in the current classificatory systems created by either American Psychiatric Association or World Health Organization. Also, no unanimously accepted, well-defined diagnosis criteria were detected in the literature during this review. However, the vast majority of the found papers used the same criteria for food addiction that are commonly used for substance use disorders.

A set of psychometric instruments has been validated (YFAS, mYFAS, YFAS 2.0, YFAS-C) for quantification of the food addiction severity in adult and children populations (25). As in the case of other behavioral addictions, the neurobiological, and psychological factors contributing to the food addiction pathophysiology are common with other substance use disorders. The main explanation for the pathogenesis of food addiction remains a dysfunction in the reward system. Similar clinical, neurobiological, psychopathological, and sociocultural risk factors have been identified in food addiction and substance use disorders. Data derived from genetic studies are still sparse, but the less functional dopamine 2 receptor allele has been associated with food addiction and substance dependence (15, 27). Food addiction has been also described as "eating addiction" or "eating dependence" by several researchers, who placed the emphasis on the behavior and not on the food itself.

High-sodium foods, artificially flavored-foods, rich carbohydrate- and saturated fats-containing foods are triggers for the activation of the same neural pathways, therefore they act similarly to any drug of abuse (30). Craving refers to an intense desire to consume a substance and frequent experiences of craving are a core feature of substance use disorders (SUDs). However, the term craving does not only refer to drug-related, but also to other substances like food or non-alcoholic beverages. In Western societies, individuals usually crave foods that are high in sugar or fat (or both) and, thus, highly palatable. Accordingly, the most often craved food is chocolate, followed by pizza, salty foods, ice cream and other sweets and desserts but there also cultural differences in the types of food craved. These same types of foods are more likely to be consumed in an addictive-like manner as assessed by the YFAS. As such, experiences of craving are a prime example of the similarities between eating and substance use (13,19). Similarly, activation patterns of neuronal structures underlying craving experiences largely overlap across different substances, including food. Overeating is associated with more intense and more frequent experiences of food craving. For example, higher scores on self-reported food craving measures have been found in patients with BN, BED, or obesity (2,6). Similarly, food addiction as measured with the YFAS is also related to higher self-reported food craving. Thus, the criterion of frequently experiencing craving or a strong urge to consume a substance can be translated to food and represents an important symptom in food addiction (8,26).

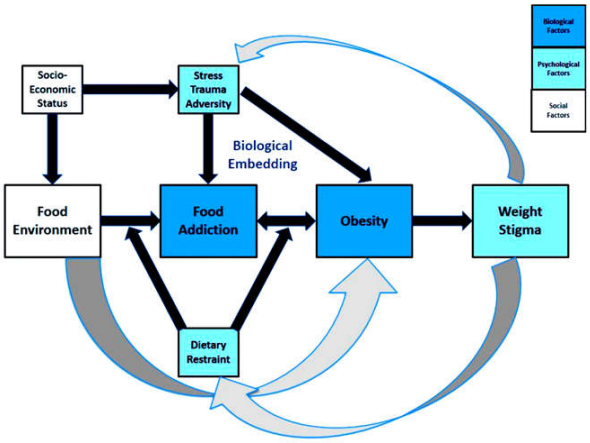

Fig 1. Food addiction and obesity following exposure to stress, trauma, and adversity: A biopsychosocial perspective of contextual factors. Davis C. (2014)

METHODOLOGY

The proposed systematic review throws light on the physiological and psychological significance in connection to the food addiction on health productivity.

Research Design: investigation aims at analyzing scientific review and original article findings from advance science, Elsevier, Blackwell and peer reviewed journals. With respect to the objectives; the research criteria include scientific articles evidences those in English and full-text from experimental, in-vivo and in-vitro studies.

Documentation: Citations used for the review study (google scholar, Research Gate, PubMed, Semantic Scholar and Science Direct).

Criteria: 80 papers were pre-reviewed, among which 42 papers were included as part of the research criteria and papers which didn't meet up criteria in relevance with the objective of establishing review findings were excluded.

Data acquisition /analysis: Among 53 research papers included: Review article (22), experimental studies (8), and original articles(12)

Findings *: Data emphasizes on the Prevalence, physiological and psychological aspects in connection to the food addiction, health productivity among young adults with food addictiion.*

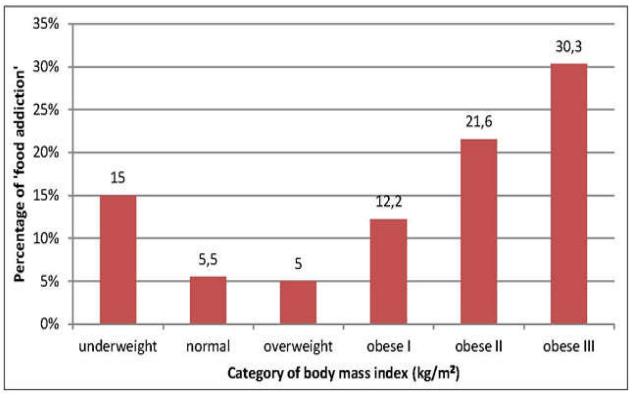

Fig 2. Food addiction: its prevalence and significant association with obesity in the general population. Pedram P, *et al.* (2013)

FINDINGS

Yale food addiction scale: The Yale food addiction scale is the first measure designed specifically to assess signs of addictive like eating behavior. The YFAS allows for a more systematic examination of the hypothesis that highly processed foods might trigger an addictive process for certain people. The YFAS includes 25 items and translates the diagnostic criteria for substance dependence as stated in the DSM-4 to relate the consumption of calorie dense foods. The scale includes items that assess specific criteria, such as diminished control over consumption, a persistent desire or repeated unsuccessful attempts to quit, withdrawal, and clinically significant impairment. The YFAS includes two scoring a "symptom count" ranging from 0 to 7 that reflects the number of addiction like criteria endorsed and dichotomous "diagnosis" that indicates whether a threshold of three or more "symptoms" plus clinically significant impairment or distress has been met. The YFAS has received psychometric support in a non-clinical population's binge eating populations, obese bariatric surgery patients, and a diverse clinical sample (7,17,18).

Pathophysiology

Hyperpalatable foods: Food addiction is defined as an "eating behavior involving the overconsumption of specific foods in an addiction-like manner". Not all foods are equally addictive, therefore an investigation of the chemical characteristics that may trigger addictive behaviors is needed. Hyperpalatable foods, containing high proportion of saturated fat, sugar, artificial flavors, or sodium have been associated with addictive properties, and public health interventions focused on

reducing the impact of addictive drugs may have also a role in targeting obesity and other, related, metabolic diseases (9,14).

Hyperpalatable foods and drug abuse noted to induce similar behavioral consequences, like craving, continuous use despite negative effects over own health, and reduced control over consumption. Reduced D2 receptor availability in obesity and substance use disorder vs. healthy controls reported dopamine deficiency. Food addiction, in a similar manner to drugs of abuse, has been noted to decrease D2 receptors density. Individuals who experience less reward to food intake observed to overeat in order to compensate for this reward dysfunction (21,10). Highly processed foods certainly exhibit pharmacokinetic properties with drugs of abuse, i.e., concentrated dose and rapid rate of absorption, due to the addition of fat and/or refined carbohydrates. These properties found to explain the highly addictive properties of hyperpalatable foods. This hypothesis was tested experimentally in a group with 120 participants associated with addictive-like eating behaviors. Processed food, higher fat, and glycaemic load were more frequently associated with problematic, addictive-like eating behaviors, probably due to their ability to induce a faster absorption of fat/sugar into the bloodstream (22,28).

Cravings: Craving is a dynamic process, engaging reward, emotional, salience, self-referential, executive control, and memory networks (Ekhtiari, *et al* 2016). A resting-state fMRI study showed increased functional connectivity between striatal reward network regions and self-referential default mode network regions (medial prefrontal cortex, posterior cingulate cortex, and angular gyrus), insula, and somatosensory cortex in overweight subjects (BMI > 25) relative to healthy controls. In addition, food-craving scores were positively correlated with the increased functional connectivity in the dorsal striatum (Contreras-Rodriguez, *et al* 2015).

Individuals with a high affinity for palatable foods tend to exhibit increasd cravings when exposed to food cues (Stojek, *et al* 2015). Food craving for sweet or carbohydrate-rich foods was found to be a partial mediator between addictive eating and both elevated BMI and binge-eating episodes (Joyner, *et al* 2015), while cravings for high-fat foods appeared to mediate the relationship between addictive eating and elevated BMI(29). Evidence for neural adaptations in brain reward circuitry contribute to addictive eating. Increased connectivity in the dorsal striatal network were observed in obese compared to normal weight subjects. In addition to the striatum, brain reward regions usually associated with food cravings include the ventral tegmental area, nucleus accumbens, amygdala, and hippocampus (Frankort *et al*., 2014). Greater brain activation in the insula, caudate, and hippocampus were noticed among subjects provided with monotonous diet for 1.5 days, consisting complete nutrition drink "Boost," in response to cues of their favorite foods (Pelchat, *et al* ; 2004). Notably Overlapping in the brain regions was observed with those seen for drug cravings during cravings for favored foods. Evidently similar activation patterns of reward circuitry related to cravings in both FA and drug addiction were noticed (Volkow, *et al*; 2002)(29).

Mood and Stress: There is evidence that those with FA or FA symptomology exhibit addictive eating behaviors to cope with negative emotional states (Micanti *et al*., 2016). For example, binge-eating behavior is associated with psychosocial factors such as negative affect and mood dysregulation, weight cycling (Zwaan, *et al* 2015), body dissatisfaction (Goldschmidt, *et al*; 2016), fear of self-compassion (Kelly, *et al*; 2014), and neuroticism (Womble *et al*., 2001). Lower food consumption were reported among underweight as well as normal weight individuals experiencing negative emotions than positive emotions. Relatively overweight individuals experiencing negative emotions consumed more food (Geliebter & Aversa, 2003). Similarly women diagnosed with FA showed significantly higher levels of depression than those whose scores did not indicate FA (Berenson, *et al* 2015(33).

There has been a substantial evidence to support the connection between stress and addiction (Cui *et al*., 2013;Taylor *et al*., 2014). **Stress** has been found to be associated with a change in eating patterns, including binging episodes and craving highly palatable foods (Sinha & Jastreboff, 2013). Negative affect and stress represent withdrawal feature in individuals with FA when consumption of highly palatable foods stops. Thus, leading to increased motivation to continue addictive eating habits in an effort to mitigate the negative symptoms (Avena, *et al* 2011; Avena, *et al* 2008). This process is consistent with other substance addictions that maintain the addictive cycle through engagement in the addictive behavior, leading to satiation and tolerance, withdrawals and cravings, and continued use of the substance (Sussman & Sussman, 2011)(33).

Impulsivity: Impulsivity, defined as an unplanned response to internal or external stimuli, without prior forethought and a disregard for potential negative consequences (Bari & Robbins, 2013), may account for reward-seeking behavior, and is associated with higher rates of relapse among addicted individuals (Doran, *et al*; 2004). Impulsivity includes difficulties with response inhibition and the inability to delay gratification, as evidenced by choosing an immediate reward over a long-term benefit (Winstanley, *et al* 2006).

Higher levels of impulsivity found to be associated with hedonic eating (ie, eating after energy requirements have been met) patterns (Nederkoorn, *et al* 2006). Impairments in impulse control mechanisms contribute to many disorders, including binge eating, bulimia nervosa, drug addiction, alcoholism, and Internet gaming (Chen *et al*., 2016;)(23). Studies indicate that aberrant activities in regions such as the prefrontal cortex, anterior cingulate cortex, inferior frontal gyrus, and orbitofrontal cortex seems to be accompanied with impulsive behaviors (Rothbart, & Volkow, 2015). In a clinical study, decreased activation of executive brain regions (ie, frontal gyri and inferior parietal lobule) in obese subjects was correlated to increased impulsivity as well as future weight gain (Cook, & Weller, 2013). Results suggest deficits of inhibitory functions in obese subjects. In addition, other self-regulatory control studies showed that people with bulimia nervosa had increased impulsivity with abnormal anterior cingulate and frontal cortical engagement (Marsh *et al*., 2011). Additional results suggest that those with disordered eating showed dysfunctional frontostriatal systems leading to a loss of control over feeding behavior. Deficient executive control, associated with impulsivity, as well as dysregulated craving and reward circuitry, found to contribute FA(23).

Gene profile: A systematic review and meta-analysis (n = 33 studies) compared patients with A1 allele of the Taq1A polymorphism (associated with a 30–40% lower number of D2

receptors, and being considered a risk factor for drug addiction) and patients without this allele, but no BMI difference between the two groups has been found. Although this meta-analysis did not support the presence of a reward deficiency in food addiction, there are reports that individuals with A1 allele are less able to benefit from an intervention aimed to reduce weight, possibly by interfering with increased impulsivity (31). In a trial, greater carbohydrate and fast food craving were associated with A1 vs. A2 allele among Asian Americans college students (N = 84), although no BMI differences were found between A1/A1 or A1A2 genotype and A2A2 genotype (31). A composite index of elevated dopamine signaling (a multilocus genetic profile score) was higher in patients with food addiction diagnosed on the YFAS scoring system, and it correlated positively with binge eating, food cravings, and emotional overeating. The relationship between the genetic index of dopamine signaling and food addiction is mediated by certain aspects of reward-responsive overeating (32).

Neural activation: Serotonin has an important role in modulating food and drug reinforcement. A 11C-DASB-PET study in 60 healthy volunteers reported a negative correlation between cortical and subcortical serotonin transporter (SERT) with BMI values, while tobacco and alcohol consumption did not affect cerebral SERT binding. Several anti-obesity drugs act through SERT blockade, which is also an argument for the involvement of serotonergic transmission in the pathogenesis of eating disorders (23). Foods modulate endogenous opioids and cannabinoids as a function of palatability, and cause delayed increases of dopamine by increasing glucose and insulin. The combination of naltrexone and bupropion is marketed for the treatment of obesity, supporting the positive impact of opioidergic neurotransmission in the regulation of food intake, food craving, and other aspects of eating behavior that affect body weight(29).

Dysfunctions of the hypothalamic-pituitary-adrenal axis and CRF have been reported in the withdrawal phase of the addictive cycle. Wihdrawal was accompanied by increased CRF expression and CRF1 electrophysiological responsiveness in the central nucleus of the amygdala in rats withdrawn from palatable foods (11). In a trial with 48 healthy adolescent females, ranging from lean to obese, food addiction scores correlated with significantly greater activation in the anterior cingulate cortex, medial orbitofrontal cortex, and amygdala in response to anticipated food consumption. Higher YFAS scores were present in patients presenting greater activation in the dorsolateral prefrontal cortex and caudate in same tests, but less activation in the lateral orbitofrontal cortex, when compared to low scores (14). Similar patterns of neural activation have been found in food addiction and substance use disorders, consisting mainly in elevated activity within the reward circuitry in response to food/drug cues and low activity in the circuitry responsible for inhibition of responses to food intake. These data are supported by meta-analyses which evidence greater activation in the amygdala/hippocampus in obese patients compared to normal weight participants in the pre-meal phase, while in the post-meal phase obese individuals had geater activation in the caudate and medial prefrontal cortex vs. normal weight individuals. Neural structures involved in the caloric evaluation, arousal, and memory were more active in obese patients before eating, while less activity was found in areas linked to interoceptive processing. In the post-meal phase, greater activity was detected in obese patients in areas related to risk vs. reward evaluation and reward processing (34). The EEG activity in food-addicted and non-food addicted obese people with alcohol-addicted and non-addicted lean controls (N = 20 healthy normal-weight adults, 46 obese participants, and 14 alcohol dependent patients). Evidences showed the neural brain activity to be similar in alcohol addiction and food addiction, a neural pattern consisting of activation in the dorsal and pregenual anterior cingulate cortex, parahippocampal area, and precuneus . Another neural pattern was correlated with obesity and consisted of activation in dorsal and pregenual anterior cingulate cortex, posterior cingulate extending into the precuneus/cuneus, and in the parahippocampal and inferior parietal area. Food-addicted and non-food-addicted obese people differed by opposite activity in the anterior cingulate gyrus (34). The involvement of an impaired cognitive control has been suggested in both substance use disorders and behavioral addictions. Patients diagnosed with food addiction according to the YFAS scores (N = 34) were compared with a control group (N = 34) while performing an Eriksen flanker test and an EEG evaluation. A higher number of errors in the cognitive test and reduced response-locked components on the EEG (ERN and Pe) have been reported in the food addiction group. Therefore, food addiction seems to be associated with impaired performance monitoring, similar to other addictions (15). A genome-wide association study (GWAS) of food addiction that used mYFAS in 9,314 women of European ancestry showed that two loci met genome-wide significance, and they were mapped to 17q21.31 and 11q13.4 areas. These loci could not be related to genes clearly involved in eating behavior. The results were significantly enriched for gene members of the MAPK signaling pathway, and no single-nucleotide polymorphism (SNP) or gene for drug addiction was significantly associated with food addiction after correction for multiple testing (12).

Three main mechanisms have been suggested in the pathogenesis of obesity as an addictive disorder: reward dysfunction, impulsivity and emotion dysregulation. The reward dysfunction is based mainly on dopamine neurotransmission abnormalities, and increased activation of the dorsal- and ventral striatum and orbitofrontal cortex by palatable food. Impulsivity is another feature shared by obesity and addictive disorders, and it is a reflection of an executive-control deficiency that favors short-term rewards of foods/drugs instead of long-term benefits, and it is correlated with decreased activation of medial prefrontal cortex and other executive-control regions. Emotional dysregulation precipitates drugs use or overeating behaviors, and consumption of foods high in fat and/or refined carbohydrates in response to emotional states like stress or negative affect may be relevant for food addiction and obesity (26).

Research	*Report*	*Ref*
Consumption of Drug and alcohol	Food addiction **	3 6
Appetite and satiety	Food addiction **	36
Food cravings	Food addiction **	36
Drug	Food addiction **	37
Psychological illness	food addiction***	37
Obesity	food addiction ***	1

Note: * Significant ; ** Highly Significant ; *** Extremely Significant

Food addiction is considered as an important link for a better understanding of psychiatric and medical problems triggered

by dysfunctions of eating behaviors eg, obesity, metabolic syndrome, binge eating disorder, or bulimia nervosa.

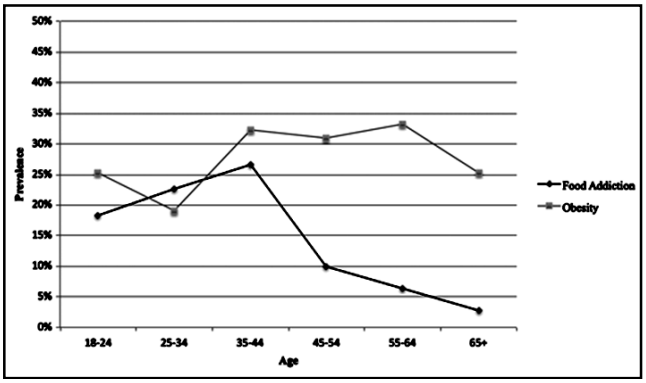

***Fig 3. Psychiatric and physiological significance with Food addiction* Volkow ND, Wang GJ, Tomasi D, Baler RD.(2013)**

At behavioral level, food addiction has high degrees of similarity with other eating disorders, a phenomenon that creates difficulties in finding specific diagnostic criteria (1).

Food addiction is considered as a disorder based on functional negative consequences, associated with distress and potential risks to both psychological well-being and physical health. A clinical scale was validated for the quantification of the eating addiction severity, namely the Yale Food Addiction Severity Scale (YFAS), constructed to match DSM IV criteria for substance dependence. Using this instrument, a high prevalence of food addiction was found in the general population, up to 20% according to a meta-analytic research. The pathogenesis of this entity is still uncertain, but reward dysfunction, impulsivity and emotion dysregulation have been considered basic mechanisms that trigger both eating dysfunctions and addictive behaviors. Genetic factors may be involved in this dependence, as modulators of higher carbohydrate and saturate fat craving(1).

Scientific evidence based reports showed the global prevalence of 'food addiction' to be 7 to 30 % across different age group using YFAS 2.0. Findings noticed that younger Individuals with increased Body Mass exhibited higher rate of food addiction. It is hypothesized that obese and underweight young adults exhibited higher rate of 'food addiction'. Occurrence of 'food addiction' noted to be higher among underweight i.e 15.0 % and obese 17.2% young adults compared to those with normal weight. Certainly there was a 'food addiction' inclination among individual associated with eating disorders i.e anorexia nervosa and underweight (3). Notably 6.7% females and 3.0% males exhibited higher rate of 'food addiction' i.e 5.4%. Often the prevalence rate was increased with obesity status. The clinical symptom counts of 'food addiction' were positively correlated with body composition measurements ($p<0.001$). Obesity measurements were significantly higher in food addicts than controls. Food addicts were noted to be 11.7 (kg) heavier, 4.6 BMI units higher, and had 8.2% more body fat and 8.5% more trunk fat. Furthermore, food addicts consumed more calories from fat and protein compared with controls (3). Food addiction has a high degree of comorbidity with other psychiatric disorders, a phenomenon which is also frequently reported in patients presenting other substance use disorders or behavioral addictions. Research has been stated that dual diagnosis is the rule, rather than the exception, especially in clinical samples. Possible explanations for this high rate of co morbidity found to include self-medication, shared genetic vulnerability, common environment, lifestyle, or neural pathways (2). Clinically and epidemiologically supported observation has severe negative consequences reflected in lower treatment adherence, higher risk for physical complications, poorer overall health, poorer self-care, increased suicide or aggression risks, possible legal problems, and greater health burden for patients with dual diagnosis. Also, co-addiction is frequently reported in patients, and multiple substance and/or behavioral addictions are being clustered together (1). Mental health needs to be more focused on the acute psychological manifestations of a certain disorder, and ignore or minimize the importance of addictive behaviors, which could occupy the background of the clinical presentation. The use of screening questionnaires or structured interviews may increase the rate of early detection, especially in cases of behavioral addictions, a nosological category that is not yet very well-acknowledged by clinicians (1)

Neuroimaging studies reports on Food Cues: Markedly the substance-related cues noted to increase the activation of reward networks involving the dorsolateral prefrontal cortex, orbitofrontal cortex, anterior cingulate cortex, amygdala, insula, and striatum and in addicted vs healthy subjects. Often food cues with addicts assumed to contribute to continued use and relapse (Franklin *et al*., 2007). Mood states considerably found to impact attention in response to food cues in those with and without FA (Frayn, Sears, & von Ranson, 2016). Increased attention to unhealthy food cues were observed among diagnosed FA with induced sad mood based on the YFAS instrument compared to healthy controls. Conversely, when the desired substance is used, it typically leads to decreased reward circuit activation (Martinez *et al*., 2005, 2007; Volkow *et al*., 1997). This suggests that addicted individuals relative to healthy controls place a higher reward value on substance-related cues, but experience a lower level of satiation when the substance is used (18). Neural activation among obese individuals diagnosed with FA resultant of food cues observed to be much greater in the left dorsomedial prefrontal cortex, orbitofrontal cortex, right precentral gyrus, anterior cingulate cortex, amygdala, striatum, mediodorsal thalamus, and right parahippocampal gyrus (Brooks, *et al*, 2013; McBride *et al*., 2006). Like other drug-addicted individuals, those who are obese show less dorsal striatal and medial orbitofrontal cortex activation during the consumption of their "drug of choice" (Stice, *et al* 2008)(17). It has been hypothesized that higher neural activation patterns were found in obese individuals diagnosed with FA. Gearhardt, *et al*. (2011) reports that individuals who scored high on the YFAS (endorsed four or more items) showed activation in the left anterior cingulate cortex, left medial orbitofrontal cortex, and left amygdala when presented with a highly palatable food cue (ie, a chocolate milkshake solution administered through a syringe into the mouth of the subject while in the scanner), while those with low YFAS scores did not. Furthermore, the FA group showed decreased activation in the lateral orbitofrontal cortex during consumption of highly palatable food (Gearhardt, Yokum, *et al*., 2011). These results mirror those reported in research on other substance addictions, as well as those seen among obese individuals (Brooks *et al*., 2013). This provides evidence for a neurobiological mechanism underlying reward circuitry activation in individuals with FA (32).

Controlled clinical studies using Humans and animals: Studies conducted on humans and animals have shown the

acceptable pleasure centers of the brain triggered by addictive substances – including drugs, such as heroin and cocaine,

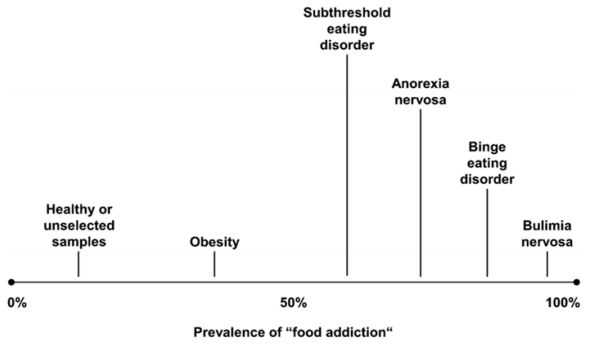

Fig 4. An evaluation of food addiction as a distinct psychiatric disorder, International Journal of Eating Disorders. Erica M. Schulte *et al*. (2020)

or alcohol as well as fat and sugars (25). As with addictive drugs, certain foods could trigger brain chemicals like dopamine. Pleasure experiences induced with certain foods leads to increased dopamine levels resulting into to cravings for eating certain foods. In addition certain foods found to reduce feelings of pain towards enhancing pleasure. Overeating, binging and food addiction to sugar and starch found to be related to serotonin levels in the pain-reduction centers of the brain (25). Leptin, a hormone produced by fat cells, is thought to be involved in the regulation of body fat. Low leptin levels – especially those with a rare genetic disorder called Prader-Willi syndrome, leads to a constant feeling of hunger resulting into a overeating of all foods. Subjects with celiac disease suggested difficulty feeling satiated and tend to overeat. Insulin deficiency could contribute to a false feeling of starving, triggering an overeating or binging episode. Research conducted on self-assessed food addicts in Overeaters noticed successful losing weight by dealing first with physical craving and then completely eliminating foods that prompted cravings and binges (31). Today, fortunately, the burgeoning field of nutritional psychiatry is finding there are many consequences and a correlation between not only what is eaten, how they feel is, and how they ultimately behave, but also the kinds of bacteria that live in the gut. Serotonin is a neurotransmitter that helps regulate sleep and appetite, and inhibit pain. Since about 95% of the serotonin is produced in the gastrointestinal tract, and the gastrointestinal tract is line with a hundred million nerve cells, or neurons, it makes sense that the inner workings of our digestive system it won't just help to digest food, but also guide emotions. What's more, the function of these neurons – and the production of neurotransmitters like serotonin – is highly influenced by the billions of "good" bacteria that make up intestinal microbiome. These bacteria play an essential role in your health. They protect the lining of your intestines and ensure they provide a strong barrier against toxins and "bad" bacteria; they limit inflammation; they improve how well they absorb nutrients from food: and they activate neural pathways that travel directly between the gut and the brain (32). Studies have compared "traditional" diets, like the Mediterranean diet and the traditional Japanese diet, to a typical "Western" diet and have shown that the risk of depression is 25% to 35% lower in those who eat a traditional diet. Scientists account for this difference because these traditional diets tend to be high in vegetables, fruits, unprocessed grains, and fish and seafood, and to contain only modest amounts of lean meats and dairy. They are also void of processed and refine foods and sugars, which are staples of the "Western" dietary pattern.

In addition, many of these unprocessed foods are fermented, and therefore act as natural probiotics. This may sound implausible, but the notion that good bacteria not only influence what your gut digests and absorbs, but that they also affect the degree of inflammation throughout the body, as well as your mood and energy level, is gaining traction among researchers (31)

CONCLUSION

A high degree of overlap between food addiction, eating disorders recognized by current classifications, and metabolic issue i.e obesity could be a significant obstacle for designing clinical trials. The importance of finding a correct conceptual framework for food addiction derived from its pathology and metabolic condition is needed (8). Regarding the existence of potential therapeutic solutions, antiepileptic drugs, opioid antagonists, antiaddictive agents are recommended for obesity and eating disorders, and they may be intuitively used in food addiction, but clinical trials are necessary to confirm their efficacy. In conclusion, a better understanding of food addiction's clinical profile and pathogenesis noted to help clinicians in finding prevention- and therapeutic-focused interventions in the near future (26).

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